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**THE MODALITY SHIFT EFFECT IN SCHIZOPHRENIA -- FACT OR
ARTIFACT?**

City University of New York

PH.D. 1982

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THE MODALITY SHIFT EFFECT IN SCHIZOPHRENIA --

FACT OR ARTIFACT?

by

SALVATORE MANNUZZA

**A dissertation submitted to the Graduate
Faculty in Psychology in partial fulfill-
ment of the requirements for the degree
of Doctor of Philosophy, The City Univer-
sity of New York.**

1982

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1982

This manuscript has been read and accepted for the Graduate Faculty in Psychology in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

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Abstract

THE MODALITY SHIFT EFFECT IN SCHIZOPHRENIA --
FACT OR ARTIFACT?

by

Salvatore Mannuzza

Adviser: Professor Mitchell L. Kietzman

The general finding of crossmodal reaction time research has been that schizophrenics are disproportionately retarded when the stimulus of the previous trial is in a different modality: i.e., they are significantly more slowed than normals on "crossmodal," relative to "ipsi-modal," trials. The interpretation of this finding has been that schizophrenics are impaired in their ability to shift attention across stimulus modalities.

An alternative interpretation is that the "modality shift effect" is simply reflecting the tendency of schizophrenics to respond more slowly than normals, coupled with the greater ability of the cross-modal condition to discriminate slow from fast responders. Chapman and Chapman contend that, whenever schizophrenics and normals are compared, experimental and control variables must be matched on "discriminating power" (i.e., the ability to discriminate "less competent" subjects from "more competent" subjects) to control for the possibil-

ity that psychometric artifact is accounting for the findings.

The purpose of the current research was to assess whether psychometric artifact could account for the modality shift effect. The sample consisted of 15 hospitalized, male patients (mean age = 29.6 yrs., SD = 8.2 yrs.) who fulfilled diagnostic criteria for schizophrenia, and 50 male controls (mean age = 29.3 yrs., SD = 7.8 yrs.) who were screened for a past or present episode of mental disorder. Ten intensities of auditory stimuli (2000 Hz tones) and 10 intensities of visual stimuli were presented in quasi-random order. Stimuli within each modality ranged from 1.8 to 2.7 log units above the threshold determined for one normal. All stimuli were 10 msec. Subjects were instructed to lift their finger from a key, as quickly as possible, when any stimulus was presented.

The major finding was that psychometric artifact could not account for the modality shift effect. Highly significant ($p < .001$) modality shift differences between patients and controls were found for both modalities. The reliabilities and variances (the critical determinants of discriminating power) of the crossmodal and ipsimodal conditions of normals ($N = 50$) and of a subgroup of slow-responding normals ($N = 15$), were assessed. Reliability coefficients were high (.98-.99), and variances did not differ significantly. Therefore, psychometric artifact could not account for the modality shift effect since crossmodal and ipsimodal conditions were comparable on estimates of discriminating power.

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CHAPTER I

INTRODUCTION

Anomalies of attention have long been considered a prominent characteristic of schizophrenia. Attentional dysfunction is described as a principal disturbance of schizophrenia ("dementia praecox") in the writings of several eminent historical persons in psychiatry, such as Kraepelin (1919), Bleuler (1924), and Jaspers (1923). Autobiographical accounts by patients have also attested to the extreme and debilitating effects of the attentional disturbance (Freedman, 1974; Freedman & Chapman, 1973; MacDonald, 1960; McGhie & Chapman, 1961; Sechehaye, 1951).

The experimental study of attention encompasses a vast and diversified literature. Numerous research paradigms, such as smooth pursuit eye-tracking (Holzman, Levy, & Proctor, 1978), dichotic listening (Payne, Hochberg, & Hawks, 1970), digit span (Oltmanns & Neale, 1975), span of apprehension (Asarnow & MacCrimmon, 1981) and the continuous performance test (Orzack & Kornetsky, 1966), have been employed to study "attention deficit" in schizophrenic patients. However, intercorrelations among different measures of attention have been low (Asarnow & MacCrimmon, 1978; Kopfstein & Neale, 1972; Payne et al., 1970), suggesting that "attention" is not a unitary concept.

In an effort to provide a conceptual framework within which some of the experimental findings could be better understood, Zubin (1975) proposed three aspects of attention: maintenance, selection, and

shift. Each of Zubin's attentive acts will be illustrated by an example in normals. In addition, excerpts of statements made by schizophrenic patients will be used to portray the subjective experience of deficits in each aspect of attention. Finally, an appropriate research paradigm for measuring each attentive act will be indicated.

During wartime, a submarine crewman is ordered to fix, and main-
tain attention on a scanning screen. One, small, flash of light on the screen could indicate that an enemy submarine is approaching and, thus, the crewman's immediate report of the flash is critical.

The crewman's task is an example of maintenance (or sustained) attention. The subjective experience of deficits in maintenance attention is portrayed in the following excerpts:

I can concentrate quite well in what people are saying if they talk simply. It's when they go on into long sentences that I lose the meanings. It just becomes a lot of words that I would need to string together to make sense.
(McGhie & Chapman, 1961, p. 106)

Sometimes when people speak to me ... It goes out as quick as it goes in. It makes you forget what you've just heard because you can't get hearing it long enough. (McGhie & Chapman, 1961, p. 106)

According to Zubin, one appropriate research paradigm for measuring maintenance attention is the rotor pursuit task. In this procedure, the subject is required to manipulate a stylus as he follows a constantly moving, erratic target. Thus, the task requires a sustained focusing of attention.

Consider an individual who is reading a newspaper while walking down the street. This individual is selectively attending to material in the newspaper article, and is essentially ignoring, or shutting out,

all other irrelevant stimuli bombarding his sense organs (e.g., the rumbling of a passing subway train, the crying of babies from nearby apartment houses, the flashing of a traffic light on the corner, the conversation of people walking by).

The subjective experience of deficits in selective attention is portrayed in the following excerpts:

Things are coming in too fast. I lose my grip of it and get lost. I am attending to everything at once and as a result I do not really attend to anything. (McGhie & Chapman, 1961, p. 104)

Everything that's going on seems to be picked up by me, almost as if I welcome these things to stop me attending fully to any one thing. (McGhie & Chapman, 1961, p. 105)

According to Zubin, one appropriate research paradigm for measuring selective attention is the dichotic listening task. In this paradigm, the subject is required to attend to a message presented to one ear, while ignoring a simultaneous, competing message presented to his or her other ear.

Returning to the individual reading the newspaper, if he inadvertently steps into the street, and a driver of an oncoming bus sounds his horn, focus of attention is quickly shifted to his immediate surroundings.

The subjective experience of deficits in shift of attention is portrayed in the following excerpts:

My responses are too slow ... Things happen but I don't respond. When something happens quickly or unexpectedly it stuns me like a shock. I just get stuck. (McGhie & Chapman, 1961, p. 106)

I may be thinking quite clearly and telling someone something and suddenly I get stuck ... What happens is that I suddenly stick on a word or an idea in my head and I just can't move past it. (McGhie & Chapman, 1961, p. 109)

One appropriate research paradigm for measuring shift of attention is the crossmodal reaction time task. In this procedure, visual and auditory stimuli are randomly presented, and the subject is required to lift his or her finger from a key when either stimulus is delivered. Thus, the task requires trial-to-trial shifts of attention from one stimulus modality to another. Preparatory intervals (or, for experiments in which no warning signals are used, interstimulus intervals) are kept brief, so as not to confound maintenance attention with shift of attention; and since only one stimulus is presented on each trial, selective attention (at least regarding task demands that are imposed by experimental conditions) is not a factor.

The importance of Zubin's system to a theory of schizophrenic attention deficit is that patients can exhibit anomalies in one aspect, or "subsystem," of attention in the absence of impairments in the other subsystems. Consider the following examples of individual differences in normals. Reading a novel while the radio is playing (selective attention) is extremely difficult for some persons, but these same individuals might be capable of sitting in a quiet room for very long periods of time, totally absorbed in highly demanding reading material (maintenance attention). Similarly, some persons can very efficiently watch a full-length movie on television, and read brief newspaper articles during commercials (shift of attention), but these same individuals might find it very taxing to sustain attention, uninterruptedly,

for more than a relatively brief period of time (maintenance attention). In conclusion, Zubin's three subsystems of attention are separable, both conceptually and operationally.

After reviewing the literature on maintenance, selection, and shift, Zubin (1975) concluded that the basic "deficit" in schizophrenic attention concerns shift, and that maintenance and selection anomalies represent "deviations," rather than "deficits":

The deviations that are observed are due not to an intrinsic deficit (in maintenance and selective attention) but to such factors as the "culture" of the schizophrenic, his previous history and reinforcement experience which makes him classify the environmental stimuli in accord with a system uniquely his own. (Zubin, 1975, p. 160)

Zubin's conclusions regarding the importance of attentional shift to a theory of schizophrenic attention deficit were primarily based on the findings of crossmodal reaction time research with schizophrenic patients. However, it is noteworthy that Cromwell and Docecki (1968) proposed a theory of schizophrenic language that is consonant with Zubin's emphasis. According to Cromwell and Docecki (1968):

... an important dysfunction in schizophrenia is the inability to disattend from stimuli. Disattention is the adaptive ability of an organism to withdraw his attention from a stimulus after having attended to it. (p. 249)

The authors go on to say that the schizophrenic "makes responses to current and immediately recent stimulus events and cannot remove these events from his attention"; he "locks his attention upon specific stimuli for a period longer than that of the average person" (p. 249).

Although the terms differ, "attentional shift deficit" and "disattention deficit" appear to be parallel, if not equivalent, concepts.

In the current research, a crossmodal reaction time task was used to study the attentional shift deficit in schizophrenic patients. Cross-modal reaction time research with psychotic patients was prompted by Mettler's (1955) neurological model of perceptual functioning in schizophrenia. Mettler reported that cats with experimentally produced lesions of the striatum (caudate nucleus and putamen) showed a characteristic incapacity to react appropriately to changing environmental events -- the organism's "capacity for sensory shift (was) impaired" (p. 99). The corpus striatum has a vascular supply whose safety margin is well below most other brain structures. Mettler speculated that the schizophrenic patient's attentional difficulty resulted from fluctuations in the vascular supply of the basal ganglia, causing the striatum to malfunction temporarily, and thus producing the anomalies observed in animals. The first crossmodal reaction time experiment employing psychotic patients was conducted to test the hypothesis that schizophrenic patients were impaired in their ability to shift attention across sensory modalities.¹

As indicated above, in the usual crossmodal reaction time experiment, suprathreshold auditory and visual stimuli are quasi-randomized within each block of trials, and the subject's task is to lift his or her finger from a key as quickly as possible when either stimulus is presented. Variations of this general paradigm (e.g., simple vs. choice reaction time, informing vs. not informing the subject which

¹Dr. Gad Hakerem, Department of Psychology, Queens College of the City University of New York, has been accredited for suggesting that the crossmodal reaction time task be used for this purpose.

stimulus will be presented on each trial) have also been employed.

In the analysis of data from a crossmodal reaction time experiment, each trial is classified in terms of its relationship to the modality of the stimulus of the preceding trial. For example, a "crossmodal/light" response is a reaction time to a visual stimulus, preceded by an auditory stimulus on the previous trial; an "ipsimodal/sound" response is a reaction time to an auditory stimulus, preceded by an auditory stimulus on the previous trial.

The general finding of crossmodal reaction time research has been that schizophrenic patients show a greater than normal lengthening of reaction time for crossmodal, relative to ipsimodal, stimulus sequences: i.e., schizophrenic patients are disproportionately retarded (significantly more than normal control subjects) when the modality of the stimulus of the previous trial is different from the modality of the stimulus of the current trial (Spring, 1980; Sutton, Hakerem, Zubin, & Portnoy, 1961; Sutton & Zubin, 1965; Waldbaum, Sutton, & Kerr, 1975). The ability of the difference between reaction times on crossmodal and ipsimodal trials ("crossmodal retardation") to discriminate patient and control groups has been referred to as the "modality shift effect." As its name implies, the interpretation of the modality shift effect has been that schizophrenic patients are impaired in their capacity to shift attention from one stimulus modality to another.

Chapman and Chapman (1973a, 1973b, 1978a) have provided a methodological critique which has been directed against many areas of psychiatric research, including crossmodal reaction time experiments. One premise of the Chapmans' treatise is the frequently cited observation

(e.g., Bannister, 1968; Sutton, 1973) that schizophrenic patients, as a group, perform more poorly than normal control subjects regardless of the particular task or measure employed. Thus, no specific deficit can be inferred from poorer patient performance on any single measure since "generalized performance deficit" (perhaps attributable to decreased motivation, uncooperativeness, or a host of other nonspecific factors) could account for the finding. To circumvent this problem, several investigators have resorted to a "differential deficit methodology" in which the performances of schizophrenic patients and normal control subjects are compared on two measures, an experimental measure (e.g., crossmodal trials in a crossmodal reaction time task) and a control measure (e.g., ipsimodal trial sequences). The hypothesis tested in these experiments is that patients will be differentially affected by the variable of the experimental measure: i.e., that patients will show a greater performance deficit (relative to normal control subjects) on the experimental measure, than on the control measure.

The Chapmans' major point is that, unless measures are matched on discriminating power (defined as the ability to discriminate "more competent" and "less competent" subjects), poorer performance on the experimental measure may simply reflect the psychometric characteristics of the particular measures chosen, coupled with the fact that schizophrenic patients, as a group, perform less competently.

The principal objective of the current research was to assess whether psychometric artifact could account for the modality shift effect. More specifically, the purpose of the present experiment was to determine whether the significant modality shift differences between

schizophrenic patients and normal control subjects that have been reported in previous studies, could be explained by differential discriminating power of crossmodal and ipsimodal trial sequences, coupled with the generalized performance deficit of schizophrenic patients.

In the remaining sections of this chapter: (1) the origins of crossmodal reaction time research (in normals) will be briefly reviewed; (2) factors that have been shown to influence crossmodal retardation will be outlined; (3) theories proposed to explain the modality shift effect in schizophrenia will be discussed; (4) unacceptable solutions to the interpretive difficulties caused by generalized performance deficit will be indicated; and, (5) the Chapmans' methodological critique (and commentaries on the Chapmans' treatise) will be stated more explicitly, with specific reference to its relevance to reaction time research in schizophrenia.

The Reaction Time Task-- An Historical Overview

One of the first measures used in the history of experimental psychology was the reaction time task. As early as 1868, Donders, and somewhat later Wundt and Titchener, employed the reaction time procedure to study a variety of psychological variables, including "choice time," "decision time," "psychological constants," and "attitude" (Boring, 1950).

Shortly after its introduction as an experimental procedure, the reaction time task was used to assess the psychological functioning of psychiatric populations (see Lewis, 1890). As early as 1922 (Wells and Kelley), reviews of reaction time studies in psychoses appeared in the literature. It is noteworthy that one of the first investigators in this area of research was Emil Kraepelin, who was trained in the laboratory of Wilhelm Wundt during the late 1800's (Alexander & Selesnick, 1966).

Interest in the reaction time procedure as a method for detecting differences between normal and pathological populations has persisted through the years (see Nuechterlein, 1977, for a recent review). An abbreviated list of the parameters that have been studied in reaction time experiments with psychotic subjects includes: stimulus intensity (King, 1962; Venables & O'Connor, 1959; Venables & Tizard, 1956a, 1956b, 1958); stimulus complexity (Venables, 1958); preparatory interval (Bellissimo & Steffy, 1972; Tizard & Venables, 1956; Zahn, Rosenthal, & Shakow, 1961), previous preparatory interval (Bellissimo & Steffy, 1975; Nideffer, Deckner, Cromwell, & Cash, 1971; Nideffer, Neale, Kopfstein, & Cromwell, 1971; Zahn, Rosenthal, & Shakow, 1963), and intertrial in-

terval (Steffy & Galbraith, 1974; Zahn, Shakow, & Rosenthal, 1961) in regular and irregular series, and; trial-to-trial changes in the modality of the imperative stimulus (i.e., crossmodal reaction time research).

The Origins of Crossmodal Reaction Time Research-- Experiments With Normals

According to Mowrer, Rayman, and Bliss (1940), the earliest investigations of sequential effects on reaction time were carried out in the late 1800's by Wilhelm Wundt:

If, for example, I introduced in a series of sound trials strong and weak stimuli at random, so that the subject could never expect a particular sound strength, the reaction time was lengthened ... The reaction time is lengthened more when the stimulus is completely unknown than when only the intensity of it is unknown ... Slighter but still very noticeable is the retardation (in quickness of response) if one arranges the experiment to have the observer in ignorance as to whether light, sound, or touch impressions (stimuli) will be forthcoming, so that the attention cannot be turned to a particular sense organ. Immediately one notes a peculiar unrest because the strain of attention continuously vacillates among the several senses. (Wundt, 1893, cited in Sutton and Zubin, 1965, p. 2)

Subsequently, Mowrer and his colleagues (Mowrer, 1941; Mowrer et al., 1940) conducted a series of reaction time experiments to test the hypothesis that "preparatory set" ("attention") is mediated principally by central (rather than peripheral) mechanisms. A selective review of those experiments that are relevant to crossmodal reaction time research follows.

The initial experiments conducted by Mowrer and his colleagues (Mowrer et al., 1940) were based on the assumption that, if preparatory set is primarily dependent on posturing and muscle tensing (i.e., peripheral mechanisms), then information concerning which stimulus to

"expect" (central) should not alter the speed of response. In one experiment, subjects were told to expect either a light or a sound. Identical stimuli were presented on 7 to 21 successive trials, thereafter followed by a crossmodal stimulus. Results supported a central hypothesis, showing a lengthening of reaction time on the trial that the crossmodal (unexpected) stimulus occurred. In a second experiment, lights and tones were alternated, except that after 9 alternations, the next stimulus (tone) was in the same modality as the preceding stimulus. Mowrer et al. reasoned that, if reaction time is lengthened when an unexpected stimulus in a different modality is presented, then a similar slowing should occur when an expected change in the modality of the stimulus does not occur. This prediction was not supported: reaction time decreased (i.e., became faster) when the second of two successive (ipsimodal) tones was presented.

In a second series of experiments, Mowrer (1941) tested the hypothesis that the unexpected presentation of an ipsimodal-nonidentical stimulus would result in a slowed response. In Experiment 1, visual stimuli consisting of the letters "T" and "S" were used. In Experiment 2, vibratory stimuli were delivered to the right and left legs. In Experiment 3, red and green lights were employed. Results of all three experiments indicated that reaction time was not significantly lengthened when the unexpected ipsimodal-nonidentical stimulus was presented.²

The above review of Mowrer's research is neither exhaustive nor

²It should be noted that failure to demonstrate ipsimodal-nonidentical retardation has not been a consistent finding. For example, the quotation appearing at the beginning of this section indicates that Wundt observed ipsimodal retardation in his early experiments.

representative. The purpose of this selective review was to illustrate an observation made by Sutton, Hakerem, Zubin, and Portnoy (1961):

The interesting generalization that emerges (ours, and not Mowrer's) is that reaction time was always longer when the "unexpected" stimulus was in a different sensory modality, and never lengthened when the "unexpected" stimulus was in the same sensory modality. (p. 231)

In conclusion, the results of Mowrer's reaction time experiments with normals, underscored the significance of sequential changes in the modality of the imperative stimulus. In the next section, crossmodal reaction time research with schizophrenic patients is reviewed.

Crossmodal Reaction Time Research with Schizophrenic Patients

To reiterate, the "modality shift effect" refers to the disproportionate (i.e., greater-than-normal) slowing of schizophrenic patients on crossmodal trials, relative to ipsimodal trials. Stated differently, "modality shift effect" refers to the ability of crossmodal retardation (i.e., the average reaction time on crossmodal trials, minus the average reaction time on ipsimodal trials) to differentiate schizophrenic patients from normal control subjects.

The relationship between crossmodal retardation and several subject and methodological variables has been studied. The following is a summary of these findings (see Mannuzza, 1980, for a detailed review):

Chronic and Acute Schizophrenic Patients. The modality shift effect has been demonstrated in chronic schizophrenic patients (Sutton et al., 1961; Sutton & Zubin, 1965, Experiment B); in acute schizophrenic patients (Spring, 1980; Waldbaum et al., 1975); and in process and re-active schizophrenic patients (Sutton & Zubin, 1965, Experiment A).

Nonschizophrenic Pathological Groups. In two experiments (Spring, 1980; Rist, Note 1), schizophrenic patients were compared to hospitalized patients with other mental disorders. Spring (1980) tested schizophrenic and depressed patients, siblings of schizophrenic patient probands, and normal control subjects. Results indicated that both patient groups differed significantly from the control groups on crossmodal retardation to sound stimuli, but only schizophrenic patients differed significantly from controls for crossmodal retardation to light stimuli. Spring concluded that increased retardation to auditory stimuli may be a general marker of psychopathologic disorder, and increased retardation to visual stimuli may be a marker of schizophrenic disorder.

Rist (Note 1) tested schizophrenic patients, alcoholic patients, and normal control subjects in two conditions of a crossmodal reaction time task. One condition included a warning signal, whereas the other condition did not. Results indicated that crossmodal retardation was significantly greater in schizophrenic patients, than in alcoholics or normal control subjects, but only for reactions to sound, and only in the no warning signal condition. These findings will be discussed in more detail in a later section (see section on The Warning Signal).

Brief mention should be made of crossmodal reaction time research in other (i.e., non-psychotic populations): (1) Benton, Sutton, Kennedy, and Brokaw (1962) found that crossmodal retardation to light (but not to sound) stimuli was significantly greater for non-psychotic, adult brain-damaged patients, than for a control group of hospitalized medical patients. (2) Deutsch and Schumer (1970) failed to find crossmodal retardation differences between brain-damaged and normal children.

(3) Raab, Deutsch, and Freedman (1960) and Katz and Deutsch (1963) found that crossmodal retardation significantly discriminated children classified as retarded readers from children with no reading deficiencies; however, Blank, Berenzweig, and Bridger (1975) failed to replicate this finding.

In comparison to the findings of crossmodal reaction time experiments with non-psychotic populations, significant modality shift differences between schizophrenic patients and normal control subjects have been shown in seven experiments (Spring, 1980; Sutton et al., 1961; Sutton & Zubin, 1965, Experiments A, B, and C; Waldbaum et al., 1975; Rist, Note 1), with only one failure to replicate (Verleger & Cohen, 1978). In conclusion, the only population for which significant modality shift differences have been a consistent finding is schizophrenic patients; however, the differential effects of crossmodal retardation have been largely unexplored in other populations, and it is not known how the performances of schizophrenic patients would compare, for example, to the performances of brain-damaged patients, since these groups have never been tested in the same experiment.

Psychotropic Medication. There is compelling evidence that psychotropic medication cannot account for the modality shift effect. First, drug-free patients were tested in most crossmodal reaction time experiments (Sutton et al., 1961; Sutton & Zubin, 1965, Experiments A, B, and C; Waldbaum et al., 1975). In addition, Spring (1980), who tested medicated schizophrenic and depressed patients, found that correlations between drug dosage and the magnitude of crossmodal retardation were not significant for either patient group, and for either

modality (light or sound). Finally, Rist (Note 1), who also tested medicated schizophrenic patients, found significant correlations between drug dosage and retardation, but only for the modality (light) in which significant patient-control differences in crossmodal retardation were not demonstrated. In conclusion, although the relationship between psychotropic medication and crossmodal retardation has never been systematically studied, there is ample convergent evidence that the significant modality shift differences between schizophrenic patients and normal control subjects cannot be explained by drug effects.

Sex. The modality shift effect has been less consistent for female, than for male, schizophrenic patients. In three experiments, both male and female patients were tested. The results of one experiment showed no significant differences between male and female patients (Sutton et al., 1961). The results of the other two experiments showed the modality shift effect for male, but not for female, patient-control comparisons (Sutton & Zubin, 1965, Experiments B and C).

Simple and Choice Reaction Time Tasks. A simple reaction time task is one in which there is no response uncertainty. In a choice reaction time task, some decision ("choice") precedes the reaction time response.

The modality shift effect has been consistently demonstrated in simple crossmodal reaction time studies (Spring, 1980; Sutton et al., 1961; Sutton & Zubin, 1965, Experiments A and C; Waldbaum et al., 1975). In two experiments (Sutton & Zubin, 1975, Experiment B; Verleger & Cohen, 1978), a choice reaction time task was used, in which subjects were required to press different keys, depending on which stimulus was

presented. Sutton and Zubin (1965, Experiment B) found the significant patient-control differences in crossmodal retardation, whereas Verleger and Cohen (1978) did not.³ In conclusion, whereas the modality shift effect is a well established finding in simple crossmodal reaction time experiments, modality shift differences between patients and controls tested on a choice reaction time task warrant further substantiation.

Stimulus Modality. Most crossmodal reaction time experiments have shown significant modality shift differences between schizophrenic patients and normal control subjects for reactions to auditory stimuli, but not to visual stimuli (Sutton et al., 1961; Sutton & Zubin, 1965, Experiments A, B, and C; Rist, Note 1). However, the two studies that yielded a positive modality shift effect for both light and sound stimuli (Spring, 1980; Waldbaum et al., 1975) were the only experiments in which visual and auditory stimuli were equated with respect to sensation level. Spring (1980) and Waldbaum et al. (1975) determined the absolute visual and auditory detection thresholds for one normal subject. The intensity of each stimulus was then increased, by an equal amount (in log units), above the respective absolute threshold values (2.0 log units above threshold in Spring's experiment; 2.9 log units above threshold in Waldbaum et al.'s experiment). Thus, in both of these experiments, light and sound stimuli were set and maintained at a constant duration (10 msec in both studies) and intensity (determined

³The experiment conducted by Verleger and Cohen (1978) was the only study in which the modality shift effect was not found. Possible reasons for Verleger and Cohen's failure to replicate the findings of seven other crossmodal reaction time experiments are indicated in the section on The Warning Signal.

by the threshold procedure) for all subjects and experimental conditions. Sutton et al. (1978) have suggested that not equating for sensation level might account for the failure of earlier investigators to find a modality shift effect for reaction times to light stimuli, in addition to sound stimuli.

Total Stimulus Energy. There is some evidence that the magnitude of crossmodal retardation is greater for less intense stimuli. Hannes (Note 2) tested schizophrenic patients and normal control subjects under two conditions in a crossmodal reaction time paradigm. In Condition 1, both auditory and visual stimuli were set at 1.8 log units above absolute detection threshold. In Condition 2, stimuli were fixed at 2.4 log units above threshold. Results suggested that patients exhibited greater crossmodal retardation in the low-intensity condition. The performance of normal control subjects was less clear. Unfortunately, no patient-control comparisons or statistical analyses were conducted.

The Warning Signal. One might question whether sensory input intervening between reaction time trials could influence the degree of crossmodal retardation. In this regard, the warning signal is suspect, since it is regularly presented at the beginning of each trial.

Warning signals were employed in four crossmodal reaction time experiments (Spring, 1980; Verleger & Cohen, 1978; Waldbaum et al., 1975; Rist, Note 1). Waldbaum et al. (1975) tested schizophrenic patients and normal control subjects in two conditions. In the "Certain" condition, subjects were told (verbal warning signal), prior to each trial, whether a light or a sound would be presented. In the "Uncertain" condition, subjects were asked to guess (i.e., the experimenter said

"Guess"), prior to each trial, which stimulus would be presented. The modality shift effect was demonstrated in both Certain and Uncertain conditions, and for reaction times to both light and sound stimuli.

In an attempt to replicate the findings of Waldbaum et al. (1975), Verleger and Cohen (1978) tested schizophrenic patients and normal control subjects under conditions of certainty and uncertainty. A visual warning signal was used in both conditions. In the Certain condition, the word "LICHT" (light) or "TON" (sound) was displayed before each trial; in the Uncertain condition, "RATEN" (guess) was presented. Verleger and Cohen failed to replicate the findings of Waldbaum et al.: i.e., no modality shift effect was found for either modality, in either condition.

The use of a visual warning signal was only one of several differences between the experiments of Waldbaum et al. (1975) and Verleger and Cohen (1978). For example: Verleger and Cohen tested chronic, medicated, non-paranoid patients with a hospital diagnosis of schizophrenia, and a median age of 41 years, whereas Waldbaum et al. tested acute, drug-free patients with a computer-derived diagnosis of schizophrenia (unselected for schizophrenia subtype), and a mean age of 24 years; Verleger and Cohen used a finger-depress response in a two-choice reaction time paradigm, whereas Waldbaum et al. required a finger-lift response in a simple reaction time task; the auditory stimulus used by Verleger and Cohen was a 1000 Hz tone delivered through stereophonic earphones, whereas the auditory stimulus of Waldbaum et al. was a white noise burst emanating from a loudspeaker; and, Waldbaum et al. equated stimuli with respect to sensation level, whereas Verleger

and Cohen did not. These differences make it difficult to compare the two experiments, and to assess why Verleger and Cohen failed to replicate the findings of Waldbaum et al.

Spring (1980) employed two ways of conveying information about the forthcoming stimulus. On "Experimenter-Told" (E-Told) blocks, before each trial, the subject was verbally told (as in the Certain condition of Waldbaum et al.) which stimulus (a light or a sound) would be presented. On "Machine-Told" (M-Told) blocks, the identical sound or light stimulus to be responded to, was used as a warning signal to forewarn the subject which stimulus was forthcoming. Thus, for both E-Told and M-Told blocks of trials, light and sound stimuli were randomized within each block, and the subject always knew which stimulus would be presented. The modality shift effect (schizophrenics vs. controls) was found for both E-Told and M-Told blocks, and for both light and sound stimuli.

The M-Told condition was initially used by Spring (1980) in an attempt to "normalize schizophrenic crossmodal retardation" by reducing social interaction between experimenter and subject and, consequently, decreasing the disruptive effects of social anxiety on schizophrenic attention. However, it subsequently became clear that since the identical stimulus to be presented, was used as a warning signal on each trial of M-Told blocks, reaction times in the M-Told condition were all ipsimodal. Despite this fact, Spring found that crossmodal retardation differentiated schizophrenics from normals. This finding led Spring (1980) to conclude that "crossmodal retardation is influenced primarily by the sequence of responded-to stimuli" (p. 139).

In the only systematic investigation of the effects of a warning signal, Rist (Note 1) tested schizophrenic patients, alcoholic patients, and normal control subjects in two conditions of a crossmodal reaction time paradigm. In one condition, no warning signal was used. In the other condition, a warning signal, whose modality (light or sound) was varied randomly and independently from the modality of the imperative (responded-to) stimulus, preceded each trial. Different from Spring's (1980) experiment, all trials were "uncertain": i.e., the warning signal conveyed no information about the forthcoming stimulus. Rist (Note 1) found that crossmodal retardation differentiated schizophrenics from alcoholics and normals only in the no warning signal condition, and only for reaction times to sound stimuli.

It is noteworthy that the warning signals used by Rist were 200 msec in duration, whereas Spring's warning signals were 10 msec. Perhaps this difference, in part, can explain why Spring found the warning signal to be (presumably) irrelevant, whereas the warning signal emerged as a major variable in Rist's experiment. In any case, Rist's findings underscore the potential significance of the warning signal in cross-modal reaction time experiments and, consequently, refute a simple theory based on the overriding importance of "responded-to" stimuli.

Summary. The results of seven experiments have shown that schizophrenic patients are disproportionately retarded in a reaction time task when the modality of the stimulus of the previous trial is different from the modality of the stimulus of the present trial. The modality shift effect has been demonstrated in chronic and acute, process and reactive schizophrenic patients. When schizophrenic and depressed pa-

tients are compared to control subjects, both patient groups differ significantly from controls on crossmodal retardation to sound stimuli, but only schizophrenics differ significantly from controls on crossmodal retardation to light stimuli. When no warning signal is used, and schizophrenic patients, alcoholic patients, and normal control subjects are compared, schizophrenics exhibit significantly greater crossmodal retardation (to sound stimuli) than alcoholics and controls.

There is ample convergent evidence that psychotropic medication cannot account for the significant modality shift differences between schizophrenic patients and normal control subjects. Furthermore, in two experiments, crossmodal retardation discriminated patients from controls despite the fact that subjects were told, prior to each trial, whether an auditory stimulus or a visual stimulus would be presented.

The modality shift effect is more consistent for male subjects, than for female subjects; for simple reaction time tasks, than for choice reaction time tasks; and for reactions to sound stimuli, than to light stimuli. However, the inconsistent findings across stimulus modalities could be due to the failure of most investigators to equate auditory and visual stimuli with respect to sensation level. In addition, there is some evidence that crossmodal retardation is greater for less intense stimuli. Finally, in one experiment, the introduction of a warning signal abolished the significant modality shift differences between patients and controls that were demonstrated in the no warning signal condition.

The major theories that have been proposed to explain the modality shift effect in schizophrenia will now be reviewed.

Theoretical Explanations of the Modality Shift Effect in Schizophrenia

Zubin's Neural Trace Theory. Zubin (1975) proposed a neural trace theory to account for crossmodal retardation. According to this theory, with the presentation of a stimulus, facilitory and inhibitory traces remain within the organism's neuron pool, and have an effect on the processing of future stimuli. More specifically, the processing of similar subsequent stimuli is facilitated, and the processing of dissimilar subsequent stimuli is inhibited, by these traces. Thus, a subject's reaction time will be faster with repeated presentations of the same stimulus (practice effect) since more facilitory neuron traces are contributing to his response. Similarly, crossmodal retardation will be more dramatic if several identical stimuli precede the crossmodal stimulus, since more inhibitory traces have accumulated. Zubin proposed that both facilitory and inhibitory traces persist longer in the schizophrenic patient; thus, the discriminative power of the modality shift effect.

As Zubin (1975) has indicated, with the passage of time, one would expect the strength of trace residues to eventually dissipate. Thus, an inverse relationship between the duration of intertrial interval and the magnitude of crossmodal retardation is predicted. Although this relationship has never been studied systematically, Waldbaum et al. (1975) reported that, in their experience, "when the interval between trials was lengthened, up to 10 sec, changes in the degree of reaction time retardation were not systematic" (p. 168).

Zubin's theory also predicts that, as a result of more persistent facilitory traces in the nervous system of schizophrenics, patients should exhibit a greater than normal practice effect (i.e., a faster

than normal decrease in reaction time over trials) when the identical stimulus is presented on successive trials. Interest in the modality shift effect has focused primarily on the "retardation" aspect of the effect. However, one cannot determine from a difference score (i.e., crossmodal minus ipsimodal reaction time) whether disproportionate facilitation or retardation (or both) is responsible for the ability of "crossmodal retardation" to discriminate schizophrenic patients from normal controls. (Incidentally, lengthening intertrial interval would also decrease ipsimodal "facilitation" according to Zubin's theory, since facilitory traces would also be expected to dissipate over time.)

The only systematic investigation of ipsimodal facilitation in a crossmodal reaction time experiment was conducted by Sutton and Zubin (1965, Experiment C). Data were analyzed for red lights and high-frequency tones (separately for males and females), with respect to the number of preceding identical stimuli (0, 1, 2, or 3). The only patient-nonpatient comparison that yielded a significant difference was that male patients displayed a significantly greater decrease in reaction time (i.e., "facilitation") than male controls for responses to high tones. However, inspection of the data shows that very little "facilitation" was exhibited by male normal controls, who displayed reaction times in the 180 msec range. Thus, a basement effect is suspect. The data would have been more convincing (with regard to Zubin's predictions) if both patients and nonpatients demonstrated a practice effect, and patients showed a significantly faster decline.

Sutton and Zubin (1965, Experiment C) also compared subjects on reaction times to low-frequency tone stimuli when these had been preceded

by either one, two, three, or four red lights. According to neural trace theory, retardation should increase as a function of the number of preceding dissimilar stimuli. However, no clear trend was found.

The relationship between Spring's (1980) and Rist's (Note 1) findings (see section on The Warning Signal) and the predictions made by neural trace theory is unclear. Contrary to Spring's results, Zubin's theory predicts that no modality shift effect should be found in her M-Told condition since only ipsimodal stimulus sequences were presented on these blocks of trials. Thus, unless a distinction is made between imperative ("responded-to") and informational (warning signal) stimuli, and a "relevant trace" theory is proposed, Spring's (1980) M-Told findings must be considered inconsistent with Zubin's theory. Rist (Note 1), on the other hand, found the modality shift effect in his no warning signal condition; however, when a warning signal was introduced, the modality shift effect was no longer demonstrated. These data suggest that intervening, non-imperative (i.e., "non-responded-to") stimuli do influence reaction time performance on successive trials.

Sutton (Note 3) has offered an explanation for the apparent, contradictory findings of Spring (1980) and Rist (Note 1). According to Sutton, "attention deficit" implies a limited, or deficient focusing of attention on relevant stimuli, as well as an inappropriate, or supra-focusing of attention on irrelevant stimuli. In Spring's M-Told condition, the warning signal was a "relevant" stimulus, since it forewarned the subject of the imperative stimulus to be presented. Therefore, in accordance with Sutton's formulation, it is not surprising that a modality shift effect was found, despite the fact that all subjects were

"certain" of the stimulus to be presented on each trial; schizophrenic patients were not able to benefit from (or, more correctly, to fully attend to) the "relevant" information that was conveyed by the warning signal. Furthermore, the imperative stimulus of the previous trial was "irrelevant," since subjects were not told that the purpose of the experiment was to investigate the effects of sequential changes in the modality of the stimulus. It follows that schizophrenic patients would be disproportionately affected by the stimuli of previous trials since, in Sutton's proposal, their attention is drawn to irrelevancy.

Different from Spring's ("relevant") warning signals, Rist's warning signals represent "irrelevant" stimuli, since they varied randomly and independently from the modality of the imperative stimuli. Thus, in accordance with Sutton's (Note 3) proposal, the warning signals in Rist's experiment would be expected to influence the reaction time performances of schizophrenic patients.

In conclusion, crossmodal reaction time experiments have offered little support for a simple neural trace theory explanation of the modality shift effect. As Sutton (Note 3) has indicated, his interpretation of the findings of Spring (1980) and Rist (Note 1) concerns a kind of "conceptual trace," as compared to the "sensory trace" reflected in Zubin's formulation. However, a comprehensive investigation of Zubin's theory has not been undertaken and, as Nuechterlein (1977) has noted, neural trace theory does seem to have adequate specificity for generating hypotheses that would distinguish it from other existing theories of schizophrenic reaction time performance anomalies.

Magnitude of Difference Hypothesis. Sutton, Spring, and Tueting (1978) have suggested that perhaps the "modality shift" effect is not specific to shifting attention across modalities, but instead can be explained by "magnitude of difference": i.e., perhaps schizophrenic patients are disproportionately affected by degree of stimulus difference on successive trials, rather than by trial-to-trial changes in stimulus modality. According to this hypothesis, the modality shift effect is found because light and sound stimuli "differ," whereas the identical stimuli employed on ipsimodal trials in (most) crossmodal reaction time experiments obviously do not. It follows that subjects should perform differently on ipsimodal-identical trials (e.g., a red light followed by a red light), than on ipsimodal-nonidentical trials (e.g., a red light followed by a green light). Furthermore, if ipsimodal-nonidentical stimuli are made "different enough," then ipsimodal retardation (i.e., mean reaction time on ipsimodal-nonidentical trials minus mean reaction time on ipsimodal-identical trials) should equal or exceed crossmodal retardation.

In three crossmodal reaction time experiments (Sutton et al., 1961; Sutton & Zubin, 1965, Experiments A and B), red and green lights, and high- and low-frequency tones were randomly presented to patients and controls. Results of all three experiments showed that, for both subject groups, reaction times to light and sound stimuli were fastest on ipsimodal-identical, slower on ipsimodal-nonidentical, and slowest on crossmodal trials. These data suggest that trial-to-trial changes of the stimulus (i.e., "difference") influence reaction time performance. In one of these experiments (Sutton & Zubin, 1965, Experiment A),

ipsimodal retardation to sound stimuli significantly discriminated schizophrenic patients from normal control subjects. This finding suggests that schizophrenic patients (compared to control subjects) are disproportionately slowed by "differences" between the stimuli of the previous, and of the present, trial.

In the only systematic investigation of the magnitude of difference hypothesis, Rist (Note 1) tested schizophrenic patients, alcoholic patients, and normal control subjects on crossmodal and ipsimodal-nonidentical blocks of trials (neither type of block included a warning signal). Crossmodal blocks included a light and a tone, whereas ipsimodal-nonidentical blocks included two tones differing in frequency. Results indicated that schizophrenics were significantly differentiated from alcoholics and normals on both types of blocks: i.e., compared to other subjects, schizophrenic patients exhibited disproportionate crossmodal and ipsimodal retardation. These results support a magnitude of difference hypothesis.

There are several possible reasons why Rist (Note 1) demonstrated a significant "ipsimodal-nonidentical retardation effect," whereas, Sutton et al. (1961) and Sutton and Zubin (1965, Experiment B) did not. First, in order to maximize "difference," Rist simultaneously varied frequency and spatial location. More specifically, for half of the subjects, the high-frequency tone was always presented to the right ear, and the low-frequency tone was presented to the left ear; for the remainder of the subjects, the localization of tones was reversed. The differential effects of ear of presentation have never been studied in any crossmodal reaction time experiment. In fact, with the single ex-

ception of Verleger and Cohen's (1978) experiment (in which the modality shift effect was not found), headphones were never employed. Instead, auditory stimuli were emitted from a loudspeaker that was positioned in front of the subject. In summary, differences in spatial localization, rather than in frequency, might have been the major determinants of the ipsimodal-nonidentical retardation demonstrated by Rist's schizophrenic patients. Furthermore, if this were the case, it is possible that ipsimodal and crossmodal retardation resulted from independent processes: i.e., perhaps schizophrenic patients are independently influenced by spatial localization and by stimulus modality changes. It seems that a more convincing demonstration of the magnitude of difference hypothesis would be to vary stimuli along a single stimulus dimension (e.g., frequency), and to observe the effects of ipsimodal-nonidentical differences along a continuum.

A second possible reason why Rist (Note 1) demonstrated significant ipsimodal retardation, whereas, Sutton et al. (1961) and Sutton and Zubin (1965, Experiment B) did not, concerns the number of different stimuli presented in each block of trials. Rist included high- and low-frequency tones in his ipsimodal blocks, whereas, Sutton et al. and Sutton and Zubin included high- and low-frequency tones and red and green lights. Rist included only two different stimuli in his ipsimodal blocks to avoid anchor effects. Perhaps the inclusion of "more different" stimuli in the experiments of Sutton et al. and Sutton and Zubin, attenuated the sequential effects of stimuli that were "relatively" less different. Similarly, perhaps the inclusion of only two stimuli in Rist's ipsimodal blocks accentuated the degree of sequential dif-

ferences by providing a greater contrast between ipsimodal-nonidentical stimuli. These observations do not detract from Rist's findings. They only point to a factor that might influence the degree of ipsimodal retardation demonstrated.

A third possible reason why Rist (Note 1) demonstrated significant ipsimodal retardation, whereas, Sutton et al. (1961) and Sutton and Zubin (1965, Experiment B) did not, concerns the frequency differences between tone stimuli used in the three experiments. Rist presented 200 Hz and 2000 Hz tones; Sutton et al. used 400 Hz and 1200 Hz tones, and; Sutton and Zubin employed 312 Hz and 1000 Hz tones. Note that Rist's tones were "most different" with respect to frequency. These observations suggest that, in support of a magnitude of difference hypothesis, a significant ipsimodal retardation effect was found by Rist because his ipsimodal-nonidentical stimuli were "sufficiently different." However, this interpretation is not supported by the findings of Sutton and Zubin (1965, Experiment A) who used 312 Hz and 1000 Hz tones (as in Experiment B), and who also found that ipsimodal-nonidentical retardation significantly differentiated schizophrenics and controls.

In conclusion, although the findings of Rist (Note 1) support a "magnitude of difference" explanation of the "modality shift" effect, several questions remain unanswered. In addition, since Rist's experiment was the only systematic investigation of ipsimodal-nonidentical retardation in schizophrenic patients, replication is needed.

Expectancy Hypothesis. According to expectancy hypothesis, schizophrenic patients and normal control subjects differ in their

expectations of the forthcoming stimulus (Sutton et al., 1978). More specifically, in a crossmodal reaction time task, patients "expect" that the same stimulus will be repeated on successive trials; thus, they are unprepared for a shift in modality and, consequently, exhibit disproportionate slowing on crossmodal trials.

There is convincing evidence that expectancy, alone, cannot account for the significant modality shift differences between patients and controls. Waldbaum et al. (1975, Certain condition) and Spring (1980) informed their subjects which stimulus would be presented on each trial, thus eliminating all uncertainty. The modality shift effect was still found in both of these experiments. Furthermore, in the Uncertain (i.e., uninformed) condition of Waldbaum et al.'s experiment, subjects guessed which stimulus (a light or a sound) would be presented on each trial. Correct-guess and incorrect-guess trials were analyzed separately. Results indicated that the modality shift effect was found only for trials associated with correct guesses. In addition, the guessing data showed that normal control subjects tended to guess, more often than patients, that the next stimulus would be ipsimodal. Both of these findings refute the hypothesis that schizophrenic patients are "more prepared for" (i.e., they expect) ipsimodal presentations of stimuli, and are "caught off guard" when a stimulus in another modality is presented.

Although expectancy cannot account for the modality shift effect, Waldbaum et al. (1975) provided evidence that expectancy (or, more

correctly, uncertainty)⁴ does influence the magnitude of crossmodal retardation exhibited by schizophrenic patients. For both modalities, the magnitude of retardation was larger in the Uncertain (i.e., uninformed), than in the Certain (i.e., informed), condition.

In summary, schizophrenic patients tend to show larger crossmodal retardation magnitudes when they are not forewarned of the modality of the forthcoming stimulus. However, a simple expectancy hypothesis cannot account for the significant differences in crossmodal retardation between patients and controls.

The current research was designed to investigate one possible explanation (based on principles of psychometric test theory) that could account for the significant modality shift differences between schizophrenic patients and normal control subjects, and that has never been explored in a crossmodal reaction time experiment. The basis of this explanation will be reviewed in the following section.

⁴A distinction is made, here, between expectancy and uncertainty. If a subject performs differently on Certain and Uncertain trials, one possible explanation is that he or she "expects" one stimulus more often than another. However, a second possibility is that uncertainty changes the state of the organism (e.g., increased arousal), and that this change results in differential performance. By obtaining pretrial guesses, Waldbaum et al. (1975) demonstrated that an expectancy theory could not explain the modality shift effect. However, uncertainty (or what it may lead to) does seem to influence the magnitude of crossmodal retardation in patients.

The Chapmans' Methodological Critique

Chapman and Chapman (1973a, 1973b, 1978a) have provided a methodological critique that has been directed against many areas of psychiatric research, including crossmodal reaction time experiments (Schuck, Leventhal, & Carbonell, 1978).

Generalized Performance Deficit. The Chapmans' methodological critique rests on the frequent observation that schizophrenic patients perform "more poorly" than normal control subjects on almost any experimental measure-- as a group, they recall fewer words on memory tasks, they are more influenced by distraction in dichotic listening experiments, they respond more slowly than normal controls on reaction time tasks, etc. As a result of the "poorer than normal" or "less adequate" performance of schizophrenic patients on a large number of tasks (the so-called schizophrenic "generalized performance deficit"), no special significance can be inferred from performance on any single experimental measure.

To circumvent the interpretive ambiguities resulting from the schizophrenic patient's generalized performance deficit, several investigators have employed a "differential deficit methodology" in which the performances of schizophrenic patients and normal controls are compared on two tasks, an experimental task and a control task. Schizophrenic patients are expected, due to their generalized deficit, to perform more poorly than normal controls on both measures. The hypothesis of these experiments is that schizophrenic patients will be differentially affected by the variable of the experimental task: i.e., they will show a greater performance deficit (relative to normal con-

trol subjects) on the experimental task, than on the control task.

Psychometric Artifact. The Chapmans' major objection to a differential deficit methodology is that unless experimental and control tasks are matched on their ability to discriminate "more competent" and "less competent" subjects (referred to as the "discriminating power" of a task), poorer performance on the experimental task simply may reflect the psychometric characteristics of the particular tasks chosen, coupled with the tendency for schizophrenic patients, as a group, to perform less competently. Stated differently, the schizophrenic patients' generalized performance deficit may be mistaken for a specific deficit in some hypothesized ability. The task yielding the greater dispersion of scores will show greater performance differences between subjects differing in general ability regardless of whom these subjects may be.

Certain statistical and methodological procedures have been used to avoid the problem of interpreting findings in light of the schizophrenic patient's generalized performance deficit. However, as the Chapmans point out, these procedures are flawed with interpretive difficulties of their own. Two of these approaches will be reviewed in the next section.

Unacceptable Solutions to the Problem of Generalized Deficit

The Analysis of Covariance. The analysis of covariance has been used to "partial out" patient-control differences in overall performance. In this procedure, the effects of an unwanted or "nuisance" variable (the covariate) are equalized across all levels of the independent variable. More correctly, in the estimation of error variance, that proportion of variance that can be attributed to linear dependency between covariate and dependent variable is removed.

In the usual experiment in psychiatric research, patient and control groups are compared on some measure, and subject group (e.g., schizophrenic and normal) is treated as an independent variable. The rationale for employing an analysis of covariance in these experiments is to "equate" subject groups on some potentially relevant, quantifiable measure (e.g., mean reaction time, pretest score on a vocabulary test) to assure that the results of the analysis are interpretable in terms of the effect of group membership (rather than initial or overall performance resulting from generalized deficit) on the dependent variable.

Several investigators have criticized the use of the analysis of covariance for comparing preformed subject groups. For example, Cochran (1957) has indicated that, when the covariate is causally affected by levels of the independent variable (i.e., if group differences in overall performance are reflecting genuine differences attributable to group membership), then the adjustment process of the analysis of covariance will remove more than what can be considered an error component from measures of the dependent variable. Stated differently, if

the covariate is affected by the treatments (either by direct causation, or through correlation with other variables), then part of the treatment effect will be removed when covariance adjustments are made. The F-value, in turn, will represent a biased estimate of the relationship between variances, in the direction of underestimating differences among subject groups and, thus, increasing the chances that a Type II error will be made.

For most experiments in which a natural-groups design is used, statements regarding correlation (as opposed to causation) are all that can be made. Thus, for example, in a reaction time experiment, it is not unlikely that "being a schizophrenic/not being a schizophrenic" (the "treatment effect") causes patient-control differences in mean reaction time (the covariate). Therefore, removing part of the variation of the dependent variable (by covariance adjustment) would distort the nature of the treatment effect, and render it uninterpretable.

Kahneman (1965) has discussed situations in which the results of an analysis of covariance would represent an overestimation of group differences and, thus, an increased probability of Type I error. According to Kahneman, whenever the covariate is measured with error (i.e., whenever some degree of unreliability exists), systematic population differences on the covariate will result in an undercorrection in the analysis of covariance and, thus, an increased probability of falsely rejecting the null hypothesis. Since all behaviors are measured with some error, and since there are systematic behavioral differences between schizophrenic patients and normal control subjects (i.e., patients perform more poorly than controls on most tasks),

Kahneman's discussion of bias in the analysis of covariance is pertinent to the use of this procedure in psychiatric research.

A detailed discussion of the specific mathematical and theoretical reasons why (and when) the analysis of covariance portrays a biased estimate of the treatment effect is beyond the scope of this report. The purpose of this section was to indicate that the general consensus of several investigators (e.g., Cochran, 1957; Fleiss & Tanur, 1973; Lord, 1967, 1969; Winer, 1971) is that the use of an analysis of covariance for removing the effects of pre-existing group differences is plagued with theoretical and interpretive difficulties. To cite Fleiss and Tanur (1973):

The overwhelming weight of logic is on the side of those who warn that neither the analysis of covariance nor any other statistical technique can undo systematic differences which were out of the investigator's control. (p. 518)

Matching Atypically Performing Subjects. Whereas some investigators have employed an analysis of covariance to partial-out group differences, other investigators have matched atypically performing "more competent" schizophrenic patients to atypically performing "less competent" normal control subjects to avoid the interpretive difficulties resulting from generalized deficit. This strategy is unacceptable for two reasons. First, "atypical" subjects may be atypical in ways other than their performance on the control task: i.e., a selection bias may be present. Thus, one's findings may also be "atypical," unrepresentative and misleading. Second, matched schizophrenic and control subjects (i.e., regarding their performances on the control task) who differ in their performances on the experimental task, simply may be ex-

hibiting statistical regression. Unless experimental and control variables correlate perfectly, extreme scorers on one measure will regress to the mean performances of their corresponding (schizophrenic and normal) groups when tested on a second measure (Minium, 1978). A more thorough explanation of statistical regression follows.

By applying the least-squares criterion (which minimizes the sum of squared deviations from the mean), Pearson derived the regression equation for predicting Y from X. In standard score form, the regression equation is:

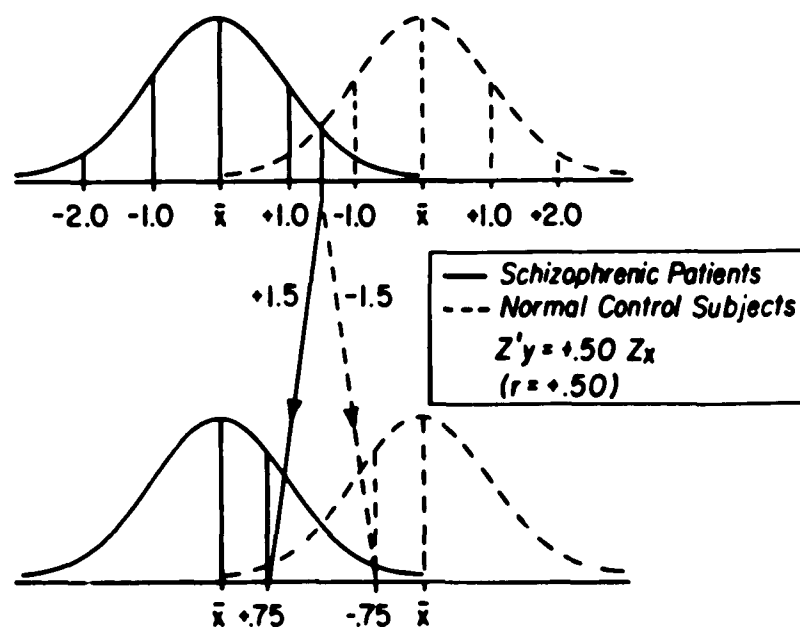
$$\underline{z}'_Y = r \underline{z}_X$$

where $\underline{z}'_Y$ is the predicted standard score value of Y, r is the coefficient of correlation between X and Y, and $\underline{z}_X$ is the standard score value of X from which $\underline{z}'_Y$ is predicted.

As the standard score form of the regression equation shows, the higher the value of r , the less the amount of regression. For example, individuals performing 1.0 standard deviation above the mean score of their group on Task A ($\underline{z}_X = +1.0$) will perform, on the average, : .9 standard deviation above the mean for Task B ($\underline{z}'_Y = +.9$) if the correlation between Tasks A and B is .9 ($r = +.90$); .5 standard deviation above the mean for Task C ($\underline{z}'_Y = +.5$) if the correlation between Tasks A and C is .5 ($r = +.50$); .2 standard deviation above the mean for Task D ($\underline{z}'_Y = +.2$) if the correlation between Tasks A and D is .2 ($r = +.20$); etc.

Figure 1 shows a hypothetical situation in which the correlation of scores on Tasks A and B is .5. In the example, schizophrenic pa-

Distributions Of (Obtained) Standard Scores On Task A



Distributions Of (Predicted) Standard Scores On Task B

Figure 1. Illustration of statistical regression to the mean. Distributions of obtained and predicted standard scores on Tasks A and B for schizophrenic patients and normal control subjects are shown. Atypically scoring patients and controls are matched on Task A. The correlation between Tasks A and B is $+.50$.

tients performing 1.5 standard deviations above the patient mean performance on Task A achieve scores comparable to normal control subjects performing 1.5 standard deviations below the normal mean performance on Task A. (The identical standard deviations for patients and controls are coincidental. Depending on the degree of overlap, the skewness, and the kurtosis of the patient and control distributions, for example, it could be that $\bar{z}_X = 1.8$ for patients would be comparable to $\bar{z}_X = -2.0$ for control subjects.) Note that atypically performing patients and controls both regress toward the means of their corresponding groups when tested on Task B.

In the context of a differential deficit methodology, the patient-control difference in performance on Task B may be misinterpreted as a genuine difference in some hypothesized ability, rather than simply as a demonstration of statistical regression to the mean. Alternatively, the Chapmans recommend that tasks, rather than individuals, be matched. Their recommended procedure for matching experimental and control variables on discriminating power will be reviewed in the next section.

Matching on Discriminating Power

According to Chapman (1979), the critical factors of the tasks to be matched are 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.

Before discussing the matching procedure, certain principles of psychometric test theory will be reviewed to elucidate some of the

points that have been made. Much of what follows is based on Chapman and Chapman (1978a).

Principles of Psychometric Test Theory. An obtained value (X_o) on a task conceptually may be divided into two components: a true score (X_t) and an error score (X_e). True score refers to that portion of the obtained score that is replicable or reliable. Error score reflects errors of measurement, which are assumed to occur randomly. Thus, for example, on a reaction time task, a subject may be very fatigued during the first day of testing, and fully rested on the second day; or, in a forced-choice paradigm, one subject may be very lucky in guessing, whereas another subject may be very unlucky; or, on a vigilance task, one subject may be alert and attentive, whereas another subject may be poorly motivated and not fully engaged in the task. In summary, errors of measurement (and, in turn, error variance) include any variable, or any interaction of variables, that are irrelevant to the purposes of the experiment (Anastasi, 1982).

In addition to the assumption of randomness, true scores and error scores are also assumed to be uncorrelated. Thus:

$$\underline{S}_o^2 = \underline{S}_t^2 + \underline{S}_e^2$$

where $\underline{S}_o^2$ is the variance of observed scores, $\underline{S}_t^2$ is the variance of true scores, and $\underline{S}_e^2$ is the variance of error scores.

It can also be shown (Nunnally, 1967) that:

$$\underline{S}_t^2 = \underline{r} \underline{S}_o^2$$

where $\underline{r}$ is the reliability of the test.

To recapitulate, both reliability and observed-score variance are needed to estimate true-score variance. Observed-score variance, alone, is insufficient since errors of measurement contribute to observed scores.

Discriminating Power of a Test. A hypothetical example will be used to illustrate how tests differing on true-score variance can lead to false inferences concerning differential deficit. Figure 2 shows the frequency distributions of true scores for (normal) subjects who have taken Tests 1 and 2. Note that the variance of Test 1 is much larger than the variance of Test 2. The frequency distributions of Tests 1 and 2 have been divided at the median to form subsamples of the performances of "more competent" and "less competent" scorers on each test. (For the purposes of this illustration, assume that the error variances of the subsamples for each test are identical.) The locations of the means for each subsample are shown above the two distributions. Note that the difference between the means of the low and the high scorers on Test 1 ($\bar{X}_{L1} - \bar{X}_{H1}$) is greater than the difference between the means of the low and the high scorers on Test 2 ($\bar{X}_{L2} - \bar{X}_{H2}$). In summary, the greater the true-score variance of a test, the greater its power to discriminate between more competent and less competent subjects-- tests have identical "discriminating power" when their true-score variances are identical.

It should now be apparent that, if schizophrenic patients ("less competent" subjects, or low scorers) and normal control subjects are tested on two tasks, the task with the greater true-score variance will yield a larger patient-control difference in performance. This dif-

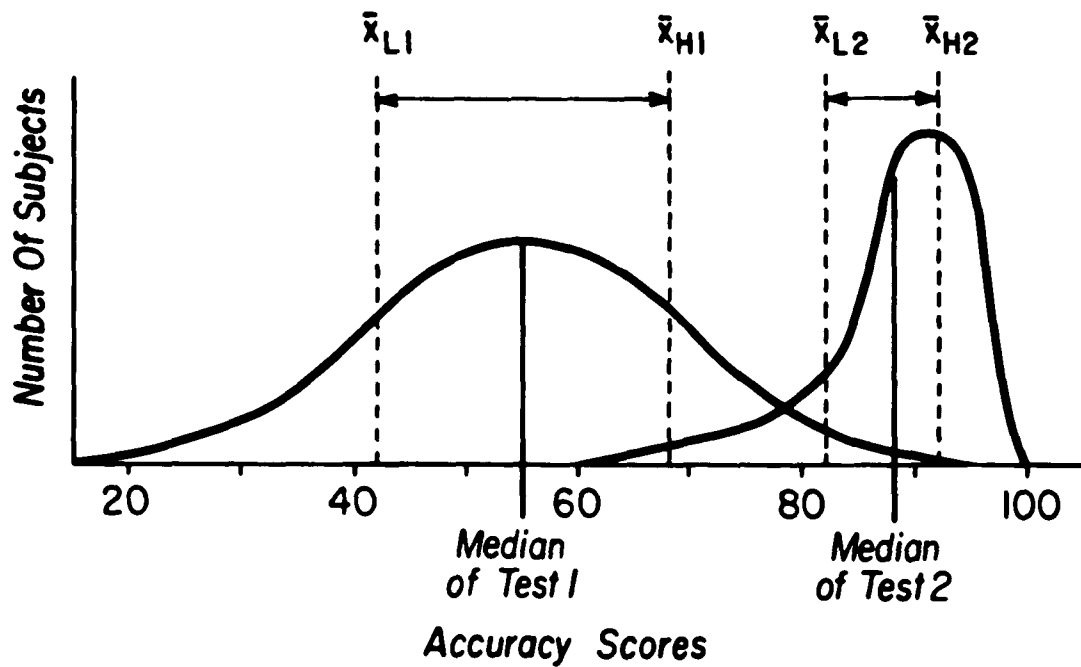


Figure 2. Illustration of two tests differing in discriminating power. Distributions of true scores on Tests 1 and 2 are shown. Each distribution is divided at the median. The means of the half-distributions of low scorers ($\bar{x}_{L1}$, $\bar{x}_{L2}$) and of high scorers ($\bar{x}_{H1}$, $\bar{x}_{H2}$) are shown above the figures.

ference can be explained without resorting to hypothesized, specific deficits-- generalized deficit, coupled with the reliability and the observed-score variances of the tests, are sufficient to account for the difference.

The Matching Process. Matching tasks on discriminating power may be achieved by manipulating a variable known to affect accuracy of performance, since both reliability and (observed-score) variance are heavily influenced by difficulty level: both measures increase with departures from zero difficulty (e.g., an easy test in which all subjects obtain a perfect score) and 100% difficulty (e.g., an impossible test in which all subjects fail all items).

Concerning the choice of a reliability estimate to be used in the matching process, several reliability coefficients have been described (see Anastasi, 1982). The Chapmans recommend coefficient alpha for use when reliability is to be estimated from a single test administered in a single setting. Coefficient alpha, a measure of internal consistency, has been shown (mathematically) to be the mean of all split-half reliability coefficients resulting from different splittings of the same test (Cronbach, 1951). When items are dichotomously scored (pass-fail), coefficient alpha is equivalent to Kuder-Richardson Formula 20.

The standardization sample used for matching tasks on reliability and variance should consist of a large number of normal control subjects of a wide range of "competence," a substantial proportion of whom perform as poorly as schizophrenics. Patients should not be included in the standardization sample since any true differential deficit in ability would affect the amount of variance on the two tasks. The equiva-

lence of the matched tasks should be verified by recalculating reliability and variance for a subgroup of the standardization sample that scores at the level of the patient group. The purpose of this final procedure is to assure that the two tasks not only are equated on the degree to which they discriminate more competent from less competent subjects, but also on the degree to which they discriminate less competent subjects demonstrating different levels of competency.⁵

The Chapmans contend that criticisms of psychometric artifact pervade nearly all areas of psychiatric research. However, Carbotte (1978) and Strauss (1978) have contested the broad applicability claimed by the Chapmans for their treatise. Each of these investigator's commentaries, and the Chapmans' reactions to these criticisms, follow.

Commentaries on the Chapmans' Critique

Matched Tasks or Converging Operations? According to Carbotte (1978), two different orientations towards the study of overt behavior can be distinguished: one involving the assessment of individual differences, and the other concerning the identification or measurement of processes. The Chapmans' methodological recommendations most appropriately apply to the first of these approaches. Conversely, in the

⁵It should be noted that the Chapmans have indicated situations for which matching on discriminating power is not needed: (1) if two pathological groups perform in different directions; (2) if the control task has a larger true-score variance than the experimental task; and, (3) if a non-pathological population that typically exhibits "generalized deficit" (e.g., the elderly), performs differently from schizophrenics on the experimental task.

latter approach (which may be referred to as the "functional" orientation), the primary focus is on "identifying lawful relationships between stimuli and responses in spite of individual differences in levels of performance." (p. 313) Carbotte avers that employing a matched control task in these studies may result in serious confounding and, in turn, interpretive difficulties could prevail. According to Carbotte, process-oriented investigations should use a converging operations design, in which different methodologies are employed to illuminate one hypothesis, and to refute competing hypotheses.

Carbotte also questions the conceptualization and purpose of a "control task" (as the Chapmans use the term) in the context of a functional orientation. According to Chapman and Chapman, a control task differs from the experimental task in that the former does not involve the "ability" assessed by the latter. Carbotte objects to this definition and, using the example of a span of apprehension paradigm, contends that performance on all conditions (one element or multi-element) involves the same processes. Furthermore, in processing experiments, a "control task" may be represented by a condition that allows the investigator to rule out the influence of certain problematic factors often encountered in patient research (e.g., poor motivation).

In conclusion, Carbotte contends that a matched-tasks methodology is not suitable for process-oriented investigations, and that the use of this research design may preclude the discovery of lawful relationships that would otherwise be observed. Carbotte advocates the use of a converging operations design as the most appropriate research strategy for process-oriented studies.

The Chapmans' Reaction to Carbotte. According to Chapman and Chapman (1978b), regardless of the theoretical beliefs of the investigator, or the number of experiments that are purported to measure a particular construct or "process," valid inferences cannot be made about the results of any single study unless psychometric artifact has been ruled out. Stated differently, five or ten or twenty equally flawed experiments cannot be offered in support of whether a particular finding is genuine. Furthermore, regardless of the terminology used to describe an experiment ("control task," "process," "ability," "individual differences," etc.), if two conditions are employed, the one yielding the greater dispersion of scores will display a greater difference between the performances of "more competent" and "less competent" subjects.

Between-Group or Within-Group Comparisons? Similar to Carbotte (1978), Strauss (1978) distinguishes between two experimental approaches to the study of behavior: the individual differences paradigm (which relies on subject group X task interactions) and the experimental paradigm (in which within-group, rather than between-group, performance is the principal focus). Strauss contends that, although the individual differences paradigm (for which psychometric matching is most appropriate) may yield reliable differences between certain groups administered certain tasks under certain conditions, the experimental paradigm is, first, needed to provide an understanding of which processes are under study.

According to Strauss, before a differential deficit in an ability or process can be inferred from a subject group X matched tasks inter-

action, two assumptions must be made: (1) the ability or process being assessed is known, and; (2) the test assesses the same ability in different subject groups. However, in most experiments with schizophrenic patients, the "process" purported to be under investigation is not known, and any one of a number of theories can be invoked to account for the same findings. Further, Strauss contends that it cannot be assumed that a particular task which measures one process in normal control subjects necessarily measures the same process in schizophrenic patients. Strauss advocates the use of an experimental paradigm since the individual differences paradigm can only yield theoretically ambiguous results until proposed processes are thoroughly understood.

The Chapmans' Reaction to Strauss. To recapitulate, Carbotte (1978) and Strauss (1978) both contend that, when the primary focus of the experiment is to evaluate a "process," a matched-tasks design should not be considered the preferred paradigm. However, these investigators recommend somewhat different, alternative approaches. Whereas Carbotte advocates the use of converging operations in the framework of an experimental paradigm whenever a process is being evaluated, Strauss proposes that within-group, rather than schizophrenic-normal, comparisons be made until more is learned about the process.

Chapman and Chapman's (1978b) reaction to Strauss' recommendation is that matched tasks should first be used to determine whether psychometric artifact can explain the phenomenon under study. Otherwise, a great deal of time and effort may be devoted to within-group comparisons that are not worth pursuing. Thus, they contend, Strauss' strategy seems impractical with regard to advancing our knowledge of schizo-

phrenia with any degree of expediency.

Most discussions of the Chapmans' treatise have concentrated on paper and pencil procedures (e.g., multiple choice tests), or tasks in which adequacy of performance has been defined within some restricted range of values (e.g., 0-100% correct on a free recall task). However, performance on a reaction time task can also be placed in the framework of the Chapmans' critique.

Psychometric Artifact and the Modality Shift Effect

An analogy can be made between paper and pencil tests and reaction time tasks, in which accuracy of performance ("competence") is defined in terms of latency of response: the faster the subject's mean reaction time on a given condition, the "better" his or her performance. In a crossmodal reaction time task, crossmodal trials may be conceptualized as forming the experimental task, and ipsimodal trials as forming the control task. In view of their generalized performance deficit, schizophrenic patients, as a group, are expected to perform "less accurately" (i.e., their reaction times are expected to be slower than those of normal control subjects) on both the crossmodal condition and the ipsimodal condition.

Regarding the Chapmans' methodological treatise, the criticism of crossmodal reaction time experiments is that perhaps the schizophrenic patients' disproportionate (greater than normal) slowing on crossmodal trials (i.e., the modality shift effect) may be explained by their generalized deficit, coupled with the greater discriminating power of the crossmodal condition. More explicitly, perhaps when the stimulus of

the preceding trial is in a different modality than the stimulus of the present trial (i.e., the crossmodal condition), the dispersion of mean reaction times of a representative sample of normal subjects (including both fast responders and slow responders) is substantially greater than the dispersion of reaction times obtained when the modality of the stimulus of the previous trial is the same as the modality of the stimulus of the present trial (i.e., the ipsimodal condition). If this situation were true, then a larger performance difference between faster ("more competent") and slower ("less competent") subjects would be expected on crossmodal trials, than on ipsimodal trials, regardless of whom these subjects may be (normal control subjects, schizophrenic patients, elderly subjects, mental retardates, neurological patients, etc.).

To control for the possibility that differing dispersions of reaction times of normal control subjects are accounting for schizophrenic-normal differences, crossmodal and ipsimodal trials must be equated on the degree to which they discriminate between fast and slow responders (i.e., discriminating power). Matching may be achieved by manipulating a variable known to affect response latency ("accuracy of performance"). For example, stimulus intensity, preparatory interval, or probability of stimulus occurrence could be manipulated until reliability and variance (critical factors in the estimation of discriminating power) are equated on crossmodal and ipsimodal trials for normal control subjects. After matching is achieved, patient-control comparisons can be made on equated tasks, now free of criticisms regarding psychometric artifact.

Overview of the Current Research

Artifactual findings pervade all areas of psychiatric research, including biochemical studies (Kety, 1975), studies of electrodermal habituation (Iacono & Lykken, 1979), evoked potential research (Sutton & Tueting, 1978), and investigations of critical flicker frequency (Clark, Brown, & Rutschmann, 1967), to name only a few. It follows that any viable interpretation in terms of artifact must be explored if research is to progress. This point is particularly true of the current research since all of the previous crossmodal reaction time experiments are susceptible to an interpretation in terms of psychometric artifact.

The present research was designed to assess whether the modality shift effect could be attributed to psychometric artifact. Crossmodal and ipsimodal conditions were intended to be matched on discriminating power by manipulating intensity, since several experiments have shown a systematic relationship between response latency and intensity over a wide range of values, and for both auditory and visual stimuli (King, 1962; Teichner & Krebs, 1972; Woodworth & Schlosberg, 1954).

A semi-structured psychiatric interview was administered to hospitalized, male psychiatric patients, and only those patients who were given a Definite (as opposed to Suspected, or Probable) diagnosis of Schizophrenic Disorder (based on specific diagnostic criteria) were tested. Normal control subjects were administered a comprehensive semi-structured interview to screen for a past or present episode of mental disorder. Only those individuals who were judged to be "Never Mentally Ill" (based on specific criteria) were recruited for testing. The final

sample consisted of 15 schizophrenic patients and 50 normal control subjects.

It is noteworthy that only two previous investigators of the modality shift effect employed semi-structured interviews and diagnostic criteria to screen their prospective normal control subjects. When specific criteria were applied, Waldbaum et al.'s (1975) initial normal control sample of 22 subjects was reduced to 11 subjects, and Spring's (1980) initial sample of 19 subjects was reduced to 15 subjects (Spring, Note 4). Thus, the current research was the only experiment in which a large group of individuals judged to be not mentally ill (by specific criteria) was tested.

All subjects were tested on a simple crossmodal reaction time task, and were instructed to make the identical finger-lift response on all trials. There were 10 intensities of visual stimuli and 10 intensities of auditory stimuli.

Four stages of data analyses were planned: (1) using the data from all subjects and all intensities, confirm the modality shift effect; (2) using the data of normal control subjects, systematically delete reaction times to stimuli of certain intensities until cross-modal and ipsimodal conditions are matched on reliability (as measured by coefficient alpha) and variance; (3) confirm the matching on the data of a subgroup of normals whose reaction times are within the patient range; and, (4) using the data of patients, delete those trials (corresponding to certain intensities) that were deleted to achieve a matching (Stage 2), and compare patients and controls on matched conditions. A demonstration of the modality shift effect on matched

conditions would refute an explanation in terms of psychometric artifact.

CHAPTER II

METHOD

Subjects

Overview of Subject Groups. The 65 subjects who participated in the present experiment were 15 schizophrenic patients hospitalized at the New York State Psychiatric Institute, and 50 normal control subjects who were judged to be Never Mentally Ill on the basis of the Research Diagnostic Criteria (RDC: Spitzer, Endicott, & Robins, 1978). All subjects were males between the ages of 18 and 49 years.

Demographic characteristics of patient and control groups, and the results of tests of differences between groups on each demographic variable, are shown in Table 1. (Individual subject data are presented in Appendix A.) There was no significant difference between groups in age, education, or ethnicity.

All patients fulfilled RDC criteria for a Definite (as opposed to a Probable) diagnosis of Schizophrenia. (RDC criteria for Schizophrenia and its subtypes are presented in Appendix B.) Patient information regarding diagnostic subtypes, drug dosage, and hospitalization is shown in Table 2. (Individual patient data are presented in Appendix C.) All patients were taking some form of neuroleptic (antipsychotic) medication. With respect to category subtype, about half of the patients were Paranoid, and the remainder were Undifferentiated or Disorganized. Patients were approximately evenly distributed among RDC chronicity

Table 1
Demographic Characteristics of Schizophrenic Patient
and Normal Control Subject Groups

Variable	Group		Difference ^a
	Patient	Control	
	(<u>N</u> = 15)	(<u>N</u> = 50)	
Age (years)			
Range	18-48	18-49	
Mean	29.6	29.3	
<u>SD</u>	8.2	7.8	<u>t</u> (63) = .15
Education (years)			
Range	8-16	9-19	
Mean	11.9	12.7	
<u>SD</u>	2.5	2.6	<u>t</u> (63) = 1.14
Ethnicity (<u>n</u>)			
White	7	27	
Hispanic	5	13	
Black	3	10	<u>x</u> ² (2) = .30

^aNone of the comparisons were significant.

Table 2
Diagnostic, Drug, and Hospitalization Details
of the Schizophrenic Patient Group

RDC Category Subtype	<u>N</u>
Paranoid	7
Undifferentiated	5
Disorganized	3
RDC Chronicity Subtype	
Acute	4
Subacute	4
Subchronic	4
Chronic	3
Previous Hospitalizations	<u>number</u>
Range	0 - 30
Mean	6.2
<u>SD</u>	7.9
Duration of Hospitalization	<u>days</u>
Range	2 days - 10 years
Mean	329.3
<u>SD</u>	929.0
Daily Drug Dosage	<u>mg/kg body weight^a</u>
Range	7.3 - 49.9
Mean	19.4
<u>SD</u>	12.8

^aDaily dosages of different neuroleptic medications were translated into equivalent dosages of Chlorpromazine (Thorazine) in accordance with a table presented in Davis (1976).

subtypes.

Recruitment and Evaluation of Patients. Hospital charts on the two cooperating wards of the Psychiatric Institute were checked twice a week to determine whether any males between the ages of 18 and 50 years had been admitted with a diagnosis of Schizophrenia. If a patient's chart contained any evidence of a chronic, ongoing history of drug or alcohol abuse, of mental retardation, of organic brain damage, or of any visual or auditory defects, the patient was automatically excluded from recruitment. In addition, a patient was not recruited if there was sufficient information in his chart to substantiate an RDC diagnosis of Schizoaffective Disorder, or a DSM-III diagnosis of Brief Reactive Psychosis, Paranoid Disorder, or Atypical Psychosis. Finally, a patient was excluded if only residual symptoms were indicated (i.e., if he was not currently exhibiting psychotic symptoms).

The charts of 54 male patients with a hospital diagnosis of Schizophrenia were reviewed. Of these 54 patients, 15 (28%) were tested in the present experiment. The remainder were excluded for the following reasons: (1) Diagnosis other than Schizophrenia ($N = 20$). Symptomatology indicated in the hospital chart suggested a diagnosis of Schizoaffective Disorder for 11 patients, Atypical Psychosis for 4 patients, Paranoid Disorder for 3 patients, and Brief Reactive Psychosis for 2 patients. (2) Substance abuse/substance-induced psychosis ($N = 14$). The hospital charts of these patients indicated either a chronic, ongoing history of alcohol or drug abuse (with a superimposed episode of Schizophrenia), or a substance-induced episode of psychosis. In some cases, it could not be determined which of these two categories was

most accurate. (3) Visual defect ($N = 2$). One patient had an eye infection, and the other patient was blind in one eye. (4) Organic impairment unrelated to substance abuse ($N = 1$). This patient showed positive findings on the neurological examination and (according to the treating physician) a history suggesting multiple sclerosis. (5) Presently not exhibiting psychotic symptomatology ($N = 1$). This patient was given a hospital diagnosis of Residual Schizophrenia. He had been hospitalized several times in the past, but currently was only manifesting residual symptoms (e.g., social isolation). The patient denied hallucinations and delusions (to the admitting psychiatrist), and spoke coherently. (6) Untestable ($N = 1$). This patient was markedly agitated, and could not perform the reaction time task. He was transferred to another facility shortly after testing was attempted.

All patients who had not been excluded on the basis of the hospital chart review, were administered the Schedule for Affective Disorders and Schizophrenia (SADS: Endicott & Spitzer, 1978) by the experimenter. Patients were recruited as subjects only if, on the basis of SADS ratings, RDC criteria for Definite Schizophrenia were fulfilled. It is important to emphasize that the SADS was not scored on the basis of information in the hospital charts. For example, "loose associations" was indicated in nearly every chart, but was frequently not observed during the SADS evaluation. When this symptom was not observed by the experimenter (who often met with the patient on a number of occasions), it was not scored. There were several reasons for adopting this coding strategy: e.g., (1) the admitting psychiatrist's use of certain diagnostically significant terms might not conform to the RDC's use of the

terms; (2) the admitting psychiatrist might have observed and noted transient symptoms that were only present at the height of a confusional state, and; (3) in some cases, the period of time between date of admission and date of interview was years. In conclusion, the primary purpose of administering the SADS was to provide a systematic means for evaluating and quantifying current mental status.

Symptomatology of the Patient Group. Table 3 shows the patient group averages on each of eight summary scales (Endicott & Spitzer, 1978), based on the ratings of SADS items. (The composition of each scale is presented in Appendix D. Individual patient data are presented in Appendix E.) Averages were based on Percent Maximum Possible Scores (i.e., the scale score divided by the range of that scale) since the number of items contributing to each scale, and the range of each item, differ across scales.⁶ Percent Maximum Possible Scores for each subject range from 0 (i.e., every item contributing to the designated scale was scored as absent) to 100 (i.e., every item contributing to the designated scale was given the maximum, or most extreme, score).

As shown in Table 3, Scale 7 (Delusions and Hallucinations) yielded the largest mean score, and every patient was given a positive score on this scale. In addition, 4 patients attempted suicide during their present episode of illness (see Scale 4), and 6 patients exhibited formal thought disorder (see Scale 8). Finally, most patients reported some depressive symptomatology (see Scales 1-3).

⁶Dr. Jean Endicott (Research Assessment and Training Unit, New York State Psychiatric Institute) suggested the use of the Percent Maximum Possible Score since it is easily interpreted, and since other investigators have made use of it.

Table 3

SADS Summary Scale Scores for the Schizophrenic Patient Group

Scale Number and Name	<u>Percent Maximum Possible Scores</u>			Number of Patients with Non-Zero Scores
	Range	Mean	<u>SD</u>	
1 Depressive Mood and Ideation	0.0-53.8	19.0	16.7	12
2 Endogenous Features	0.0-25.5	12.0	8.4	13
3 Depressive- Associative Features	0.0-27.0	11.5	7.5	14
4 Suicidal Ideation and Behavior	0.0-63.0	14.3	24.8	4
5 Anxiety	0.0-42.3	9.0	12.2	8
6 Manic Syndrome	0.0-16.4	6.2	6.6	8
7 Delusions and Hallucinations	26.5-59.2	43.5	8.8	15
8 Formal Thought Disorder	0.0-40.8	8.6	14.5	6

Note. There were 15 patients in the schizophrenic group. Each patient received a score ranging from 0 to 100 on each Summary Scale.

The International Pilot Study of Schizophrenia (IPSS: World Health Organization, 1973) is the largest transcultural psychiatric investigation that has every been conducted. The IPSS involved the psychiatric assessment of 1,202 hospitalized mental patients in nine countries. Patients were administered a semi-structured psychiatric interview (comparable to the SADS, which was used in the current research), and the same inclusion and exclusion criteria were used by psychiatrists in all countries to formulate a psychiatric diagnosis. Of the 1,202 patients, 81 were excluded because of ambiguous diagnoses. The remaining 1,121 patients consisted of 811 patients with a Definite diagnosis of Schizophrenia, and 310 patients with diagnoses other than Schizophrenia.

Based on the ratings of the semi-structured interviews used in the IPSS, a stepwise discriminate function analysis was employed to determine the most diagnostically discriminating symptoms (Carpenter, Strauss, & Bartko, 1973). A number of symptom groups (e.g., Schneider's first rank symptoms) were tested for differentiating schizophrenics from nonschizophrenics, but the most effective group was comprised of 12 symptoms. These symptoms, and the number of patients in the current research who showed each symptom, are presented in Table 4. Note that all patients of the current research showed lack of insight (Symptom 2) and all but one patient was characterized by widespread delusions (Symptom 8). These findings are not surprising since all patients of the current research were exhibiting psychotic symptoms at the time of testing, and all were sufficiently ill to warrant hospitalization. Table 4 also shows that only three patients reported terminal

Table 4

Number of Patients in the Current Research
Who Showed Each of the Discriminating Symptoms
of the International Pilot Study of Schizophrenia^a

Symptom Number	Symptom	Number of Patients ^b
1	Restricted Affect	5
2	Lack of Insight	15
3	Thought Broadcasting	5
4	Absence of Terminal Insomnia	12
5	Poor Rapport	4
6	Absence of Depressed Facies	14
7	Absence of Elated Mood	15
8	Widespread Delusions	14
9	Incoherent Speech	6
10	Unreliable Information	7
11	Bizarre Delusions	9
12	Nihilistic Delusions	2

^aWorld Health Organization, 1973

^bThere were 15 schizophrenic patients in the current research.

insomnia (Symptom 4), only one patient exhibited depressed facies (Symptom 6), and no patient was elated (Symptom 7) when interviewed. These three symptoms are all characteristic of an affective syndrome, and schizoaffective patients were not recruited as subjects in the current research.

Table 5 shows the percentage of schizophrenic and nonschizophrenic patients in the IPSS who exhibited different numbers of the 12 discriminating symptoms. Note that the greater the number of symptoms, the smaller the number of IPSS nonschizophrenic patients (i.e., the fewer the number of false positive diagnoses of schizophrenia). Table 5 also shows the percentage of patients in the current research who exhibited different numbers of the 12 discriminating symptoms. All patients of the current research showed at least 6 symptoms (IPSS false positive rate = 4%), and most patients showed at least 7 symptoms (IPSS false positive rate = 1%). These findings provide corroborating evidence that patients of the current research fulfilled relatively stringent criteria for schizophrenia.

It is important to note that none of the 12 discriminating IPSS symptoms are necessary for an RDC diagnosis of Schizophrenia. In fact, most of these symptoms are not included among any of the RDC criteria for Schizophrenia. In addition, the above comparisons with IPSS findings were based on SADS ratings, and were conducted after patients of the current research were recruited for testing. Therefore, comparisons with IPSS findings represent post-hoc analyses that were unrelated to the selection process of the current research.

Table 5

Percentage of Schizophrenic and Nonschizophrenic Patients
in the International Pilot Study of Schizophrenia (IPSS)^a,
and Percentage of Patients in the Current Research,
Who Showed Differing Numbers of IPSS Discriminating Symptoms

Number of Symptoms (<u>N</u> = 12)	IPSS Patient Groups		Patients of the Current Research (<u>N</u> = 15)
	Schizophrenic (<u>N</u> = 811)	Nonschizophrenic (<u>N</u> = 310)	
4 or more	91	28	100
5 or more	80	13	100
6 or more	66	4	100
7 or more	44	1	67
8 or more	23	0	27
9 or more	? ^b	0	20
10 or more	? ^b	0	7

^aWorld Health Organization, 1973

^bNot reported by Carpenter, Strauss, and Bartko (1973)

Recruitment and Evaluation of Normal Control Subjects. Normal control subject were recruited by a variety of means. Subjects who had participated in other experiments were contacted, announcements were made at hospital senior staff meetings, hospital personnel (e.g., mental hygiene therapy aides, elevator operators, security guards) were notified, and friends of the experimenter were asked.

All prospective control subjects were administered the Schedule for Affective Disorders and Schizophrenia-Lifetime Version (SADS-L: Spitzer & Endicott, 1978) by the experimenter. Individuals who provided evidence of either a past or a present episode of mental disorder (which includes Alcoholism and Drug Use Disorder) were not tested. Only persons judged to be Never Mentally Ill (on the basis of the RDC) were recruited as normal control subjects.

Only 4 (of the 54) prospective normal control subjects were not tested on the basis of the interview findings: one person presented a questionable past history of affective disturbance, two persons fulfilled RDC criteria for (Probable) Drug Use Disorder, and one person fulfilled RDC criteria for Alcoholism. An exclusion rate of less than 8% is low compared to other studies. When specific criteria were applied, Waldbaum et al.'s (1975) initial normal control sample of 22 subjects was reduced to 11 subjects (exclusion rate = 50%), and Spring's (1980) initial sample of 19 subjects was reduced to 15 subjects (exclusion rate = 21%). Probably, the main reason for the low exclusion rate of the present experiment is that most subjects were partially "pre-screened" by the selection process. More specifically, subjects who had participated in other experiments had been evaluated at least once

before, which reduced the probability that a mental disorder would be indicated during the present interview. In addition, many subjects were long-standing, close friends of the experimenter, and Alcoholism, Drug Use Disorder, and Antisocial Personality Disorder were known to be absent before the standard questions on these disorders were asked. In conclusion, it is not surprising that the exclusion rate was relatively low since sampling was not random (nor could it have been random, since matching on certain demographic characteristics was a concern).

All normal control subjects were paid \$5.00 an hour for participating in the present research.

Rationale for Administering Different Interviews to Patients and Controls. The rationale for administering the SADS to patients, and the SADS-L to prospective normal control subjects concerns the content, organization and intended purposes of these schedules. The SADS consists of two sections. Part I focuses on details of the current episode, and Part II focuses on previous psychiatric history. The SADS-L is a briefer schedule that is similar to Part II of the SADS, except that the time period covered in the SADS-L includes any current disturbance. Thus, the SADS-L is most suitable when the respondent is not known to have a current mental disorder, or when a detailed description of the current episode is not needed. Therefore, the SADS-L was appropriately chosen as the screening instrument used in the current research.

Apparatus

General Description of Equipment. A diagram of the reaction time

apparatus is shown in Figure 3. The subject was seated at a wooden table whose surface was 44.0 inches (111.8 cm) in length, 41.0 inches (104.1 cm) in width, and 28.5 inches (72.4 cm) in height (distance from the floor to the table surface). A rectangular, closed stimulus box was positioned directly in front of the subject at a distance of 16.5 inches (41.9 cm) from the front of the table. The stimulus box was 19.75 inches (50.2 cm) in height, 9.5 inches (24.1 cm) in width, and 22.25 inches (56.5 cm) in depth. The front panel of the box was made of black plexiglass; the side panels, top, and base were made of wood. The front panel contained a circular opening (1 inch in diameter) located 5.25 inches (13.3 cm) from the top of the panel, and 13.5 inches (34.3 cm) from the surface of the table. A rectangular piece of satin finish ground glass (Edmund Scientific Company No. 2144) covered the circular opening, and acted as a diffusing plate. Four dim, red fixation lights were situated .25 inch (.6 cm) from the border of the circle, at radial loci 0° , 90° , 180° , and 270° (see Figure 3).

The stimulus box contained a filter holder and a loudspeaker, both of which were centered with respect to the width of the box, and positioned at the height of the circle on the front panel. The end slot of the filter holder (i.e., the slot furthest from the subject) was 2.5 inches (6.4 cm) from the front panel. The filter holder contained a 2.0 log unit neutral density filter, a .2 log unit neutral density filter, and (in the end slot) eight yellow (585 nm), diffused, light-emitting diodes (Monsanto No. MV 5353), which were mounted on a rectangular piece of plastic, and arranged in a circular pattern, with seven surrounding LED's and one center LED.

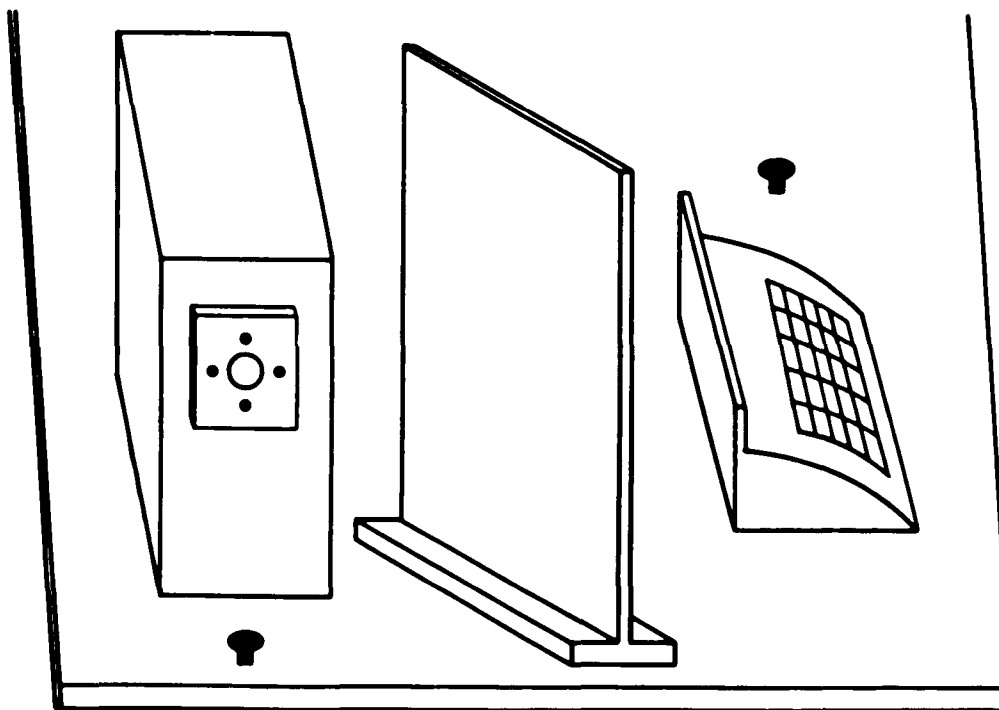


Figure 3. Diagram of reaction time apparatus, showing: (1) a rectangular stimulus box, whose front panel contains four fixation lights and a circular opening covered by a piece of ground glass; (2) a (subject) response key; (3) a wooden board, which prevented the subject from seeing the experimenter; (4) an AIM microcomputer (right); and, (5) an experimenter-abort key (to the right of the AIM).

The response key was located directly in front of the subject, 11.0 inches (27.9 cm) from the front of the table.

The experimenter was seated at the side of the table to the subject's right. A wooden board 35.5 inches (90.2 cm) in height, 19.0 inches (48.3 cm) in width, and .25 inch (.6 cm) in thickness stood upright, between the experimenter and the subject (see Figure 3), and prevented the subject from seeing (and possibly being distracted by) the experimenter during testing.

An AIM 6502 microcomputer (Rockwell International Corporation) was positioned on the table, directly in front of the experimenter. An experimenter-abort key was mounted on the surface of the table, approximately 6 inches to the right of the AIM (see Figure 3).

All remaining equipment (to be discussed) rested on a shelf located beneath the table and walled off from the subject's feet.

Regulation of Events During a Block of Trials. The entire sequence of events during a block of trials (e.g., determination of which stimulus to deliver on each trial, presentation of stimuli, variation of interstimulus interval, regulation of stimulus duration, replacement on "miss" and "anticipation" trials, registration and storage of reaction times) was automatically controlled by the AIM. The only exception was that the experimenter was able to abort a trial (e.g., because the subject turned his head away, or left his chair) by depressing the experimenter-abort key any time from stimulus onset to 1 sec after the subject's response. The computer program written for the present research was stored on a cassette tape, and fed into the AIM at the beginning of each session.

A table of 8-bit binary code values corresponding to each stimulus was stored in the memory of the AIM. As shown in Figure 4, the AIM was connected in common with two programmable attenuators. On each trial, 8 lines communicated the binary values for stimulus intensity, and 1 line communicated the binary value for stimulus modality (auditory or visual). Attenuator A (Auditory) received input from an oscillator (Wavetek, Model 136) which generated a 2000 Hz sinusoidal wave. Following attenuation, the auditory signal was passed through an amplifier and delivered to the subject through a loudspeaker. Attenuator V (Visual) received input from a reference voltage (which is the analog of the oscillator). Following attenuation of the visual signal, a current source activated the light-emitting diodes, which illuminated the ground glass that covered the circular opening on the front panel of the stimulus box.

Stimuli, Auditory Noise Level, and Background Illumination. Auditory stimuli were 2000 Hz tones, 10 msec in duration. Visual stimuli were 10 msec flashes of light that uniformly illuminated the ground glass covering the circular opening on the front panel of the stimulus box. (When asked, subjects reported seeing a single, white, circular flash, rather than 8 separate lights.) The visual stimuli appeared at a viewing distance of approximately 16.5 inches (41.9 cm) from the subject, and subtended a visual angle of 3.9 degrees.

The 50% detection thresholds for light and sound stimuli were determined for one normal subject (see Determination of Detection Thresholds). These values were used to determine the intensity values employed in the experiment proper.

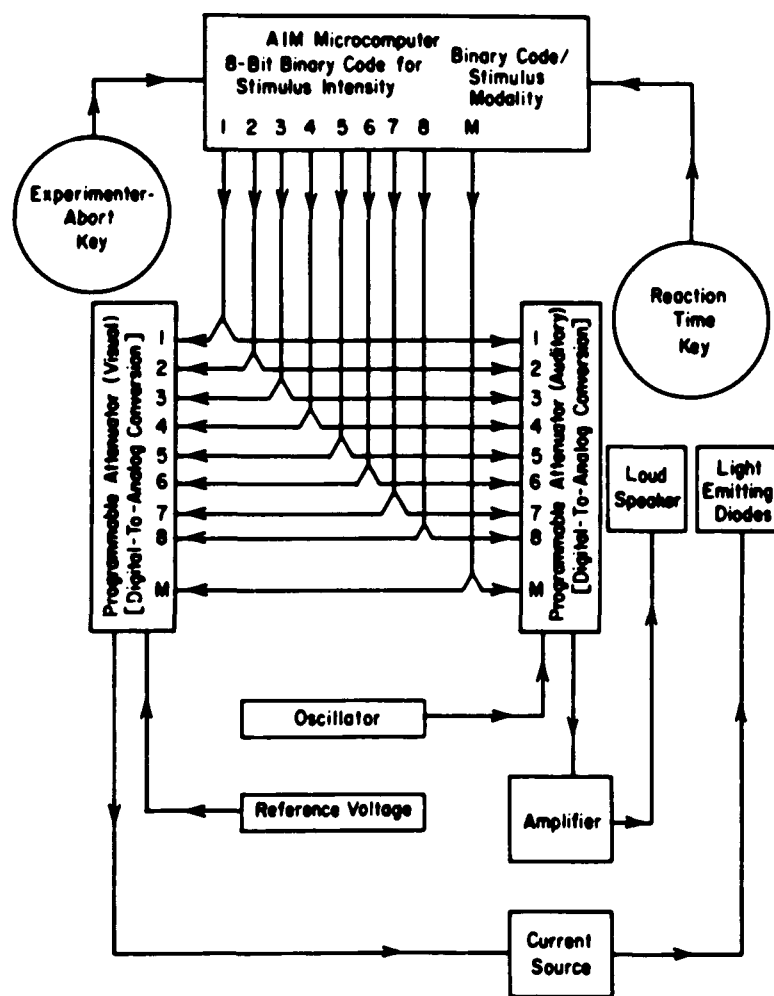


Figure 4. Block diagram of apparatus circuitry. Inputs and outputs are indicated by arrows.

There were 10 sound stimuli (S) and 10 light stimuli (L). The lowest intensity stimulus of each modality (S_1 and L_1) was set at 1.8 log units above the threshold values determined for the one normal subject. Subsequent values ($S_2, S_3, \dots, S_{10}$ and $L_2, L_3, \dots, L_{10}$) ranged from 1.9 to 2.7 log units above threshold, consecutive intensities differing by .1 log unit. Stated differently, the ten intensity values within each modality spanned a 1.0 log unit range in increments of .1 log unit.

The background illumination at the level of the subject's eyes (i.e., the amount of light present in the room, during the testing session) was .0055 foot-lamberts, as measured by a Spectra Pritchard Photometer (Photo Research Corporation). An air conditioning unit set at a constant level provided background noise, and partially masked extraneous sounds coming from outside the room. The background noise level was 58 dB (A) SPL (re $.0002 \text{ dynes/cm}^2$) as measured by a sound level meter (Quest Electronics, No. 215).

Initial Stimulus Calibration. The 8-bit binary input to a digital-to-analog converter was adjusted by manipulating a set of 8 switches. A current source, in turn, activated the light-emitting diodes and illuminated the ground glass covering the circular opening of the panel of the stimulus box.

A Spectra Pritchard Photometer was positioned at the level of the circular opening. The Pritchard was connected to a digital volt meter (Photo Research Corporation, Serial No. 145) and the read-out (in foot-lamberts) was noted. The 8 switches were manipulated until the desired output (from the LED's) was achieved. Readings were taken until the

10 binary values corresponding to light intensities differing by .1 log unit were obtained. (The maximum light output of the LED's was taken as reference 0.) Care was taken not to have the junction temperature of the LED's rise above 1° Celsius of ambient (about 22° C).

It was not necessary to conduct a calibration of the auditory apparatus since the output of the speaker is proportionate to the input signal, and the digital-to-analog converter is strictly linear.

Determination of Detection Thresholds. The 50% detection thresholds for light and sound stimuli were determined for one normal subject who was not known to have an auditory or a visual defect⁷, and these threshold values were used as reference points to set the lowest intensity values (S_1 and L_1) used in the experiment proper. A method of limits (D'Amato, 1970) was employed to determine the auditory and visual absolute thresholds. In this procedure, the subject responded "Yes, I saw the light" (or heard the sound), or "No, I didn't see the light" (or hear the sound) on each trial. Stimulus duration was held constant at 10 msec for all trials. The attenuation of the sound stimulus was varied by regulating a vernier. The intensity of the light stimulus was varied by removing and adding neutral density filters from a filter holder located in the stimulus box. The intensity difference between consecutive trials within a series was .1 log unit.

⁷The subject was a 21-year-old White male who had completed four years of college. He had had two complete physical examinations within a year of the testing date, and both of these examinations included hearing and vision tests. The most recent examination (within 3 months of testing) showed 20/20 vision and hearing within normal limits. Neither the subject, nor any of his family members, has ever had a hearing or vision problem.

For each modality, five ascending series and five descending series were administered. Ascending and descending series were alternated across all 10 series. The detection threshold within each modality was estimated by calculating the mean of the midpoint of consecutive values on which reversals in each series ("Yes" followed by "No," or "No" followed by "Yes") occurred.

A signal detection analysis was used to assess whether the auditory and visual threshold values (determined by the method of limits) were comparable on measures of sensitivity that are free of response bias influences. A six-category rating scale procedure was conducted for each modality. The categories were:

Yes 1-- absolutely certain that the stimulus was presented

Yes 2-- thinks that the stimulus was presented, but is uncertain

Yes 3-- very uncertain; best guess is "Yes"

No 3-- very uncertain; best guess is "No"

No 2-- thinks that the stimulus was not presented, but is uncertain

No 1-- absolutely certain that the stimulus was not presented

The subject was encouraged to make use of all categories.

For each modality, there were 10 blocks of 50 trials per block. Stimulus trials and blank trials (i.e., those on which no stimulus was presented) were quasi-randomized with the sole constraint that each block contained 50% stimuli and 50% blanks. In summary, there were 250 stimulus trials and 250 blank trials. The subject was given a 1-min rest period between blocks, and a 5-min rest period after the first 5 blocks.

Receiver Operating Characteristic (ROC) curves⁸ were fitted on linear coordinate axes by visual inspection. For both modalities, ROC curves increased monotonically with monotonically decreasing slopes, and were approximately symmetrical about the negative diagonal. Points making up the ROC curves were plotted on z-score axes, and lines were fitted by visual inspection. For both modalities, the z-score plots were approximately linear. Mutual regression equations⁹ (Grice, 1966) were then derived from z-values. The resulting equations were: Auditory-- $y = 1.07x + 1.04$; Visual-- $y = 1.00x + .77$.

One assumption of signal detection theory is that the signal and noise distributions underlying the theory are Gaussian in form. The finding that z-score plots were approximately linear, indicates that the Gaussian assumption was not violated (McNicol, 1972). A second assumption of signal detection theory is that signal and noise distributions have equal variances. The finding that the slopes of the lines derived from z-values were close to 1.00, suggests that the equal variance assumption was not violated (McNicol, 1972).

⁸An ROC curve is an isosensitivity curve obtained (for rating scale data) by plotting cumulative percent response to stimulus trials, as a function of cumulative percent response to blank trials, for each pair of values from Yes 1 through No 2.

⁹The method of least squares was not employed since this method assumes error (variation) in the y-axis measure, only: i.e., values of the independent variable (represented on the x-axis) are fixed. With points on an ROC curve, however, there is error in determination of the values on both axes since both are dependent measures. The mutual regression equation minimizes the perpendicular (rather than vertical) distances of obtained points from the fitted line. It should be noted, however, that although mutual regression is preferred to the method of least squares, strict adherence to its assumptions renders even this method inappropriate for rating scale data since the points obtained by that method are not independent.

Estimates of d' (a parametric measure of sensitivity that is not affected by response bias) were calculated: Auditory-- $d' = 1.04$; Visual-- $d' = 1.00$. These values suggest that valid measures of threshold had been obtained, since d' was comparable for both modalities.

Estimates of β ¹⁰ (β), a parametric measure of response bias, were also calculated: Auditory-- $\beta = 1.39$; Visual-- $\beta = 1.60$. These values suggest that the subject had adopted a relatively strict response criterion (i.e., a high degree of cautiousness for saying "Yes" when a stimulus was not presented), since both values exceed 1.00, the value of Optimal β for an a priori probability of stimulus occurrence of 0.5 (McNicol, 1972).

Procedure

Instructions and Informed Consent. The subject was told that the purpose of the task was to determine how fast he could lift his finger from a key when either a light or a sound was presented. After being shown the apparatus, the subject was informed that he may terminate his participation in the study at any time. Patients were told that their participation or discontinuation would not affect their treatment or stay in the hospital in any way. Written consent to participate in the experiment was then obtained (see Appendix F and Appendix G for patient and control consent forms), and the subject was given a copy of the

¹⁰A separate β can be calculated for each point on an ROC curve. However, since a thorough evaluation of the subject's response criterion is not of interest to the present study, only a single estimate of β was obtained. The Yes-3 coordinates (located at the center of the ROC curve) were used to calculate this estimate.

consent form to keep. The following instructions were then read to the subject:

This session will last about an hour, but we will stop for several rests. Which hand do you use to write with? (RESPONSE) Okay, for the entire session, I'd like you to use the index finger of your (WRITING HAND SPECIFIED). Please do not use any other finger.

Place your index finger on the key in front of you. (CONFIRM) Good. Now look at the circle on the box. Notice that it is surrounded by four small red lights. (POINT) These lights will remain on all the time. They will show you where to look, since you won't be able to see the large circle in the dark. It is very important that you look straight ahead, right between the four small red lights all the time, except during rest periods.

In a moment, I will turn off the light in the room, and present various lights and sounds. Some lights will be dim, and others bright. Some sounds will be low, and others loud. You won't be told when a light or a sound will be presented, so you must pay attention.

Whenever either a light or a sound is presented, lift your finger from the key as quickly as possible, and then return your finger to the key. Do you have any questions? We will now have a few practice trials to be sure the directions are clear.

The subject was dark-adapted for 5 minutes before testing began.

Block and Session Composition. There were 10 blocks of 41 trials per block. Since the first trial of each block cannot be classified as either crossmodal or ipsimodal, the total number of reaction times that were used in the analyses was 400 per subject. However, a minimum of 410 stimuli (41×10) were delivered.

All subjects were given the identical sequence of stimuli. The following constraints were used to generate this sequence:

- (1) Each stimulus (e.g., S_6 , L_4 , L_2) was preceded by every stimulus (including itself) once. Since there were 20 stimuli ($S_1, S_2, \dots, S_{10}, L_1, L_2, \dots, L_{10}$) and 400 trials, there was one trial for each categorization ($S_1S_1, S_1S_2, \dots, S_1S_{10}, S_2S_1, S_2S_2, \dots, \dots, S_{10}S_{10}, L_1L_1, \dots, \dots, L_{10}L_{10}, S_1L_1, \dots, \dots, S_{10}L_{10}, L_1S_1, \dots, \dots, L_{10}S_{10}$).
- (2) The 40 usable trials of each block consisted of 10 crossmodal light, 10 crossmodal sound, 10 ipsimodal light, and 10 ipsimodal sound trials.

The results of a runs test (Siegel, 1956) indicated that the sequence of light and sound stimuli within each block of trials was random ($z = -.16$, n.s.).¹¹

¹¹The most correct statement of the null hypothesis is that the number of runs ($\bar{r}$) in a block of trials is not different from the sampling distribution of values of $\bar{r}$ expected from repeated random samples of size 41 (the number of trials per block). A z -value of $-.16$ indicates that we would fail to reject the null hypothesis. Since all blocks contained an equal number of crossmodal and ipsimodal sequences, there were 21 runs in each block. Thus, the z -value is the same for all 10 blocks.

Interstimulus Interval. The timing of events during a block of trials was independent of the subject's response: i.e., the subject's speed of responding had no effect on interstimulus interval, and each trial was cycled automatically, with no warning signal, and no subject initiation required.

Interstimulus interval (measured from the onset of the stimulus of the present trial to the onset of the stimulus of the next trial) varied between 2.0 sec and 3.5 sec. On each trial, a fixed interval of 2.0 sec, plus an added interval ranging from 0.0 sec to 1.5 sec, constituted the interstimulus interval for that trial. The maximum duration of the added interval (1.5 sec) was equally divided into 256 parts, each differing by approximately 5.9 msec (i.e., 1.5 sec divided by 256). On each trial, the AIM initiated a 2.0 sec interval and randomly chose one of the 256 parts to be added to this interval. Thus, interstimulus interval varied from trial to trial and from subject to subject. The rationale for using variable intervals, rather than fixed intervals, was to minimize the number of anticipatory responses, and to assure that the subject was responding to the stimulus, rather than to the termination of a constant interval of time.

"Misses" and "Anticipations." A "miss" was operationally defined as a failure to respond within 1 sec of the onset of the stimulus. An "anticipation" was defined as either a response that is less than 100 msec (measured from stimulus onset), or a response prior to the onset of the stimulus. Miss and anticipation trials are not included in the major analyses: i.e., only reaction times between 100 msec and 1 sec were considered legitimate responses.

An upper cut-off value (1 sec) was used since it was assumed that relatively long responses to suprathreshold stimuli in a simple reaction time task (i.e., a task in which the identical response is made on all trials and, thus, response uncertainty is not a factor) probably resulted from a lack of engagement in the task. The rationale for employing a lower cut-off value (100 msec) concerns the physiological limits that are set on an organism's response time. For example, when a sound is presented, a certain amount of time is required for nerve impulses to be conducted from the receptors, to the auditory nerve, to cortex, and along descending pathways to the particular muscle groups involved in the response. Based on electrophysiological experiments with cats, Woodworth and Schlosberg (1954) have estimated that the minimum time required for sensory and motor nerve conduction of an impulse initiated by an auditory stimulus is about 70 msec. For visual stimulation, an additional 20-30 msec elapses since the transduction process is slower at the receptor level (i.e., visual vs. auditory). Thus, 100 msec was chosen as the lower cut-off value in the current research.

Trial Replacement. Miss and anticipation trials were replaced ("made up") to assure that an equal number of (usable) crossmodal and ipsimodal light and sound trials was maintained for all subjects. The procedure for trial replacement (which was automatically instituted by the AIM) involved the addition of two trials, and the deletion (from the data analysis) of one trial.

The hypothetical sequences shown in Table 6 illustrate the replacement procedure. In this example, the subject "missed" the S_9 stimulus presented on Trial 4. Instead of presenting L_2 (of the pre-planned

Table 6

**Illustration of the Trial Replacement Procedure
for "Misses" and "Anticipations"**

Pre-Planned Stimulus Sequence for Block X		Replacement of "Miss" Trial	
Trial Number	Stimulus	Trial Number	Stimulus
1	L ₁	1	L ₁
2	S ₃	2	S ₃
3	L ₆	3	L ₆
4	S ₉	4	S ₉
5	L ₂	5	L ₆
6	L ₅	6	S ₉
'	'	7	L ₂
'	'	8	L ₅
'	'	'	'
40	L ₇	'	'
41	S ₄	'	'
		42	L ₇
		43	S ₄

MISS

Trial 3
repeatedTrial 4
repeated

sequence) on Trial 5, Trial 3 (and, subsequently, Trial 4) is repeated. The reaction time on Trial 5 (which is a repetition of Trial 3) would not be included in the data analysis. Trial 5 only serves the purpose of preserving the crossmodal-sound (L_6 - S_9) response on Trial 6.

An alternative trial replacement procedure is to add trials to the end of the block. However, this procedure would change the relative position of crossmodal and ipsimodal sequences within the block. For example, if several patients missed three or four ipsimodal-sound trials located at the beginning of the block, and these trials were "made up" at the end of the block, then patient-control comparisons may be confounded by potentially relevant order variables. Missed trials were replaced immediately, to preserve, as best as possible, the relative positions of sequences within the block. Obviously, the same applies to anticipation trials, and all that has been said is underscored when one considers the relative positions of intensities and modalities within a block.

Rest Periods, Data Retrieval, and the Monitoring of Subject Performance. The subject was given a 1-min rest period between blocks, and a 5-min rest period at the end of the fifth block of trials. Reaction times (measured from the onset of the stimulus to the finger-lift response) were printed (by the AIM) at the end of the session.

The experimenter was essentially a passive observer during the testing session. Neither he nor the subject knew which stimulus would be presented on any single trial, and the experimenter could not alter the sequence of stimuli in any way. Therefore, unintentional cueing of crossmodal and ipsimodal sequences was impossible. Furthermore, the

experimenter was unaware of the subject's reaction time on any trial, since only the trial number was displayed by the AIM. If the trial number decreased from one trial to the next, the experimenter knew that the subject had either missed or anticipated the stimulus. If the trial number continued to decrease on several consecutive trials, the experimenter halted the program (by pressing the experimenter-abort key) and reviewed the task with the subject. The experimenter would also check the subject periodically, to assure that he was responding with the correct finger, and that he was not leaning back, or bending forward, in his chair. In summary, the experimenter's principal role during the testing session was to be ready to intervene if the subject was not performing the task properly.

General Comments on the Analyses

Logarithmic Transformations and Geometric Mean Reaction Times. 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 studies of reaction time (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. For example, the crossmodal/light condition for each subject was represented by the arithmetic mean of 10 geometric means (one for each intensity value). The rationale for this strategy was that displaying results in logarithmic units would be less meaningful, and more difficult to inspect, since investigators in this area of research are accustomed to viewing reaction time data in millisecond units.

Separate Analyses for Light and Sound. Analyses were conducted separately, for reaction times to light stimuli and reaction times to sound stimuli. Most experiments have shown that the modality shift effect is more consistent for reaction times to sound stimuli (Sutton et al., 1961; Sutton & Zubin, 1965, Experiments A, B, and C). The two studies showing a positive modality shift effect for both light and sound stimuli (Spring, 1980; Waldbaum et al., 1975) were the only experiments in which visual and auditory stimuli were equated with respect to sensation level. However, varying methodologies were employed in these two experiments. Separate analyses for light and sound were conducted in the current research to further assess whether schizophrenic patients exhibit the modality shift effect for both modalities, when auditory and visual stimuli are equated with respect to sensation level.

Preliminary Analyses

Chronicity Subtypes of Schizophrenia. The modality shift effect has been demonstrated in chronic schizophrenic patients (Sutton et al., 1961; Sutton & Zubin, 1965, Experiment B); in acute schizophrenic patients (Spring, 1980; Waldbaum et al., 1975); and in process and reactive schizophrenic patients (Sutton & Zubin, 1975, Experiment A). However, chronic and acute patients have never been tested in the same crossmodal reaction time experiment. Since one premise of the Chapmans' methodological treatise concerns the concept of "generalized deficit" (in reaction time research, overall slowness of schizophrenic patients, as a group), it was reasoned that differing degrees of generalized deficit which emerged from a relevant subject variable (e.g., chronicity

subtype) could influence the results.

Mann-Whitney U tests were conducted to determine whether chronic/subchronic and acute/subacute patient subgroups differed significantly with respect to overall reaction time level. It was also of interest whether these patient subgroups differed on crossmodal retardation (i.e., average geometric mean crossmodal reaction time minus average geometric mean ipsimodal reaction time), a principal variable in later analyses. As shown in Table 7, there were no significant differences between these patient subgroups in any comparison. Therefore, chronic and acute patients were combined in all subsequent analyses.

Category Subtypes of Schizophrenia. Although paranoid and nonparanoid schizophrenic patients have never been compared in a crossmodal reaction time experiment, investigators in other areas of reaction time research (see Nuechterlein, 1977), as well as in other areas of attentional research in schizophrenia (see Venables, 1977), have studied these subgroups separately. Mann-Whitney U tests were conducted to determine whether paranoid and nonparanoid patient subgroups of the current research differed significantly with respect to ipsimodal and crossmodal reaction time, and crossmodal retardation. As shown in Table 7, there were no significant differences between these patient subgroups in any comparison. Therefore, paranoid and nonparanoid patients were combined in all subsequent analyses.

Comparisons of Skewness Measures. Measures of skewness (g_1) for untransformed (raw) and transformed (geometric mean) reaction time data are shown in Table 8 (light) and Table 9 (sound). A g_1 value of: 0.00 denotes a symmetric, bell-shaped (normal) distribution; greater than

Table 7

Mann-Whitney U-test Comparisons of Acute vs. Chronic
and Paranoid vs. Nonparanoid Patient Subgroups
on Crossmodal and Ipsimodal Reaction Times
and Crossmodal Retardation

	Crossmodal		Ipsimodal		Retardation	
	<u>U</u>	<u>P</u>	<u>U</u>	<u>P</u>	<u>U</u>	<u>P</u>
Acute vs. Chronic ^a						
Light	25	.78	21	.46	15	.15
Sound	21	.46	28	1.00	20	.39
Paranoid vs. Nonparanoid ^b						
Light	20	.39	28	1.00	25	.78
Sound	23	.61	24	.69	21	.46

Note. All p-values are for two-tailed tests; $\underline{n}_1/\underline{n}_2 = 7/8$ for all comparisons.

^aThere were 8 acute patients and 7 chronic patients.

^bThere were 7 paranoid patients and 8 nonparanoid patients.

0.00 denotes positive skewness, and; less than 0.00 denotes negative skewness.

The following points are noteworthy: (1) For both untransformed and transformed data, and light and sound analyses, the patient group did not exhibit significant skewness (either positive or negative) in any condition. (2) With few exceptions, the control group exhibited significant departures from normality in all conditions, and for both untransformed and transformed data, in the direction of positive skewing. In general, the degree of skewness was greatest in the ipsimodal condition. (3) The patient group exhibited negative skewness primarily in the crossmodal-sound condition (see Table 9) and for the higher intensities (6 through 10) of the crossmodal-light condition (see Table 8), and positive skewness in all other conditions. (4) The effect of transforming the patient data was not uniform: i.e., for some conditions, the transformation increased the degree of skewness, whereas for other conditions, the degree of skewness was decreased. (5) With the exception of the ipsimodal-sound condition (see Table 9), the effect of transforming the control data was to reduce the degree of skewness. However, with only one exception (crossmodal-light, intensity 10), none of the transformations resulted in a non-significant degree of skewness.

Measures of skewness are rarely reported in reaction time experiments, probably because the F distribution is robust with respect to violation of the normality assumption of parametric analyses (Kirk, 1968). However, Woodworth and Schlosberg (1954) have noted that, whereas the distribution of reaction times of an individual subject is characteristically positively skewed, the distribution of mean reaction

Table 8

g_1 Measures of Skewness of Arithmetic Mean (Untransformed) and Geometric Mean (Transformed)

Reaction Time Data for Patient and Control Groups in Each Light Condition

Group/Condition	Intensity Value									
	1	2	3	4	5	6	7	8	9	10
Patients										
Crossmodal										
Untransformed	.00	.08	.40	.26	.12	-.21	-.02	-.32	-.40	-.22
Transformed	.12	.15	.45	.42	.21	-.10	.04	-.17	-.32	-.06
Ipsimodal										
Untransformed	.68	1.17	.32	-.06	.67	.58	1.03	.21	.91	.00
Transformed	.73	1.20	.32	-.05	.64	.73	1.13	.34	.89	-.10

Table 8 (continued)

 g_1 Measures of Skewness of Arithmetic Mean (Untransformed) and Geometric Mean (Transformed)

Reaction Time Data for Patient and Control Groups in Each Light Condition

Group/Condition	Intensity Value									
	1	2	3	4	5	6	7	8	9	10
Controls										
Crossmodal										
Untransformed	.49	.98**	.66	.33	1.32***	1.13**	.61	1.49***	.77*	.70*
Transformed	.49	.95**	.58	.29	1.18***	1.07**	.62	1.44***	.72*	.61
Ipsimodal										
Untransformed	1.43***	1.64***	1.28***	1.40***	1.34***	1.08**	1.22***	1.17***	1.12**	1.56***
Transformed	1.41***	1.63***	1.26***	1.32***	1.30***	1.11**	1.17***	1.13**	1.03**	1.51***

Note. Values of g_1 are shown. Test for normality: $\frac{z}{\sqrt{g_1}} = g_1$ divided by the square root of $(6/N)$.

There were $N = 15$ patients and $N = 50$ control subjects.

* $p < .05$

** $p < .01$

*** $p < .001$

Table 9

g₁ Measures of Skewness of Arithmetic Mean (Untransformed) and Geometric Mean (Transformed)

Reaction Time Data for Patient and Control Groups in Each Sound Condition

Group/Condition	Intensity Value									
	1	2	3	4	5	6	7	8	9	10
Patients										
Crossmodal										
Untransformed	-.16	-.34	-.45	-.11	-.10	-.67	-.14	.18	.07	.63
Transformed	-.08	-.38	-.42	.12	-.11	-.57	.11	.26	-.09	.60
Ipsimodal										
Untransformed	.00	.35	-.10	.74	.59	.36	.23	.28	.62	.17
Transformed	.10	.39	-.03	.93	.58	.23	.01	.33	.62	.13

Table 9 (continued)

 g_1 Measures of Skewness of Arithmetic Mean (Untransformed) and Geometric Mean (Transformed)

Reaction Time Data for Patient and Control Groups in Each Sound Condition

Group/Condition	Intensity Value									
	1	2	3	4	5	6	7	8	9	10
Controls										
Crossmodal										
Untransformed	.87*	.63	1.21***	1.21***	1.24***	1.41***	.86*	1.02**	1.13**	1.03**
Transformed	.77*	.63	1.18***	1.12**	1.15***	1.31***	.79*	1.10**	1.14**	1.00**
Ipsimodal										
Untransformed	1.51***	1.59***	1.56***	1.33***	1.09**	1.46***	1.43***	1.28***	1.49***	1.71***
Transformed	1.54***	1.66***	1.62***	1.37***	1.08**	1.50***	1.36***	1.31***	1.55***	1.72***

Note. Values of g_1 are shown. Test for normality: $\frac{z}{g_1} = g_1$ divided by the square root of $(6/N)$.

There were $N = 15$ patients and $N = 50$ control subjects.

* $p < .05$

** $p < .01$

*** $p < .001$

times for a large number of subjects ($N = 1,000$ subjects in the experiment cited by Woodworth and Schlosberg) is generally normally distributed. It is not apparent why the distributions of mean reaction times of the normal control subjects in the current research showed significant departures from normality. Perhaps a more systematic, random sampling of "normal" subjects (i.e., compared to the selection/screening procedure used in the current research, in which individuals with a Probable or Definite, past or present mental disorder were excluded) would have resulted in more symmetric distributions of mean reaction times.

Comparisons of Variances. Table 10 shows the F -values of the untransformed (raw) and transformed (geometric mean) reaction time data comparing patient and control groups in each of the 40 experimental conditions (i.e., light/sound X crossmodal/ipsimodal X 10 intensities = 40 conditions). The following points are noteworthy: (1) For both untransformed and transformed data, a significant F -value was found for only one (of 40) condition: crossmodal-light, intensity 1. Stated differently, there were no significant differences between the intra-group variabilities of patients and controls in 39 of the 40 experimental conditions. (2) in 26 of the 40 conditions, the effect of transforming the data was to reduce the ratio of patient-control variances (i.e., to decrease the value of F) and thus to increase homogeneity. (3) Differences between the F -values of untransformed and transformed data are small, and in no condition was a significant F -value changed to a non-significant F -value as a result of the transformation.

The finding that, in 39 of 40 comparisons, the variances of pa-

Table 10

F-values of Arithmetic Mean (Untransformed) and Geometric Mean (Transformed)
 Reaction Time Data Comparing Patient and Control Groups in Each Experimental Condition

Condition	Intensity Value									
	1	2	3	4	5	6	7	8	9	10
Light										
Crossmodal										
Untransformed ^a	2.48*	1.63	1.75	1.85	1.55	1.82	1.25	1.52	1.85	1.69
Transformed ^a	2.46*	1.53	1.68	1.75	1.45	1.90	1.28	1.50	1.88	1.61
Ipsimodal										
Untransformed ^b	1.11	1.19	1.25	1.03	1.47	1.45	1.42 ^c	1.06	2.21	1.76
Transformed ^b	1.05	1.12	1.18	1.00	1.32	1.43	1.49 ^c	1.06 ^c	2.22	1.55

Table 10 (continued)

F-values of Arithmetic Mean (Untransformed) and Geometric Mean (Transformed)

Reaction Time Data Comparing Patient and Control Groups in Each Experimental Condition

Condition	Intensity Value									
	1	2	3	4	5	6	7	8	9	10
Sound										
Crossmodal										
Untransformed ^a	1.06	1.44	1.23	1.03	1.58	1.06 ^d	1.45	1.25	1.84	1.71
Transformed ^a	1.03 ^d	1.45	1.29	1.06	1.59	1.03 ^d	1.29	1.15	1.90	1.73
Ipsimodal										
Untransformed ^b	1.16	1.19	1.73	1.57	1.18 ^c	1.58	1.92	1.99	1.03	1.26
Transformed ^b	1.05	1.10	1.56	1.50	1.37 ^c	1.34	1.51	1.76	1.15 ^c	1.22

^adf = 14/49 unless otherwise indicated^bdf = 49/14 unless otherwise indicated^cdf = 14/49^ddf = 49/14

*p < .05

tients and controls did not differ significantly, warrants further discussion. A general finding in reaction time research (regardless of the particular reaction time task employed) is that psychotic patients are more variable than control subjects (King, 1975). Two points are noteworthy: (1) Few investigators have reported whether reaction time cut-off values were used (i.e., whether only reaction times within a certain range were included in their data analyses). When indicated, upper cut-off values frequently have been more liberal (i.e., longer) than the 1 sec limiting value used in the current research. For example, Spring (1980), in a crossmodal reaction time experiment, included all reaction times less than 2 sec in her analyses. Tizard and Venables (1956), in a simple reaction time paradigm in which preparatory intervals in regular and irregular blocks were varied, used 5 sec as an upper cut-off value. Perhaps very slow patient reaction times, in part, can account for the large patient-control differences in variance found in most experiments. (2) In the current research, 50 normal control subjects were tested. With few exceptions, reaction time experiments with psychotic patients have typically included control groups of 10, 15, or 20 subjects (Huston, Shakow, & Riggs, 1937; Knehr, 1954; Rodnick & Shakow, 1940; Tizard & Venables, 1956; Venables & O'Connor, 1959; Venables & Tizard, 1956a, 1956b; Waldbaum et al., 1975; Zahn, Rosenthal, & Shakow, 1961; Zahn, Shakow, & Rosenthal, 1961). The purpose of testing a large number of control subjects in the current research was to optimize the chances that both slow responders and fast responders would be represented. Perhaps, by sampling a larger number of control subjects, extreme responders at either tail, or at both tails, of the

reaction time distribution were recruited as subjects. It follows that the intragroup variability (of control subjects) would be larger than that found in most studies, and that patient-control differences in group variability would be reduced.

Epsilon Adjustments and Alpha Levels

Although in 39 of 40 comparisons, the variances of patients and controls did not differ significantly, repeated-measures analyses of variance additionally assume that covariances are also homogeneous¹². Tests for compound symmetry were conducted to determine whether this assumption was violated. Results were significant ($p < .001$) in all analyses (light and sound, for both transformed and untransformed data), indicating that the degree of heterogeneity was beyond acceptable standards.¹³

Jennings and Wood (1976) have discussed the problem of non-homogeneity in a repeated-measures design. These authors have described an adjustment procedure (originally formulated by Geisser and Greenhouse,

¹²In a factorial design, in which different subjects are randomly assigned to different treatment levels, it can be assumed that experimental errors between levels are independent. However, in a repeated-measures design, in which the same subjects are tested under different levels of the same treatment, assessment of homogeneity of covariances is imperative since, by definition, measures across treatment levels are not independent (Kirk, 1968).

¹³A variance-covariance matrix is a $K \times K$ array in which each k_i is a different level of a repeated-measures factor. Values within diagonal cells are the variances (pooled across subject groups) for each k_i factor level. Values within off-diagonal cells are covariances (i.e., the product of r_{xy} , SD_x , and SD_y) for each combination of k_i factor levels. A variance-covariance matrix shows compound symmetry when values along the main diagonal are homogeneous, and off-diagonal values are also homogeneous (Winer, 1971)

1958) in which the degrees of freedom for the usual F test are reduced by a factor of epsilon (which is estimated from the variance-covariance matrix). The epsilon adjustment procedure is only applicable for significant F -values involving repeated-measures factors with at least three levels; for a two-level factor, $df = k - 1 = 1$, and no further reduction in the degrees of freedom is possible. Since the homogeneity of variance-covariance assumption was violated in the current research (as indicated by the tests for compound symmetry), the epsilon adjustment procedure was employed for all significant F -values involving the repeated-measures intensity factor (10 levels).

Although the epsilon adjustment procedure is only applicable for factors with at least three levels, violations of the homogeneity of variance-covariance (and of the normality) assumption can result in a Type I error regardless of the number of factor levels. For example, a "significant" interaction between the two-level factors, modality shift (crossmodal, ipsimodal) and subject group (patient, control), which resulted from heterogeneous covariances, would lead to the false conclusion that the null hypothesis should be rejected. McNemar (1969) has recommended that "when using a repeated-measure design, the researcher would be wise to require F to reach the .01 level in order to have assurance that significance can be claimed at the .05 level" (pp 357-358). Therefore, an alpha level of 1% was considered "significant" for all tests in the current research. However, for comparability with other experiments, p -values of less than .05 are included in all Tables, but are discussed (in the text) as "approaching" or "tending towards" significance; $p > .05$ is recorded as "not significant" (abbreviated

"n.s.") in all Tables.

CHAPTER III

RESULTS

Overview of the Major Analyses

Four stages of analyses were planned:

Stage 1: Confirmation of the Modality Shift Effect. The main objective of this stage was to replicate the findings of other investigators by demonstrating the modality shift effect. More specifically, a significant subject group (patient, control), X modality shift (cross-modal, ipsimodal) interaction was predicted, in the direction of disproportionate slowing of schizophrenic patients on crossmodal trials. Several additional analyses (e.g., within-group comparisons of cross-modal and ipsimodal reaction times, assessment of the modality shift effect for each intensity) were also planned for this stage.

Stage 2: Equating Conditions on Discriminating Power. The purpose of this stage was to "match" the crossmodal and ipsimodal conditions of the 50 normal control subjects on reliability and variance. The matching procedure was to entail the selective deletion of reaction times to certain intensities.

Stage 3: Further Assessment of Discriminating Power. The purpose of this stage was to assess whether crossmodal and ipsimodal conditions remained matched (on reliability and variance) for a subgroup of slow-responding normal control subjects.

Stage 4: Evaluation of Psychometric Artifact. The purpose of this stage was to assess whether patients exhibited the modality shift effect on matched conditions. The trials (corresponding to certain intensity values) that were deleted to achieve a matching in Stage 2, were to be deleted from the data of the schizophrenic patients. The resulting distributions were to be used to compare patients and controls, as in Stage 1. A significant subject group X modality shift interaction (on matched conditions) would refute the hypothesis that the modality shift effect could be explained by psychometric artifact.

Stage 1: Confirmation of the Modality Shift Effect

Reaction Times of Patients and Controls in Each Experimental Condition. Group geometric mean reaction times and standard deviations for each intensity value and modality shift condition are shown in Table 11 (light) and Table 12 (sound). For ease of inspection, mean reaction time data for light and sound are portrayed in Figures 5 and 6, respectively. The following observations are noteworthy: (1) for all intensity values, both modality shift conditions, and both light and sound stimuli, the patient group responded more slowly than the control group. (2) For all intensity values, and both light and sound stimuli, the patient group and the control group responded more slowly on crossmodal trials than on ipsimodal trials. Stated differently, crossmodal retardation was demonstrated for both subject groups, regardless of the intensity, or of the modality, of the imperative stimulus. (3) The magnitude of crossmodal retardation was greater for the patient group (compared to the control group) in every condition. (4) For both light and sound

Table 11
Average Geometric Mean Reaction Times to Light Stimuli
for Patient and Control Groups in Each Experimental Condition

Group/Condition	Intensity Value										Mean	<u>SD</u>
	1	2	3	4	5	6	7	8	9	10		
Patients												
Crossmodal												
Mean	514	489	482	481	466	464	443	460	462	466	473	77
<u>SD</u>	104	83	78	83	81	81	67	79	83	78		
Ipsimodal												
Mean	430	405	396	403	385	372	389	374	357	373	388	56
<u>SD</u>	74	67	67	67	56	54	69	65	41	52		

Table 11 (continued)

Average Geometric Mean Reaction Times to Light Stimuli
for Patient and Control Groups in Each Experimental Condition

Group/Condition	Intensity Value										Mean	<u>SD</u>
	1	2	3	4	5	6	7	8	9	10		
Controls												
Crossmodal												
Mean	346	342	334	329	326	318	315	318	314	316	326	60
<u>SD</u>	66	65	59	61	65	60	60	64	61	60		
Ipsimodal												
Mean	345	330	333	325	321	314	309	313	302	306	320	66
<u>SD</u>	78	73	75	68	68	65	58	67	61	69		

Note. All means and standard deviations are expressed in milliseconds.

Table 12
Average Geometric Mean Reaction Times to Sound Stimuli
for Patient and Control Groups in Each Experimental Condition

Group/Condition	Intensity Value										Mean	<u>SD</u>
	1	2	3	4	5	6	7	8	9	10		
Patients												
Crossmodal												
Mean	465	474	460	453	451	450	441	443	444	435	451	69
<u>SD</u>	72	84	80	70	84	66	82	76	84	85		
Ipsimodal												
Mean	393	377	367	354	370	373	354	354	349	347	364	61
<u>SD</u>	78	78	60	59	75	62	57	56	68	65		

Table 12 (continued)
Average Geometric Mean Reaction Times to Sound Stimuli
for Patient and Control Groups in Each Experimental Condition

Group/Condition	Intensity Value										Mean	SD
	1	2	3	4	5	6	7	8	9	10		
Controls												
Crossmodal												
Mean	321	321	306	300	299	299	304	292	288	291	302	65
SD	70	70	72	69	66	68	68	68	62	65		
Ipsimodal												
Mean	303	306	299	294	291	291	286	289	285	285	293	75
SD	84	85	79	74	69	78	79	79	69	73		

Note. All means and standard deviations are expressed in milliseconds.

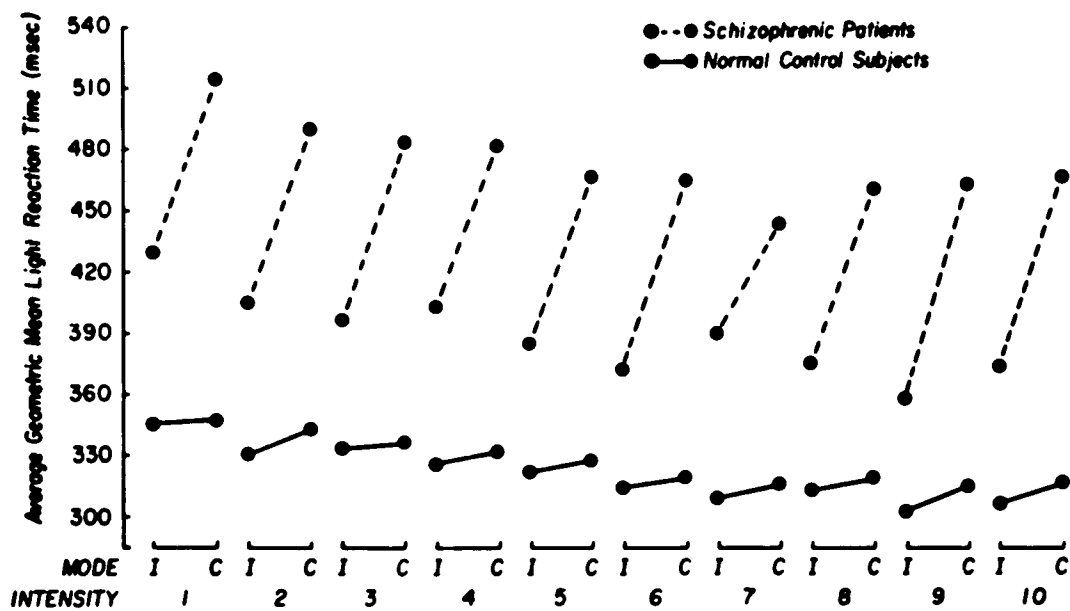


Figure 5. Reaction times of patient and control groups to light stimuli, as a function of stimulus sequence (I = ipsimodal, C = crossmodal) and intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. There were 15 schizophrenic patients and 50 normal control subjects.

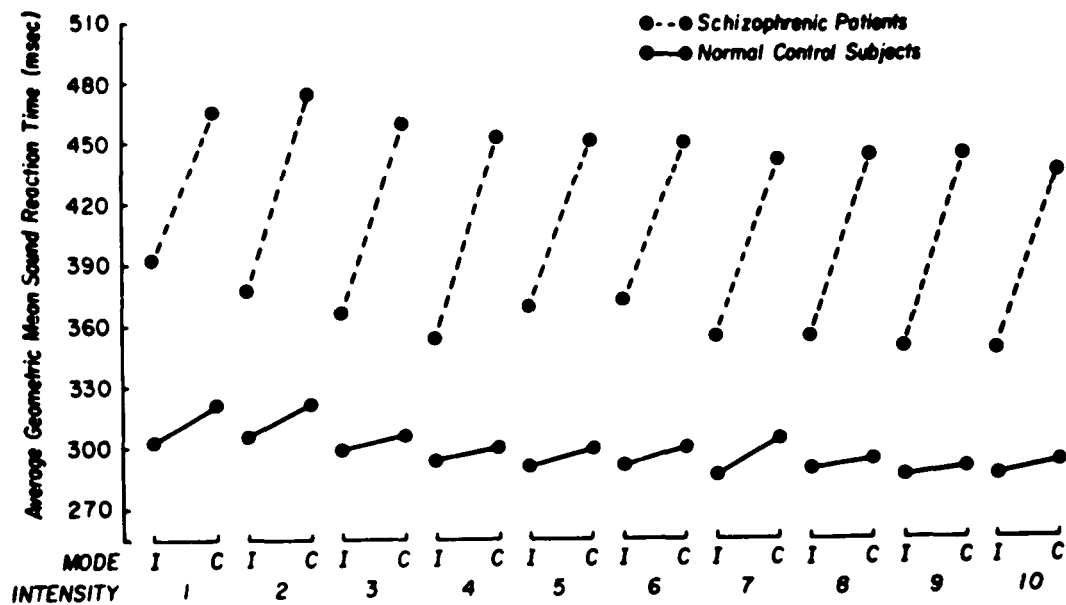


Figure 6. Reaction times of patient and control groups to sound stimuli, as a function of stimulus sequence (I = ipsimodal, C = cross-modal) and intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. There were 15 schizophrenic patients and 50 normal control subjects.

stimuli, both modality shift conditions, and both subject groups, reaction time tended to decrease as a function of increasing intensity (1 through 10). (5) For both light and sound stimuli, in the cross-modal condition, the patient group tended to be more variable (i.e., greater intra-group variability) than the control group; whereas in the ipsimodal condition, the control group tended to be more variable than the patient group (see Tables 11 and 12). However, as indicated in an earlier section (see Comparisons of Variances, page 92), with only one exception (crossmodal-light, intensity 1), there were no significant differences between patient and control group variances.

Confirmation of the Modality Shift Effect. Data were analyzed by a three-factor mixed model analysis of variance with subject group (patient, control) as a between factor, and modality shift (crossmodal, ipsimodal) and intensity (1 through 10) as repeated-measures factors. Analyses were conducted separately for reactions to light and sound. The results of these analyses are shown in Table 13 (light) and Table 14 (sound)¹⁴. Light and sound findings will be discussed together, since results are similar for both modalities.

For both light and sound analyses, significant main effects were found for subject group, modality shift, and intensity; also, a significant interaction was found for subject group X modality shift. The

¹⁴Analyses of variance were also conducted on the untransformed data. As shown in Appendix I (Tables 41 and 42), for both light and sound analyses, the same results were found. Every significant main effect and interaction shown by the transformed data, was also found for the untransformed data. The results following epsilon adjustments were also the same for transformed and untransformed data.

Table 13
Analysis of Variance for Effects of
Group (Patients, Controls), Modality Shift, and Intensity
on Geometric Mean Reaction Times to Light Stimuli

Source	<u>SS</u>	<u>df</u>	<u>ms</u>	<u>F</u>	<u>p</u>
Total	9,421,718	1299	-----	-----	-----
Between Subjects	7,085,120	64	-----	-----	-----
Subject Groups (G)	2,686,826	1	2,686,826	38.49	.001 ^a
Error _b	4,398,294	63	69,814	-----	-----
Within Subjects	2,336,598	1235	-----	-----	-----
Modality Shift (M)	469,907	1	469,907	37.72	<.001 ^b
Intensity (I)	207,090	9	23,010	50.38	<.001 ^c
G x M	354,519	1	354,519	28.50	<.001 ^d
G x I	13,602	9	1,511	3.31	<.001 ^e
M x I	11,474	9	1,274	3.17	<.01 ^f
G x M x I	7,909	9	879	2.18	<.05 ^g
Error ₁	784,900	63	12,459	-----	-----
Error ₂	258,949	567	457	-----	-----
Error ₃	228,248	567	403	-----	-----

^aepsilon adjustment not applicable. G is not a repeated-measures factor.

^bepsilon adjustment not applicable. df cannot be reduced further.

^cepsilon adjustment-- df = 1,63; p < .001

^depsilon adjustment not applicable. df cannot be reduced further.

^eepsilon adjustment-- df = 5,304; p < .01

^fepsilon adjustment-- df = 1,75; n.s.

^gepsilon adjustment-- df = 1,75; n.s.

Table 14
Analysis of Variance for Effects of
Group (Patients, Controls), Modality Shift, and Intensity
on Geometric Mean Reaction Times to Sound Stimuli

Source	<u>SS</u>	<u>df</u>	<u>ms</u>	<u>F</u>	<u>p</u>
Total	10,453,049	1299	-----	-----	-----
Between Subjects	8,136,270	64	-----	-----	-----
Subject Groups (G)	2,796,794	1	2,796,794	33.00	<.001 ^a
Error _b	5,339,476	63	84,754	-----	-----
Within Subjects	2,316,779	1235	-----	-----	-----
Modality Shift (M)	539,036	1	539,036	47.37	<.001 ^b
Intensity (I)	97,438	9	10,826	19.03	<.001 ^c
G x M	354,543	1	354,543	31.16	<.001 ^d
G x I	5,894	9	655	1.15	n.s.
M x I	3,960	9	440	.93	n.s.
G x M x I	7,303	9	811	1.71	n.s.
Error ₁	716,903	63	11,379	-----	-----
Error ₂	322,526	567	569	-----	-----
Error ₃	269,176	567	475	-----	-----

^a epsilon adjustment not applicable. G is not a repeated-measures factor.

^b epsilon adjustment not applicable. df cannot be reduced further.

^c epsilon adjustment-- df = 1,63; p < .001

^d epsilon adjustment not applicable. df cannot be reduced further.

reasons for the significant main effects for subject group and for modality shift have already been indicated. To reiterate: the patient group responded more slowly than the control group in all conditions; and both subject groups responded more slowly on crossmodal trials, than on ipsimodal trials, for all intensity values and for both modalities (see Figures 5 and 6).

The effect of intensity on reaction time is shown in Figure 7 (see Combined Subject Groups) for light, and in Figure 8 for sound. For both modalities, there is a definite tendency for reaction times (averaged across subject groups and modality shift conditions) to decrease as a function of increasing intensity.

The subject group X modality shift interaction is portrayed in Figure 9. For both light and sound stimuli, the patient group was disproportionately retarded on crossmodal trials. The magnitude of crossmodal retardation for light was 85 msec for the patient group as compared to 6 msec for the control group; crossmodal retardation for sound was 87 msec for the patient group as compared to 9 msec for the control group. Within-group comparisons showed that, for reactions to both light and sound, the patient group was significantly slower on crossmodal trials, than on ipsimodal trials, whereas the reaction times of the control group were not significantly different in these conditions (see Table 15).

For reactions to light, a significant subject group X intensity interaction was also found (see Table 13). The relationship between group and intensity is shown in Figure 7. For both groups, reaction times (averaged across modality shift conditions) tended to decrease as

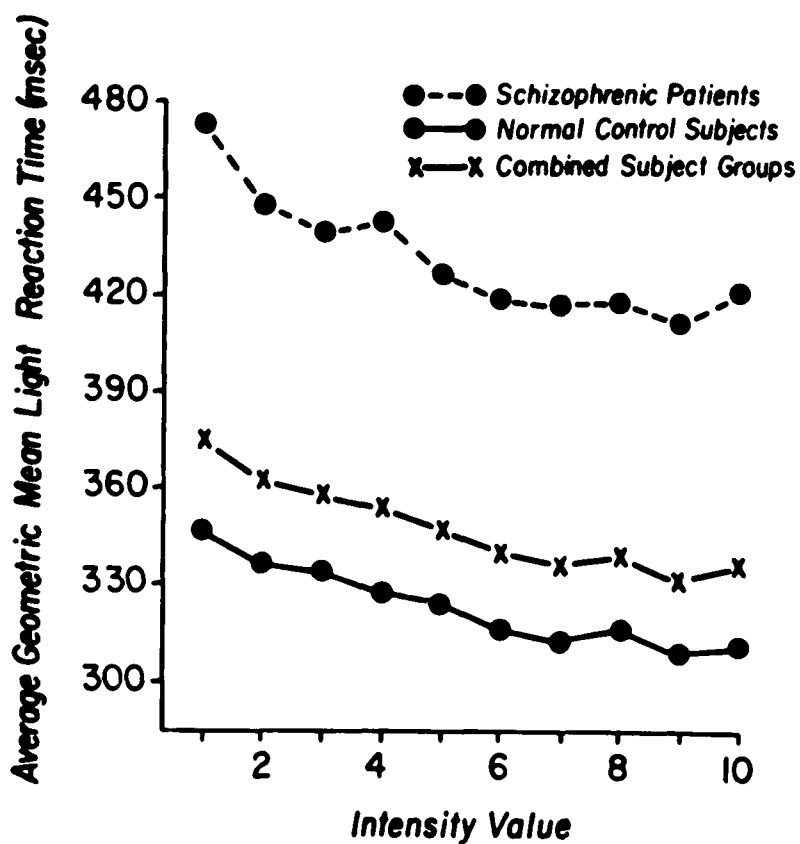


Figure 7. Reaction times of patient and control groups to light stimuli, as a function of intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. There were 15 schizophrenic patients and 50 normal control subjects.

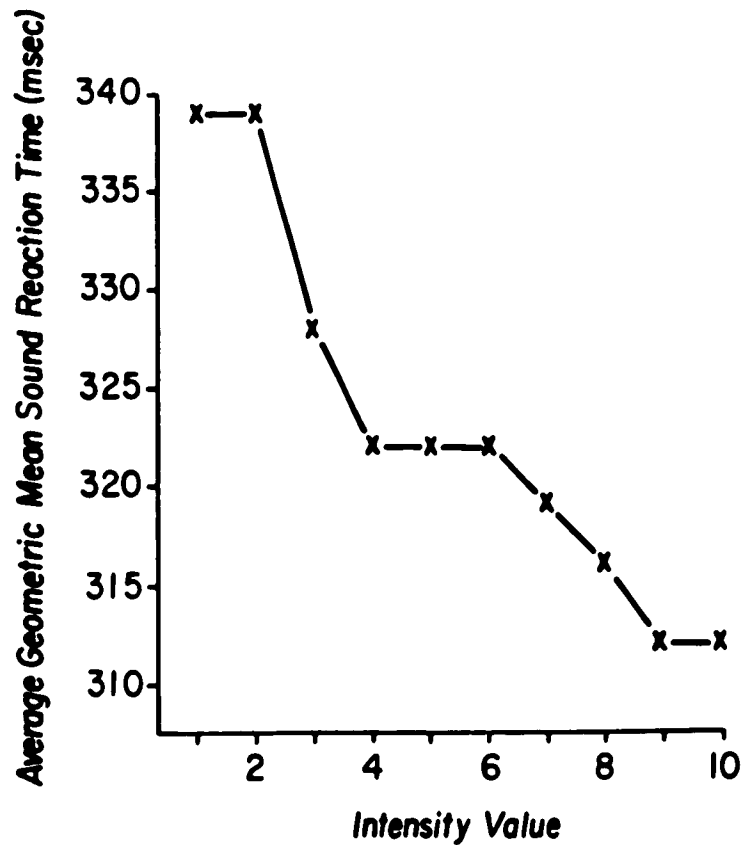


Figure 8. Reaction times to sound stimuli as a function of intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. Each point represents an average of the data from 65 subjects (15 schizophrenic patients and 50 normal control subjects).

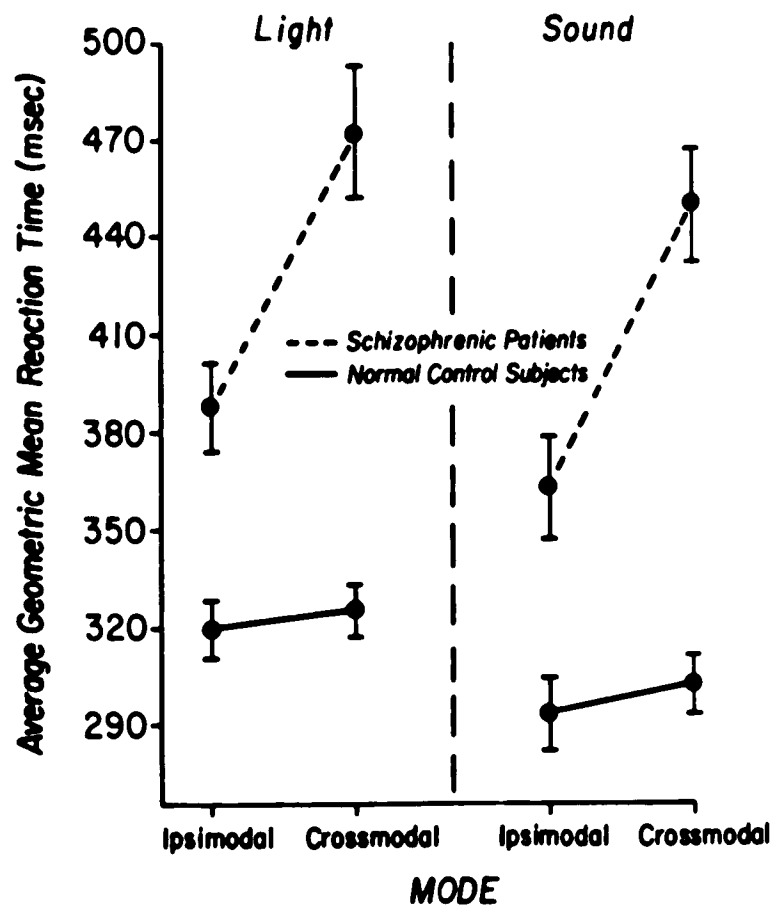


Figure 9. Reaction times of patient and control groups to light and sound stimuli, as a function of stimulus sequence (ipsimodal, crossmodal). There were 15 schizophrenic patients and 50 normal control subjects. Brackets indicate one standard error above and below each group mean.

Table 15
Within-Group Comparisons of
Crossmodal and Ipsimodal Reaction Times

Group/ Modality-Condition	Mean	<u>SD</u>	<u>df</u>	<u>t</u>	<u>p</u>
Patients					
Light-					
Crossmodal	473	77	----	----	----
Ipsimodal	388	56	14	4.91	<.001
Sound-					
Crossmodal	451	69	----	----	----
Ipsimodal	364	61	14	6.32	<.001
Controls					
Light-					
Crossmodal	326	60	----	----	----
Ipsimodal	320	66	49	.96	n.s.
Sound-					
Crossmodal	302	65	----	----	----
Ipsimodal	293	75	49	1.37	n.s.

Note. Each t test for related measures is based on the arithmetic means of 10 geometric means (one for each intensity) of each subject.

a function of increasing intensity. The significant $G \times I$ interaction indicates that the relationship between reaction times to (at least) two consecutive intensities, differs for patients and controls. Since patient and control functions in Figure 7 do not differ markedly (or systematically), and specification of the precise reason for the $G \times I$ interaction would not clarify any other relationships, additional analyses were not conducted.

For reactions to light, a significant $M \times I$ interaction was also initially found, and the $G \times M \times I$ interaction approached significance (see Table 13). However, after the degrees of freedom had been reduced by a factor of epsilon, these interactions were no longer significant at the 5% level. In addition, the nature of these interactions is not apparent in Figure 5. Therefore, it is possible that the $M \times I$ and $G \times M \times I$ interactions resulted from non-homogeneity.

Modality Shift Comparisons of Patients and Controls Within Intensities. To assess whether the modality shift effect was consistent across intensities, the data for each intensity value were treated as constituting separate experiments. Two-factor mixed model analyses of variance, with subject group (patient, control) as a between factor, and modality shift (crossmodal, ipsimodal) as a repeated-measures factor, were conducted for each intensity value. As shown in Table 16, for both light and sound analyses, all intensity values showed significant main effects for subject group and modality shift, and a significant interaction for subject group $\times$ modality shift. As noted above (see Figures 5 and 6): (1) in all conditions, patients responded more slowly than controls; (2) for both modalities and all intensities, pa-

Table 16
F-values of Analyses of Variance for Factors
 Subject Group (Patients, Controls) and Modality Shift (Crossmodal, Ipsimodal)
 Within Each Intensity Condition

Modality/ Source of Variation	Intensity Value									
	1	2	3	4	5	6	7	8	9	10
Light										
Subject Groups (G)	36.1***	35.0***	33.5***	40.6***	32.6***	36.2***	43.4***	33.1***	38.3***	38.7***
Modality Shift (M)	27.6***	29.6***	24.0***	27.4***	25.3***	35.4***	11.9**	26.8***	56.8***	42.4***
G x M	26.6***	16.4***	22.9***	22.3***	20.1***	29.4***	7.7**	21.6***	35.7***	28.0***
Sound										
Subject Groups (G)	31.3***	28.6***	30.6***	31.7***	34.9***	36.6***	26.2***	30.1***	35.5***	27.8***
Modality Shift (M)	26.9***	32.9***	34.8***	38.1***	40.7***	25.6***	47.1***	31.0***	34.0***	42.8***
G x M	10.0**	17.7***	25.0***	29.5***	27.6***	17.1***	20.0***	27.6***	29.9***	32.9***

Note. df = 1,63 for all F values. Epsilon adjustment is not applicable since df cannot be reduced further.

*p < .05

**p < .01

***p < .001

tients and controls responded more slowly on crossmodal trials than on ipsimodal trials, and; (3) for both modalities and all intensities, patients (compared to controls) showed a disproportionate slowing on crossmodal trials. In conclusion, a significant modality shift effect was found regardless of the modality, or of the intensity, of the imperative stimulus.

Crossmodal Retardation Across Intensities. The magnitudes of crossmodal retardation for each subject group and intensity value are shown in Figure 10. One-factor repeated-measures analyses of variance, with intensity value as the repeated-measures factor, were conducted on the crossmodal-ipsimodal difference scores of each subject, for each group and modality. As shown in Table 17, none of these analyses were significant. In summary, the magnitude of crossmodal retardation did not differ significantly, across intensity values for either the patients' or the control subjects' reactions to light or sound stimuli.

Crossmodal Retardation Within Subject Groups. Figure 11 shows the cumulative percentages of subjects in each group at different magnitudes of crossmodal retardation (averaged across intensities). For both light and sound stimuli, all of the 15 schizophrenic patients demonstrated positive crossmodal-ipsimodal difference scores. Stated differently, all patients responded more slowly on crossmodal trials than on ipsimodal trials. In comparison, zero or negative difference scores were exhibited by 16% of control subjects for light stimuli, and by 8% of control subjects for sound stimuli. In summary, the individual subject data corroborate the group comparisons: i.e., not only are group mean crossmodal retardation magnitudes for light and sound

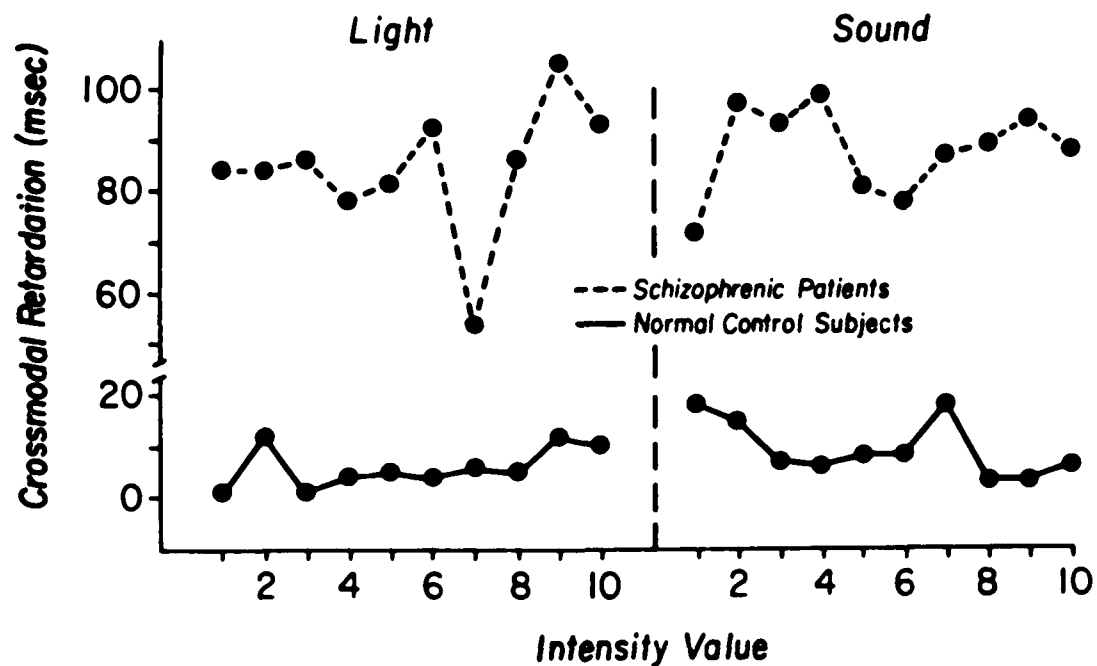


Figure 10. Crossmodal retardation for patient and control groups, as a function of intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection thresholds determined for one subject. Each point represents the differences between crossmodal and ipsimodal geometric mean reaction times averaged across subjects. There were 15 schizophrenic patients and 50 normal control subjects.

Table 17
Analyses of Variance for Effect of
Intensity on Crossmodal Retardation
for Each Subject Group and Modality

Group-Modality/ Source	<u>SS</u>	<u>df</u>	<u>ms</u>	<u>F</u>	<u>p</u>
Patients-Light/					
Total	831,669	149	-----	-----	----
Subjects	628,071	14	-----	-----	----
Intensity	23,642	9	2,627	1.84	n.s.
Error	179,956	126	1,428	-----	----
Patients-Sound/					
Total	677,587	149	-----	-----	----
Subjects	390,320	14	-----	-----	----
Intensity	15,745	9	1,749	.81	n.s.
Error	271,522	126	2,155	-----	----
Controls-Light/					
Total	1,225,125	499	-----	-----	----
Subjects	941,699	49	-----	-----	----
Intensity	7,677	9	853	1.36	n.s.
Error	275,749	441	625	-----	----
Controls-Sound/					
Total	1,319,304	499	-----	-----	----
Subjects	1,043,552	49	-----	-----	----
Intensity	9,386	9	1,043	1.73	n.s.
Error	266,366	441	604	-----	----

greater for the patient group, compared to the control group (see Figure 9), but a higher percentage of patients (100%) than controls, demonstrated crossmodal retardation.

Figure 11 also shows that, for reactions to light, 47% of the patients had larger magnitudes of crossmodal retardation than any control subject, and; for reactions to sound, 53% of the patients had larger magnitudes than any control subject. Also, for reactions to light, 54% of the control subjects (70% minus 16%) vs. 27% of the patients showed crossmodal retardation magnitudes between 0 msec and 20 msec; for reactions to sound, 58% of the control subjects (66% minus 8%) vs. none of the patients showed retardation magnitudes between 0 msec and 20 msec. In summary, about half of the patients had larger crossmodal retardation magnitudes than any control subject and, different from patients, the majority of control subjects exhibited crossmodal retardation magnitudes between 0 msec and 20 msec.

Pearson product-moment correlations between crossmodal-ipsimodal difference scores and ipsimodal reaction time were computed to assess the relationship between crossmodal retardation and overall reaction time level.¹⁵ As shown in Table 18, for both light and sound analyses, there was a significant, positive correlation between retardation and reaction time, but only for control subjects. Stated differently, the slower the control subject, the larger the magnitude of crossmodal retardation, and the faster the control subject, the smaller the magni-

¹⁵As shown in Figure 11, two normal control subjects (i.e., 4% of 50 subjects) exhibited very large, negative difference scores. These subjects were deleted from the correlational analyses, since their extreme scores would influence the correlation coefficient inordinately.

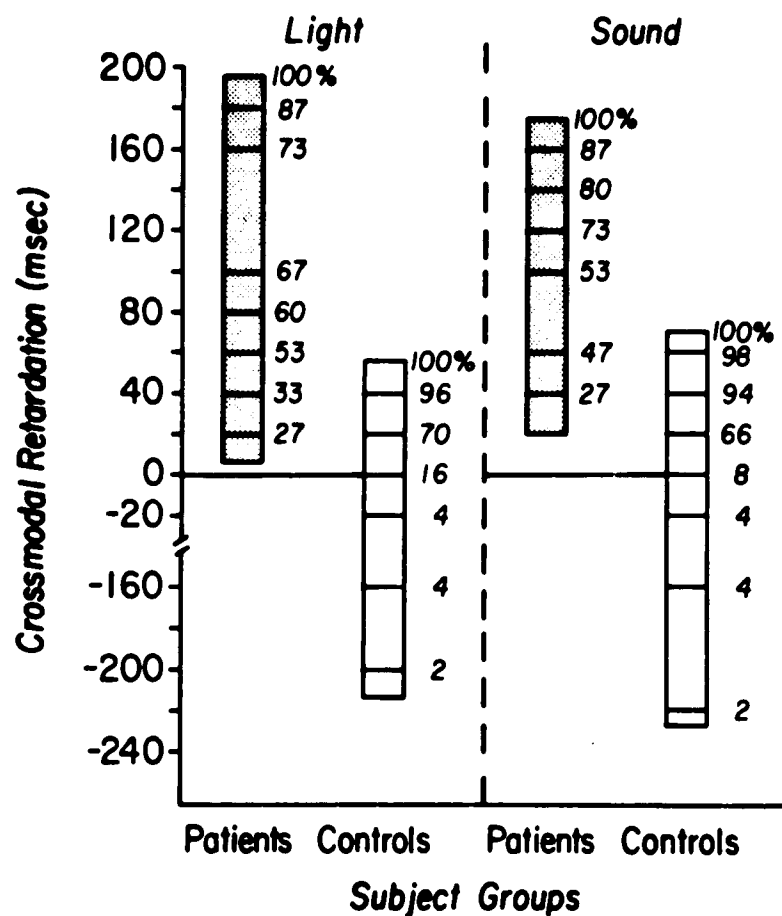


Figure 11. Cumulative percentages of 15 schizophrenic patients and 50 normal control subjects at different magnitudes of crossmodal retardation. Numbers to the right of each bar show the percentage of subjects in each group with crossmodal-ipsimodal difference scores less than or equal to the value shown on the ordinate.

Table 18
 Pearson Product-Moment Correlations between
 Crossmodal Retardation and Ipsimodal Reaction Time

Subject		
Group	Light	Sound
Patients ^a	-.23	-.26
Controls ^b	.48***	.37**

^adf = 13

^bdf = 46

**p < .01

***p < .001

tude of crossmodal retardation.

Pearson product-moment correlations between crossmodal-ipsimodal difference scores to light and sound stimuli were also computed. For both patients ($r(13) = .88, p < .001$) and for control subjects ($r(46) = .46, p < .001$), these correlations were highly significant. In summary, subjects who showed large retardation magnitudes to light stimuli, also showed large retardation magnitudes to sound stimuli, and vice versa (regarding small retardation magnitudes).

Crossmodal Retardation and Hospitalization Variables. Pearson product-moment correlations were calculated between the magnitude of crossmodal retardation and: (1) medication dosage (expressed in equivalent daily dosages of Chlorpromazine per kilogram of body weight); (2) number of previous hospitalizations, and; (3) duration of hospitalization. As shown in Table 19, none of these correlations were significant.

Ipsimodal Reaction Time Across Intensities. One reason for employing different intensity values in the current research was that several studies have shown a systematic relationship between intensity and reaction time. Stated differently, intensity was known to affect "accuracy of performance" on a reaction time "test." However, the difference between consecutive intensity values was arbitrarily chosen as .1 log unit. Thus, it was of interest to determine whether subjects did, in fact, "perform more poorly" on the "difficult items" (i.e., the lower intensities) of the "control test" (i.e., the ipsimodal condition).

The relationship between ipsimodal reaction time and intensity is shown in Figure 12 (light) and Figure 13 (sound). For both subject

Table 19
Pearson Product-Moment Correlations between
Crossmodal Retardation and Hospitalization Variables

Hospitalization		
Variable	Light	Sound
Medication		
Dosage (mg/kg body weight)	.08	-.07
Previous		
Hospitalization (<u>n</u>)	.06	.11
Duration of		
Hospitalization (days)	.35	.30

Note. None of the correlations are significant at the 5% level. df = 13 for all correlations.

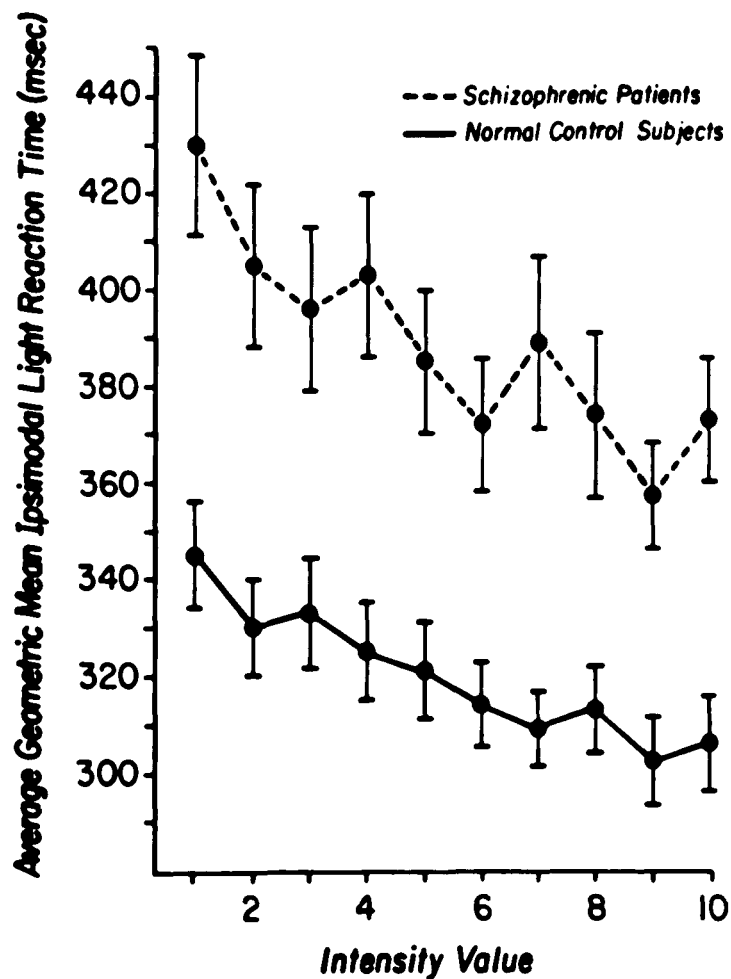


Figure 12. Ipsimodal reaction times of patient and control groups to light stimuli, as a function of intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. There were 15 schizophrenic patients and 50 normal control subjects. Brackets indicate one standard error above and below each group mean.

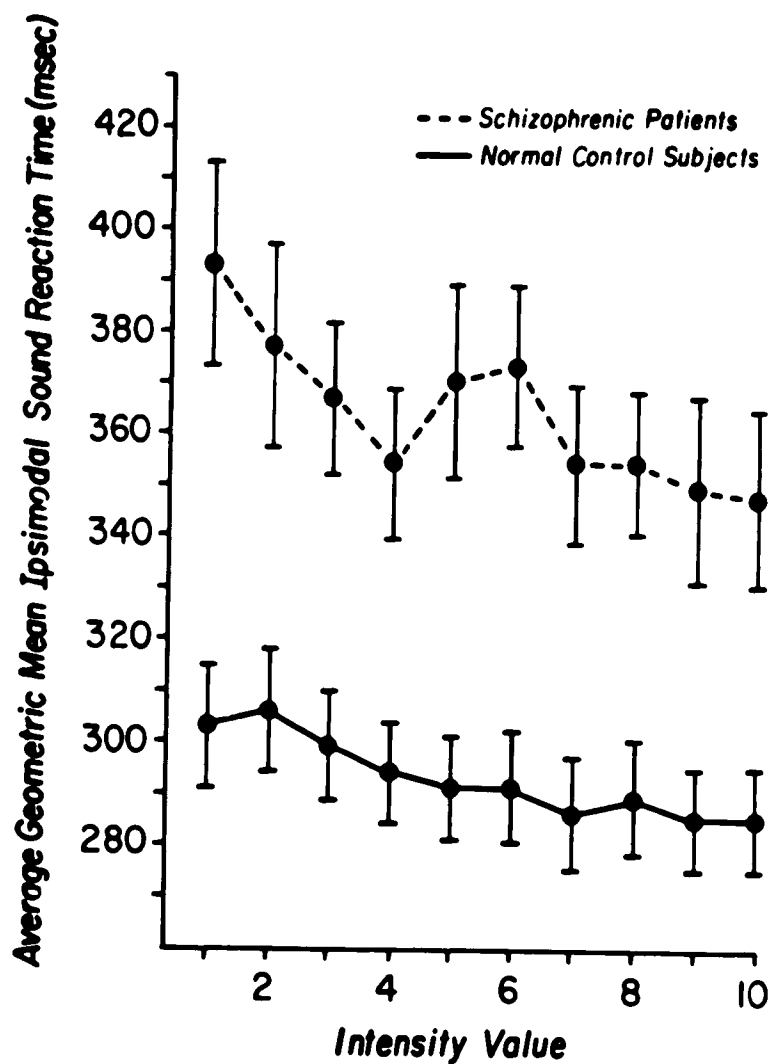


Figure 13. Ipsimodal reaction times of patient and control groups to sound stimuli, as a function of intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. There were 15 schizophrenic patients and 50 normal control subjects. Brackets indicate one standard error above and below each group mean.

groups and both modalities, ipsimodal reaction times tended to decrease as a function of increasing intensity. Trend analyses were conducted separately, for each subject group and modality, to determine whether there was a significant linear, quadratic, or cubic trend for ipsimodal reaction time across intensities. The results of these analyses are shown in Table 20. For both subject groups and both modalities, a significant linear trend is indicated. However, for the control group's reactions to ipsimodal light stimuli, a significant quadratic trend is also shown, indicating that there is a tendency for these data to deviate from a straight line in a curving manner (e.g., as in a parabolic arc). As shown in Figure 12, the function for normal control subjects does seem to be decelerating at the higher intensities.

Review of Stage 1 Results. The major objective of Stage 1 was to confirm the modality shift effect. This finding was demonstrated (in both light and sound analyses) by a significant subject group X modality shift interaction. Furthermore, a significant modality shift effect was found for each individual intensity value. In addition, within-group analyses for both modalities showed that patients were significantly retarded on crossmodal (compared to ipsimodal) trials, whereas control subjects were not. The relationship between intensity and crossmodal retardation was not significant. However, crossmodal retardation was significantly positively correlated with overall reaction time level, but only for control subjects. Also, for both subject groups, there was a tendency for reaction time to decrease as a function of increasing intensity. Finally, none of the correlations between crossmodal retardation and medication dosage, number of previous hospitalizations,

Table 20

Tests for Trends^a of Ipsimodal Geometric Mean Reaction Times
Across Intensity Values for Each Subject Group and Modality

Modality/ Subject Group	Linear		Quadratic		Cubic	
	<u>F</u>	<u>p</u>	<u>F</u>	<u>p</u>	<u>F</u>	<u>p</u>
Light						
Patients ^b	28.68	<.001	2.76	n.s.	.24	n.s.
Controls ^c	121.22	<.001	8.32	<.01	0.00	n.s.
Sound						
Patients ^b	10.30	<.01	.79	n.s.	4.20	n.s.
Controls ^c	45.34	<.001	3.70	n.s.	.26	n.s.

^aThis test conducts an orthogonal polynomial decomposition of the 10 levels of the Intensity factor into linear, quadratic, and cubic components.

^bdf = 1,14

^cdf = 1,49

or duration of hospitalization were significant for either modality.

The purpose of Stage 2 analyses is to determine whether a significant subject group X modality shift interaction is still shown after crossmodal and ipsimodal conditions are matched on reliability and variance.

Stage 2: Equating Conditions on Discriminating Power

Coefficient alphas¹⁶ were calculated for the crossmodal and ipsimodal conditions of the normal control subject groups. The results of these analyses showed that all alphas were identical: $\alpha = .99$ for both modality shift conditions and both modalities.

A t test for the difference between correlated variances (Guilford, 1965) was then conducted separately, for each modality. Results indicated that there were no significant differences between the variances of crossmodal 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 10 intensities and all 50 control subjects are analyzed, crossmodal and ipsimodal conditions are 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 "compe-

¹⁶Coefficient alpha is a reliability estimate of the internal consistency of a test (Anastasi, 1982). The calculation of coefficient alpha is discussed in Appendix K. The interpretation of coefficient alpha is elaborated on in the Discussion chapter.

tency." Analyses with "slow normal" subjects are pursued in Stage 3.

Stage 3: Further Assessment of Discriminating Power

Slow Normal Subgroups. Inspection of the data of Spring (1977) and of Kriegel (1967) indicates that about 20% of the normal control subjects in their experiments demonstrated mean reaction times slower than the patient means, and 30-50% of their control subjects showed mean reaction times that were within 1 standard deviation of the means for their patient groups. Based on these observations, 10-15 of the 50 normal control subjects in the present experiment were expected to perform in the schizophrenic patient range.

The following procedure was employed to form the two slow normal subgroups, one for each modality. The 15 normal control subjects who exhibited the slowest average geometric mean ipsimodal reaction times in each modality were tentatively chosen to constitute each subgroup. The ipsimodal reaction times and standard deviations of these subgroups are shown in Table 21. These data were then examined to confirm that the fastest ipsimodal reaction time (among each group of 15 reaction times) was within 1 standard deviation of the patient group mean for modality (i.e., to confirm that slow normal subjects did, in fact, perform within the patient range). Finally, t tests were conducted to determine whether the patient group and the slow normal subgroups differed significantly in the ipsimodal condition. As shown in Table 21, they did not.¹⁷

¹⁷If the fastest ipsimodal reaction time was not within 1 SD of the patient mean, or if patients and slow normals differed significantly in the ipsimodal condition, then the data of the 14 slowest controls would have been examined (then 13, 12, etc.).

Table 21

Average Geometric Mean Ipsimodal Reaction Times and Standard Deviations
of the Patient Group and the Slow Normal Subgroups

	Group		
Modality	Slow Normal	Patient	Difference ^a
Light			
Range	334 - 537	316 - 507	
Mean	398	388	
<u>SD</u>	61	56	<u>t</u> (28) = .47
Sound			
Range	313 - 539	267 - 469	
Mean	383	364	
<u>SD</u>	72	61	<u>t</u> (28) = .79

Note. All groups (patient, slow normal/light, and slow normal/sound) were made up of 15 subjects.

^aNeither of the comparisons was significant.

The two slow normal subgroups were not entirely made up of the same subjects, since the 15 control subjects who exhibited the slowest ipsimodal mean reaction times to light stimuli, were not the same subjects who exhibited the slowest reaction times to sound stimuli. However, 11 subjects were common to both subgroups.

The demographic characteristics of the patient group and the slow normal subgroups are shown in Table 22. There were no significant differences between the patient group and either subgroup on age, education, or ethnicity.

Reliabilities and Variabilities of the Slow Normal Subgroups. Co-efficient alphas were calculated for the crossmodal 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/crossmodal-- $\alpha = .98$; light/ipsimodal-- $\alpha = .99$; sound/crossmodal-- $\alpha = .98$; sound/ipsimodal-- $\alpha = .99$.

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 crossmodal and ipsimodal conditions for either light ($t(13) = .65$) or sound analyses ($t(13) = 1.07$).

Assessment of the Modality Shift Effect in Patients and Slow Normals. 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 (crossmodal, ipsimodal) and intensity (1 through 10) as repeated-measures factors. The results of these analyses are shown

Table 22
Demographic Characteristics of the Schizophrenic Patient Group
and the Slow Normal Control Subgroups

Variable	Light			Sound		
	Patients	Controls	Difference ^a	Patients	Controls	Difference ^a
Age (years)						
Range	18 - 48	18 - 42		18 - 48	18 - 45	
Mean	29.6	28.7		29.6	28.2	
<u>SD</u>	8.2	7.7	$\underline{t}(28) = .31$	8.2	9.1	$\underline{t}(28) = .45$
Education (years)						
Range	8 - 16	9 - 17		8 - 16	9 - 16	
Mean	11.9	12.7		11.9	12.3	
<u>SD</u>	2.5	3.0	$\underline{t}(28) = .72$	2.5	2.7	$\underline{t}(28) = .43$
Ethnicity						
White	7	6		7	6	
Hispanic	5	6		5	5	
Black	3	3	$\underline{\chi}^2(2) = .18$	3	4	$\underline{\chi}^2(2) = .22$

Note. All groups were made up of 15 subjects.

^aNone of the comparisons were significant.

in Table 23 (light) and Table 24 (sound).¹⁸ For both light and sound analyses, significant main effects were found for modality shift and intensity, and a significant interaction was found for subject group X modality shift.

The effect of intensity on reaction time is shown in Figure 14 (light) and Figure 15 (sound). For both modalities, there is a tendency for reaction times to decrease as a function of increasing intensity.

The subject group X modality shift interaction is portrayed in Figure 16. As indicated by the Stage 1 analyses, for both light and sound stimuli, the patient group was disproportionately retarded on crossmodal trials. In comparison, both slow normal subgroups exhibited what appeared to be a crossmodal facilitation (i.e., faster reaction times on crossmodal trials, than on ipsimodal trials). However, within-group comparisons showed that there was no significant difference between crossmodal and ipsimodal reaction times for either slow normal subgroup— light: $t(14) = .35$; sound: $t(14) = .19$. The actual crossmodal-ipsimodal differences for the slow normal subgroups were -7 msec for reactions to light, and -4 msec for reactions to sound.

Since patients and slow normals did not perform similarly under different levels of the modality shift factor (see Figure 16), the significant main effect for modality shift in both light and sound analyses

¹⁸Analyses of variance were also conducted on the untransformed data. As shown in Appendix I (Tables 43 and 44), for both light and sound analyses, the same results were found. Every significant main effect and interaction shown by the transformed data, was also found for the untransformed data. The results following epsilon adjustments were also the same for transformed and untransformed data.

Table 23

Analysis of Variance for Effects of
Group (Patients, Slow Normals), Modality Shift, and Intensity
on Geometric Mean Reaction Times to Light Stimuli

Source	<u>SS</u>	<u>df</u>	<u>ms</u>	<u>F</u>	<u>p</u>
Total	3,449,164	599	-----	-----	-----
Between Subjects	1,620,318	29	-----	-----	-----
Subject Groups (G)	195,373	1	195,373	3.84	n.s.
Error _b	1,424,945	28	50,891	-----	-----
Within Subjects	1,828,846	570	-----	-----	-----
Modality Shift (M)	225,932	1	225,932	8.55	<.01 ^a
Intensity (I)	185,432	9	20,604	28.21	<.001 ^b
G x M	310,674	1	310,674	11.76	<.002 ^c
G x I	3,967	9	441	.60	n.s.
M x I	8,234	9	915	1.43	n.s.
G x M x I	9,676	9	1,075	1.68	n.s.
Error ₁	739,937	28	26,426	-----	-----
Error ₂	184,020	252	730	-----	-----
Error ₃	160,974	252	639	-----	-----

^aepsilon adjustment not applicable. df cannot be reduced further.

^bepsilon adjustment-- df = 1,28; p < .001

^cepsilon adjustment not applicable. df cannot be reduced further.

Table 24

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Analysis of Variance for Effects of
Group (Patients, Slow Normals), Modality Shift, and Intensity
on Geometric Mean Reaction Times to Sound Stimuli

Source	<u>SS</u>	<u>df</u>	<u>ms</u>	<u>F</u>	<u>p</u>
Total	3,570,407	599	-----	-----	-----
Between Subjects	1,769,684	29	-----	-----	-----
Subject Groups (G)	106,533	1	106,533	1.79	n.s.
Error _b	1,663,151	28	59,398	-----	-----
Within Subjects	1,800,723	570	-----	-----	-----
Modality Shift (M)	261,125	1	261,125	11.11	<.005 ^a
Intensity (I)	94,302	9	10,478	10.86	<.001 ^b
G x M	314,692	1	314,692	13.39	<.001 ^c
G x I	5,013	9	557	.58	n.s.
M x I	3,464	9	385	.45	n.s.
G x M x I	6,041	9	671	.79	n.s.
Error ₁	658,298	28	23,511	-----	-----
Error ₂	243,120	252	965	-----	-----
Error ₃	214,668	252	852	-----	-----

^aepsilon adjustment not applicable. df cannot be reduced further.

^bepsilon adjustment-- df = 1,28; p < .01

^cepsilon adjustment not applicable. df cannot be reduced further.

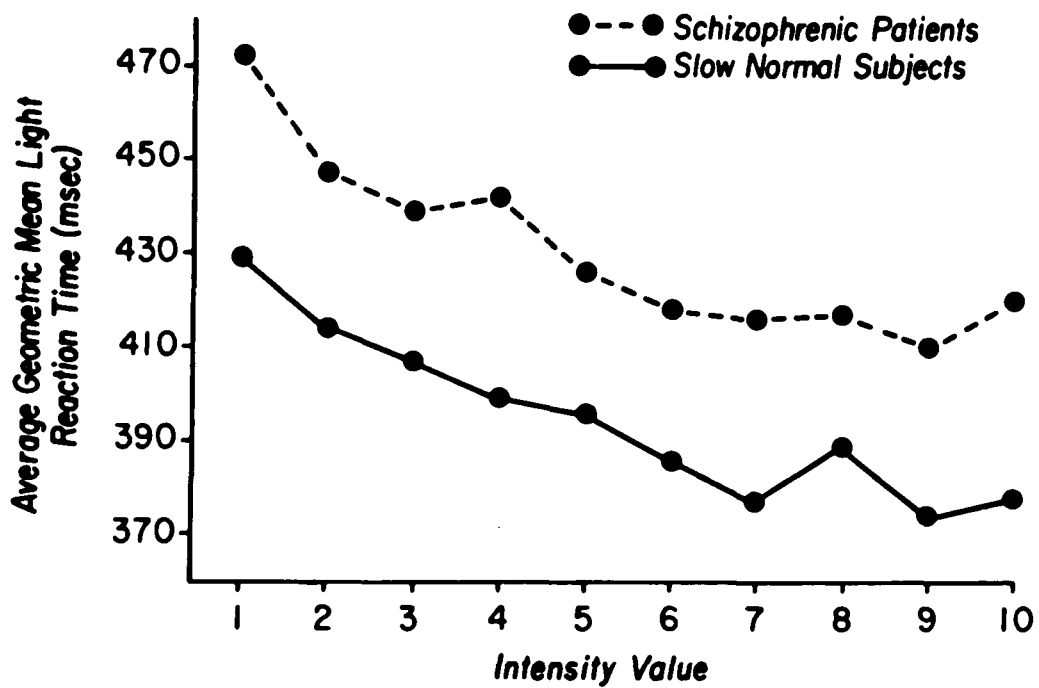


Figure 14. Reaction times of patient and slow normal groups to light stimuli, as a function of intensity. Intensity values range from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. There were 15 schizophrenic patients and 15 slow normal control subjects.

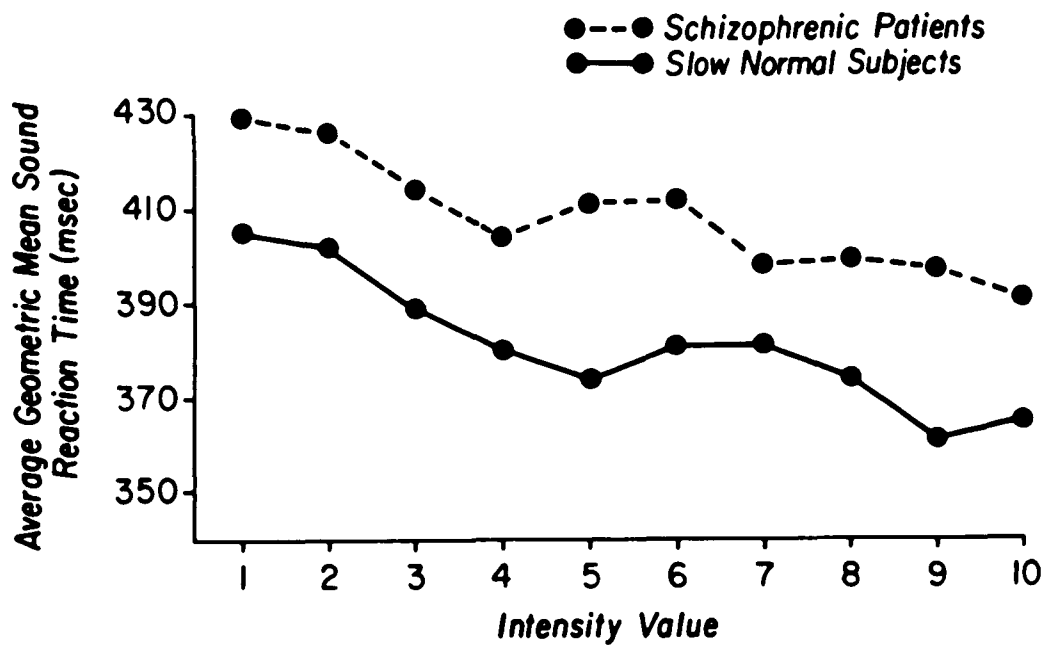


Figure 15. Reaction times of patient and slow normal groups to sound stimuli, as a function of intensity. Intensity values ranged from 1.8 (1) to 2.7 (10) log units above the absolute detection threshold determined for one subject. There were 15 schizophrenic patients and 15 slow normal control subjects.

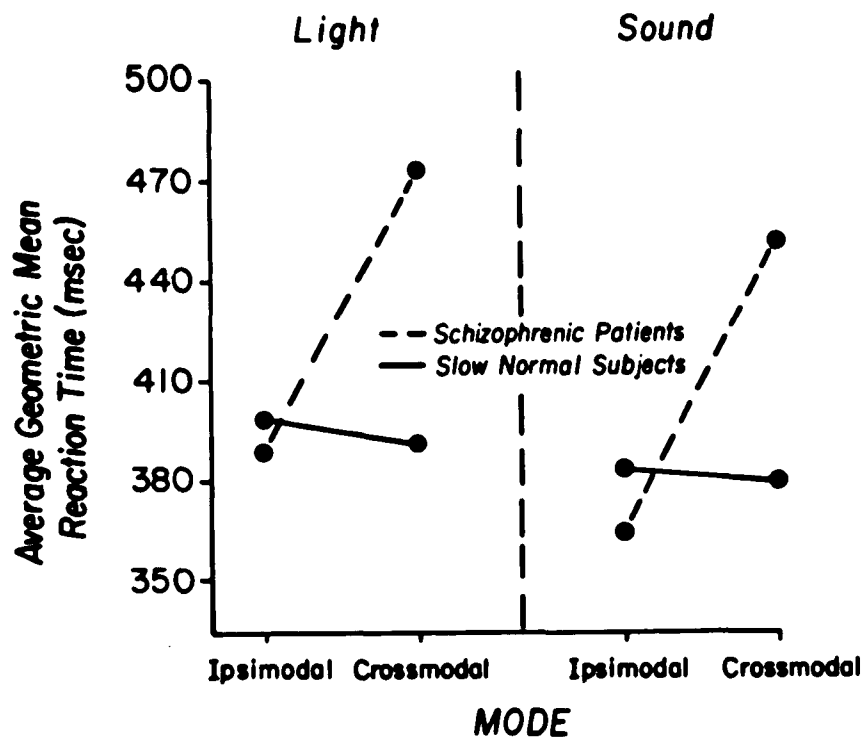


Figure 16. Reaction times of patient and slow normal groups to light and sound stimuli, as a function of stimulus sequence (ipsimodal, crossmodal). There were 15 schizophrenic patients and 15 slow normal control subjects.

(see Tables 23 and 24) is not informative.

Review of Stage 3 Results. The major objective of Stage 3 was to assess whether crossmodal and ipsimodal conditions remained matched (on reliability and variability) for a subgroup of slow-responding normal control subjects. This finding was demonstrated for both slow normal subgroups.

Stage 3 analyses also showed a significant modality shift effect when patients and slow normals were compared. This finding provides further evidence that generalized deficit cannot explain the effect.

Stage 4: Evaluation of Psychometric Artifact

In summary and conclusion, since both the entire normal control group of 50 subjects (Stage 2) and the slow normal subgroups (Stage 3) are already "matched" on reliability and variability, no trial deletion matching procedure is needed, and Stage 1 results (showing a significant modality shift effect for both modalities) cannot be attributed to psychometric artifact. This conclusion is further strengthened by the fact that, since all trials and all intensity values were employed, the results of the current research could not have capitalized on chance factors in the matching process. (No matching process was used.) Furthermore, the significant modality shift effect (for both modalities) in the patient-slow normal comparisons of Stage 3, provides additional evidence that neither generalized deficit nor psychometric artifact can account for the modality shift effect.

CHAPTER IV

DISCUSSION

The current research was designed to assess whether the modality shift effect¹⁹ could be attributed to psychometric artifact. More specifically, the principal question addressed was whether the disproportionate (i.e., greater-than-normal) slowing of schizophrenic patients on crossmodal trials, relative to ipsimodal trials, could be explained by the greater discriminating power of the crossmodal "test," coupled with the generalized performance deficit shown by schizophrenic patients.

Data analyses of the current research were initially planned to be conducted in four consecutive stages: (1) confirm the modality shift effect; (2) using the data of normal control subjects, match crossmodal and ipsimodal conditions on reliability and variance by selectively deleting reaction times to certain intensities; (3) confirm the matching for a subgroup of slow-responding normal control subjects, and; (4) re-evaluate the modality shift effect on matched conditions by deleting, from the patient data, the same trials that were deleted from

¹⁹Consistent with its use throughout the current report, "modality shift effect" (also, "positive modality shift effect," "significant modality shift effect") refers to a significant subject group (patients, controls) X modality shift (crossmodal, ipsimodal) interaction, showing patients disproportionately slowed on crossmodal trials. The meaning of the term is repeated here, to avoid confusion, since some authors have occasionally referred to a "significant modality shift effect," and have meant "significant crossmodal retardation" (i.e., within a single subject group, crossmodal reaction times are significantly slower than ipsimodal reaction times).

the control data in Stage 2, and comparing patients and controls on matched conditions.

The Modality Shift Effect and Psychometric Artifact

The major finding of the current research was that the modality shift effect shown in the present experiment could not be attributed to psychometric artifact. After the modality shift effect was confirmed by a significant subject group X modality shift interaction (Stage 1 of the analyses), reliabilities and variances of the crossmodal and ipsimodal reaction time distributions of control subjects were assessed. As indicated in the Introduction (see section on Principles of Psychometric Test Theory), the product of reliability and observed-score variance is true-score variance, and two tests have comparable discriminating power (i.e., they discriminate between more competent subjects and less competent subjects equally well) when the variances of error-free true scores of the two tests are comparable. Thus, the rationale for assessing the reliabilities and (observed-score) variances of the crossmodal and ipsimodal conditions ("tests") was to obtain estimates of true-score variance and, consequently, to evaluate whether these conditions displayed comparable discriminating power.

The data of both the entire normal control subject group (50 subjects), and of the slow normal subgroups (15 subjects), showed that the reliabilities of the crossmodal and ipsimodal conditions were high (coefficient alphas were .98- .99), and that the variance of these conditions did not differ significantly. Thus, a trial-deletion procedure for equating crossmodal and ipsimodal conditions on reliability and

variance was not needed since these conditions were already "matched on discriminating power."

In conclusion, the disproportionate slowing of schizophrenic patients on crossmodal (relative to ipsimodal) trials cannot be attributed to differences between the discriminating power of the crossmodal and ipsimodal conditions, coupled with the fact that schizophrenic patients are "less competent" subjects.

Several factors bolster this conclusion: (1) Since all trials and all intensity values were employed (i.e., crossmodal and ipsimodal conditions were already "matched" on discriminating power), results of the current research are not susceptible to the criticism that chance factors in the matching procedure could have contributed to the findings. (2) The modality shift effect was highly significant, and was consistent across modalities: for reactions to both light and sound stimuli, and for both transformed and untransformed data, the subject group X modality shift interaction was significant at the .001 level. (3) Within-intensity patient-control comparisons showed a significant subject group X modality shift interaction for all intensity values, and for both modalities. Therefore, the modality shift effect shown when all intensity values are analyzed together, did not result from the inordinate effects of one or two intensities; all "test items" contributed to the significant patient-control differences in modality shift. (4) The current research was the only crossmodal reaction time experiment in which stimuli of several intensities were used. One advantage of this aspect of the design was that it permitted an assessment of the patients' engagement in the task. For example, if patients responded randomly on

all trials, then no systematic relationship between intensity and reaction time would be expected. The findings that the ipsimodal reaction times of patients (as well as control subjects) tended to decrease as a function of increasing intensity, and that significant linear trends were shown in both light and sound analyses, suggest that patients of the current research were engaged in the task. All of the above factors contribute to the high degree of confidence with which conclusions can be drawn from the present data.

Generality of the Major Finding

To reiterate, the major finding of the current research was that the obtained modality shift effect could not be attributed to psychometric artifact. This conclusion was reached by establishing that the reliabilities and variances (and, thus, the discriminating power) of the crossmodal and ipsimodal conditions were comparable. One might ask the question of whether this conclusion can be generalized to other crossmodal reaction time experiments.

There is evidence to suggest that the modality shift effect shown in other experiments did not result from psychometric artifact. With respect to the reaction time variabilities of normal control subjects, previous investigations of the modality shift effect have shown relatively small differences between the standard deviations of the crossmodal and ipsimodal conditions (1- 8 msec in most experiments). In fact, the differences between the standard deviations of these conditions in the current research (6- 18 msec) were larger than those reported in other crossmodal reaction time experiments. These observa-

tions, in part, suggest that the crossmodal and ipsimodal conditions of previous investigations exhibited comparable discriminating power since the (observed-score) variances of these conditions probably did not differ significantly. (The generality of the results of the current research will be discussed further, in the section on Application of Psychometric Principles to Sensory Data.)

The Interpretation of the Reliability Coefficient

Coefficient alpha and the split-half reliability coefficient are sometimes referred to as coefficients of "internal consistency" since, different from test-retest and alternate-form estimates of reliability, alpha and split-half coefficients are based on a single administration of a single form of a test (Anastasi, 1982). Coefficient alpha, rather than a split-half estimate of reliability, was used in the current research since alpha is not based upon an arbitrary division of the data into two halves.

Theoretically, coefficient alpha represents the expected correlation of scores on the (administered) test with scores on another (parallel) test that measures the same construct or ability. The square root of coefficient alpha represents the estimated correlation of observed scores with error-free true scores (Nunnally, 1967).

Mathematically, coefficient alpha is a measure of "interitem consistency," i.e., the consistency with which different individuals respond to the various test items. A major source of error variance that influences interitem consistency is the heterogeneity of the behavior domain sampled. In general, the more heterogeneous the behavior domain,

the lower the interitem consistency (Anastasi, 1982). For example, a test consisting of vocabulary, arithmetic, history, and comprehension items is likely to show less interitem consistency than an arithmetic test consisting of multiplication, division, subtraction, and addition items since the behavior domain of the former test is more heterogeneous. Analogously, a reaction time task requiring differential responses (e.g., finger-lift, foot-lift, head-jerk) to visual, auditory, and tactile stimuli of varying intensities, frequencies, and durations, is likely to show less interitem consistency than a task requiring the same response to visual stimuli differing only in wavelength.

In the current research, the identical finger-lift response was required on all trials. In addition, all stimuli were 10 msec in duration. Furthermore, stimuli within each modality differed only in intensity. Therefore, it is not surprising that coefficient alphas were high (.98- .99), since the behavior domain sampled was relatively homogeneous.²⁰

The high values of alpha indicate that subjects performed consistently, across intensity values. If, for example, the relationship between reaction time and intensity was described by an ascending, monotonic function for some subjects, a descending, monotonic function for other subjects, and a nonmonotonic (e.g., parabolic) function for still

²⁰Other investigators have also reported high reliabilities for simple reaction time data. For example, Eckert and Eichorn (1976) tested groups of males and females ranging from 10 to 16 years of age. Reliability coefficients ranged from .85 to .97 for male age groups, and from .81 to .93 for female age groups. Hamsher and Benton (1977) reported reliability coefficients ranging from .85 to .97 for adult brain-damaged patients, and from .89 to .95 for control subjects.

others, then the intraindividual variances (i.e., for each intensity value, the distribution of the mean reaction times of each subject) would be inflated, and the reliability coefficient diminished.

It is not surprising that the reliabilities of reaction times on ipsimodal (light and sound) trials were high, since several experiments have shown a systematic relationship between response latency and intensity (King, 1962; Teichner & Krebs, 1972; Woodworth & Schlosberg, 1954). However, the relationship between intensity and reaction time on crossmodal trials has never been studied before. The finding that reliabilities were equally high on crossmodal trials suggests that the modality of the stimulus of the previous trial does not alter the systematic relationship between reaction time and intensity, despite the fact that it does influence the reaction times, themselves (i.e., for both modalities and all intensities, normal control subjects exhibited slower group mean reaction times on crossmodal trials, than on ipsimodal trials).

Application of Psychometric Principles to Sensory Data

The reliability data raise issues concerning the application of the Chapmans' methodological treatise (which was derived from psychometric test theory) to experiments in which sensory stimuli are viewed as "test items." It was noted above that, since coefficient alpha is a measure of interitem consistency, alpha would be low if the relationship between intensity and reaction time varied from subject to subject (ascending monotonic, descending-monotonic, parabolic, etc.). However, the relationship between intensity and reaction time is a well-

established finding, as are many other relationships within the sensory domain. Items on a vocabulary test, on the other hand, are within the "intellectual" domain, and the relative difficulty levels of any single set of these items must be empirically derived for the particular circumstances in which the items are being used. This point will now be made more explicit.

Consider the analogy (between the sensory domain and the "intellectual" domain) used in the current research. Ten intensities of auditory stimuli were viewed as 10 test items ranging in difficulty from 1, most difficult, to 10, least difficult. Similarly, 10 vocabulary items can be ordered in descending level of difficulty from 1 to 10. However, intensity is an inherent property of a stimulus, whereas, difficulty level is a relative "property" that varies with a host of factors. For example, if two individuals are asked to define Vocabulary Item 5 (medium difficulty) and 7 (somewhat less difficult), one individual might fail both items, whereas the other individual might pass both items. Many factors determine whether an item is passed or failed: e.g., the individual's level of schooling, courses that the individual completed, how well the individual learned certain course material, when the individual completed certain courses, exposure to certain material in the individual's home environment, scoring criteria, level of anxiety, adequacy of the testing environment, the individual's ability to express himself, etc., etc. If these individuals are now presented with 50 trials of auditory stimuli of Intensity Value 5 ("medium difficulty") and 7 ("less difficult"), and are asked to lift their finger from a key, as quickly as possible, when either stimulus

is presented, the mean reaction time to Intensity 5 is likely to be greater than the mean reaction time to Intensity 7 for both individuals. Although a limited number of factors (e.g., practice, fatigue, motivation) could contribute to the absolute levels of reaction times, the relationship between reaction times to the two stimulus intensities will be consistent for both individuals. In addition, doubling the duration of each stimulus, or increasing the frequency of each stimulus by a constant amount, will not alter this relationship.

The purpose of the above discussion was to illustrate that, different from paper-and-pencil test items, sensory stimuli follow certain laws that are relatively unaffected by individual differences between subjects, and by differences between one experiment and another. In view of the high degree of confidence that a sensory researcher has in trusting that the findings of a well-conducted, systematic investigation will generalize from one experiment to the next (i.e., that lawful relationships which have been convincingly demonstrated in normals, will be replicated in a subsequent experiment), perhaps it is less critical to require that sensory data fulfill highly conservative criteria (regarding discriminating power) in every single experiment conducted. Perhaps a more practical (yet, admittedly, a less theoretically idealistic) approach would be to provide partial evidence that two measures show comparable discriminating power, once it has been shown that psychometric artifact cannot account for a particular phenomenon. For example, the current research provided compelling evidence that, for both modalities, the reliabilities of reaction times on crossmodal and ipsimodal trials were equally high. Assuming that this finding is genu-

ine (i.e., that sequential changes in the modality of the stimulus do not alter the consistency with which individuals respond to different intensities), then if a similar experiment were conducted, and several patient groups (as well as a normal control group of 15 or 20 subjects) were tested, it would seem adequate to demonstrate that the crossmodal and ipsimodal (observed-score) variances of control subjects do not differ significantly. The alternative strategy (i.e., to test 50 or 100 control subjects, and to formally establish that the reliabilities, as well as the variances, are comparable) would compromise certain aspects of the study, since the time and energy devoted to testing additional control subjects probably would supplant the testing of one or two of the patient groups.

The Chapmans have objected to establishing a "match" on observed-score variance, alone, since the variance of observed scores, in part, reflects error variance. Thus, the Chapmans insist that the reliabilities (as well as the variances) of experimental and control measures must be adequately matched. However, what constitutes an "adequate match" on reliability? Are alpha coefficients of .93 and .90 adequately matched? What about coefficients of .97 and .90? At what point is the investigator forced to conclude that his findings are no longer free from an interpretation in terms of psychometric artifact?

The views expressed in this section are not meant to imply that the concept of psychometric artifact is unimportant, or inapplicable to certain types of experiments. Indeed, the principles that have been promoted by the Chapmans can be applied to any experiment in which patients and controls are compared on two measures. However, the issues

that have been raised concern the "margin of error" that is tolerable for an investigator to be fairly confident that his or her findings are free from psychometric artifact. These issues are not trivial. In comparison with paper-and-pencil tests, which can be group-administered, and which often require as little as 10-15 minutes to complete, sensory tasks must be individually administered, and often require hours to obtain data on a single subject. Therefore, strict adherence to the Chapmans' recommended methodology (i.e., establishing matched conditions on a large number of normals in every experiment conducted) is laborious, time-consuming, and expensive for experiments within the sensory domain. As a compromise, it was suggested that, once measures have been shown to exhibit "equivalent" discriminating power, in view of the consistency of sensory relationships across experiments (and the absence of absolute rules designating an "adequate match" on reliability), it would be acceptable for similar subsequent experiments to show that the variances of the measures are comparable.

The Modality Shift Effect and Stimulus Modality

The modality shift effect was found for reaction times to both light and sound stimuli. Furthermore, within-group analyses for both modalities showed that patients demonstrated significantly slower reaction times on crossmodal (compared to ipsimodal) trials, whereas normal control subjects (and slow normal subjects) did not.

Most studies have shown significant modality shift differences between schizophrenic patients and normal control subjects for reaction times to sound stimuli, but not to light stimuli (Sutton et al., 1961;

Sutton & Zubin, 1965, Experiments A, B, and C; Rist, Note 1). However, the two studies showing the modality shift effect for reaction times to both light and sound stimuli (Spring, 1980; Waldbaum et al., 1975) were the only experiments in which stimuli were equated with respect to sensation level. In the current research, light and sound stimuli were also equated with respect to sensation level, and the modality shift effect was, again, found for both modalities. Thus, as suggested by Sutton et al. (1978), it is possible that this procedural difference might explain the failure of earlier experiments to show the modality shift effect for both modalities.

The above conclusion is complicated by a second procedural difference across studies. In all of the crossmodal reaction time experiments that showed a significant modality shift effect for reactions to sound, but not to light, response-terminated stimuli were employed. Thus, stimulus duration in these studies varied from trial to trial, and from subject to subject, and; stimulus duration was necessarily longer in these studies, compared to the 10 msec stimuli used in the current research, and in the experiments of Spring (1980) and Waldbaum et al. (1975).

In summary, the most correct statement that can be made is that the modality shift effect is consistent across modalities when three conditions are met: (1) brief-duration stimuli are employed; (2) stimulus duration is held constant for all conditions and both modalities; and, (3) light and sound stimuli are equated with respect to sensation level.

The Modality Shift Effect and Psychotropic Medication

The results of the current research showed that there was no

significant correlation between drug dosage and crossmodal retardation to either light stimuli ($r(13) = .08$) or to sound stimuli ($r(13) = -.07$). These findings are consistent with those of other investigators. Spring (1980), who tested medicated schizophrenic and depressed patients, found that correlations between drug dosage and crossmodal retardation were not significant for either patient group, and for either modality (light or sound). Rist (Note 1), who also tested medicated schizophrenic patients, found significant correlations between dosage and retardation, but only for the modality (light) in which significant patient-control modality shift differences were not demonstrated.

In five additional crossmodal reaction time experiments, the modality shift effect was found, despite the fact that patients were drug free for a minimum of 1 week (Sutton & Zubin, 1965, Experiments A, B, and C; Waldbaum et al., 1975) or 2 weeks (Sutton et al., 1961) before testing.

In conclusion, the failure to find significant correlations between drug dosage and crossmodal retardation in three experiments, and the demonstration of the modality shift effect in five experiments employing drug-free patients, provide compelling evidence that psychotropic medication cannot account for the modality shift effect.

Comparisons with Spring and Kriegel Dissertations

In the concluding sections of this report, detailed comparisons are made between the results of the current research, and the findings of Spring (1977) and Kriegel (1967).²¹ These two experiments have been singled out for several reasons. In common with the current research, the experiments of Spring (1977) and Kriegel (1967) were doctoral dissertations in which male patient and control subjects were tested on a simple, crossmodal reaction time task which required a fingerlift response, and for which light and sound stimuli were equated with respect to sensation level. In addition, since these experiments were both doctoral dissertations, results were available in more detail (e.g., individual subject data) than is usually included in published articles. Thus, certain comparisons could be made that otherwise would not have been possible. The results of other crossmodal reaction time experiments are also cited in the following sections when relevant.

Overall Reaction Time Level Across Studies

Table 25 shows the average ipsimodal geometric mean reaction times and standard deviations for subject groups of the current research

²¹Spring (1980) is the published summary of the results of Spring's (1977) doctoral dissertation. Waldbaum et al. (1975) is the published summary of the results of Kriegel-Waldbaum's (1967) doctoral dissertation. It is more correct to cite the doctoral dissertations, rather than the published articles, in the following sections since certain results were not reported in the published articles. Therefore, reference is made to Spring (1977) and to Kriegel (1967).

Table 25
Average Ipsimodal Geometric Mean Reaction Times (msec) and Standard Deviations
for Each Subject Group in the Experiments of Mannuzza (1982), Spring (1977), and Kriegel (1967)

Investigator	Light				Sound			
	Schizo- phrenic Patients	Normal Controls	Depressed Patients	Siblings of Schizo.	Schizo- phrenic Patients	Normal Controls	Depressed Patients	Siblings of Schizo.
Mannuzza ^a								
Mean	388	320	-----	-----	364	293	-----	-----
<u>SD</u>	56	66	-----	-----	61	75	-----	-----
Spring ^b								
Mean	354	233	392	231	308	189	360	193
<u>SD</u>	126	34	138	50	120	37	149	57

Table 25 (continued)
Average Ipsimodal Geometric Mean Reaction Times (msec) and Standard Deviations
for Each Subject Group in the Experiments of Mannuzza (1982), Spring (1977), and Kriegel (1967)

Investigator	Light				Sound			
	Schizo- phrenic Patients	Normal Controls	Depressed Patients	Siblings of Schizo.	Schizo- phrenic Patients	Normal Controls	Depressed Patients	Siblings of Schizo.
Kriegel ^c								
Uncertain								
Mean	275	219	-----	-----	235	170	-----	-----
<u>SD</u>	62	26	-----	-----	66	28	-----	-----
Certain								
Mean	220	204	-----	-----	180	163	-----	-----
<u>SD</u>	43	21	-----	-----	45	18	-----	-----

^aThere were 15 schizophrenic patients and 50 normal control subjects.

^bThere were 19 schizophrenic patients, 19 normal control subjects, 16 depressed patients, and 15 siblings of schizophrenic probands.

^cThere were 11 schizophrenic patients and 11 normal control subjects.

(Mannuzza, 1982), and of the experiments of Spring (1977) and Kriegel (1967).²² For all three experiments: schizophrenic patients responded more slowly than normal control subjects to both light and sound stimuli, and; reaction times to sound stimuli were faster than reaction times to light stimuli for all subject groups.

Kriegel's subjects were faster than the subjects of Mannuzza and of Spring. There are at least two possible reasons for this difference in overall reaction time level across experiments. First, patients in the Mannuzza and the Spring experiments were taking psychotropic medication at the time of testing, whereas patients in the Kriegel experiment were drug-free for a minimum of 1 week. Mean daily drug dosage for Mannuzza's patient group was 19.4 mg/kg ($SD = 12.8$); for Spring's groups, mean dosage was 14.3 mg/kg ($SD = 13.4$) for schizophrenic patients, and 12.1 mg/kg ($SD = 10.1$) for depressed patients.²³ Second, Kriegel's light and sound stimuli were set and maintained at 2.9 log units above threshold; Spring's stimuli were set at 2.0 log units, and Mannuzza's stimuli ranged from 1.8 to 2.7 log units above threshold. In summary, higher intensity stimuli were employed by Kriegel, and drug-free patients were tested.

Table 25 also shows that normal control subjects of the current research were much slower and more variable than the control subjects of Kriegel (1967) and Spring (1977). There are at least two possible

²²Some of the information presented in this section, and in the following section, is based on calculations of the data included in Appendix D of Spring (1977) and in Appendix B of Kriegel (1967).

²³It should be noted that correlations between daily drug dosage and ipsimodal reaction time were not significant in either experiment.

reasons for this difference across studies. First, Kriegel and Spring employed a subject-initiation procedure in which the subject pressed the response key when he was ready to begin each trial. The preparatory interval for each trial was not instituted (by the apparatus) until the response key was pressed. In the current research, interstimulus interval was independent of the subject's behavior. In general, subject-initiation (as used by Kriegel and Spring) tends to stabilize reaction time performance.

A second possible reason why control subjects of the current research were slower and more variable than those of Kriegel and Spring concerns the number of (different) stimuli used in the three experiments. Whereas Kriegel and Spring used two stimuli (a light and a sound), 20 stimuli (10 intensities of light and 10 intensities of sound) were used in the current research. In general, the inclusion of multiple stimuli in a block of trials tends to lengthen reaction time, and to increase reaction time variability.

For reaction times to both light and sound stimuli, the intragroup variability of Spring's patients was much greater than those shown by the patient groups of Mannuzza and Kriegel. One possible reason for this difference is that Spring analyzed all reaction times from 1 msec to 1999 msec. Limiting values of 100 msec and 1000 msec were employed in the current research, and of 80 msec and 1000 msec were used by Kriegel. Therefore, reaction times between 1 sec and 2 sec could have accounted for the greater intragroup variabilities shown by Spring's patients.

Crossmodal Retardation Within Subject Groups

Overview. Table 26 includes information on the crossmodal-ipsimodal difference scores of each subject group in the current research (Mannuzza, 1982), and in the experiments of Spring (1977) and Kriegel (1967). For all three experiments and both modalities, mean crossmodal retardation magnitudes were greater for the schizophrenic patient group than for the normal control subject group. In addition, with the exception of the normal control subjects in Kriegel's Sound-Uncertain and Sound-Certain conditions: mean difference scores were positive (i.e., slower reaction times to crossmodal stimuli, than to ipsimodal stimuli) for both the schizophrenic patients and the normal control subjects in all experiments, and; the majority of subjects in all groups of all three experiments exhibited crossmodal retardation (see % Positive). Furthermore, with the exception of the Light-Certain condition in Kriegel's experiment, the percentage of schizophrenic patients who demonstrated crossmodal retardation, was greater than the percentage of normal control subjects who did. Finally, patient mean crossmodal-ipsimodal difference scores were larger in Kriegel's Uncertain condition (light, 32 msec; sound, 20 msec), than in her Certain condition (light, 18 msec; sound, 10 msec).

Table 26 also shows that the average magnitudes of crossmodal retardation for the schizophrenic group of the current research (light, 85 msec; sound, 87 msec) were much larger than those found by Spring and Kriegel (range of schizophrenic patient means: 10 msec to 32 msec). One interpretation of this difference across studies concerns variability: patients were more variable in the current research, and the

Table 26
Crossmodal Retardation (msec) for Each Group
in the Experiments of Mannuzza (1982), Spring (1977), and Kriegel (1967)

Investigator	Light				Sound			
	Schizo- phrenic Patients	Normal Controls	Depressed Patients	Siblings of Schizo.	Schizo- phrenic Patients	Normal Controls	Depressed Patients	Siblings of Schizo.
Mannuzza ^a (Uncer.)								
Mean	85 (67)	6 (44)	-----	----	87 (53)	9 (46)	-----	----
% Positive	100	84	-----	----	100	92	-----	----
Spring ^b (Certain)								
Mean	16 (21)	6 (11)	5 (32)	0 (11)	12 (26)	4 (8)	8 (23)	0 (9)
% Positive	84	63	69	60	79	63	62	60

Table 26 (continued)
Crossmodal Retardation (msec) for Each Group
in the Experiments of Mannuzza (1982), Spring (1977), and Kriegel (1967)

Investigator	Light				Sound			
	Schizo- phrenic Patients	Normal Controls	Depressed Patients	Siblings of Schizo.	Schizo- phrenic Patients	Normal Controls	Depressed Patients	Siblings of Schizo.
Kriegel ^c								
Uncertain								
Mean	32 (27)	5 (9)	----	----	20 (28)	0 (6)	----	----
% Positive	82	73	----	----	64	45	----	----
Certain								
Mean	18 (13)	7 (8)	----	----	10 (11)	0 (5)	----	----
% Positive	91	91	----	----	82	36	----	----

Note. Numbers in parentheses refer to standard deviations of difference scores. % Positive refers to the percentage of subjects with slower crossmodal, than ipsimodal, mean reaction times.

^aThere were 15 schizophrenic patients and 50 normal control subjects.

^bThere were 19 schizophrenic patients, 19 normal control subjects, 16 depressed patients, and 15 siblings of schizophrenic probands.

^cThere were 11 schizophrenic patients and 11 normal control subjects.

greater the variability, the larger the patient group mean crossmodal retardation. However, this interpretation does not explain why the mean crossmodal-ipsimodal difference scores of normal control subjects in the current research (particularly for reaction times to light) were comparable to those found by Spring and Kriegel, despite large differences in variability (see Table 26) and in overall reaction time level (see Table 25) across studies. Possible reasons for the large magnitudes of crossmodal retardation exhibited by patients in the current research will be discussed in the next two sections.

Crossmodal Retardation and Uncertainty. As indicated above, for both light and sound, the magnitude of crossmodal retardation shown by Kriegel's patient group tended to be greater in her Uncertain (compared to her Certain) condition. Subjects of the current research were not forewarned which stimulus would be presented on each trial: i.e., subjects were "uncertain" on all trials. In contrast, Spring's subjects were informed which stimulus (a light or a sound) would be presented on each trial. As shown in Table 26, for both modalities, the mean crossmodal retardation magnitudes of schizophrenic patient groups across studies were largest for the current research (uncertain) and the Uncertain condition of Kriegel's experiment, and were smallest for Spring's experiment (certain) and the Certain condition of Kriegel's experiment. These trends suggest that the differences in patient crossmodal retardations across experiments, at least in part, could be related to uncertainty. However, there is still a three- to four-fold difference between the magnitudes of crossmodal retardation for the patient group of the current research (light, 85 msec; sound, 87 msec) and for the pa-

tient group in Kriegel's Uncertain condition (light, 32 msec; sound, 20 msec). Consider the following explanation. Uncertainty in Kriegel's experiment involved whether a light or a sound would be presented. Uncertainty in the present research included the additional factor of not knowing the intensity of the stimulus that would be presented. More specifically, Kriegel's subjects did not know which of two stimuli (a light or a sound) would be presented, whereas subjects of the current research did not know which of 20 stimuli would be presented. Perhaps the degree of uncertainty is related to magnitude of crossmodal retardation.

A systematic evaluation of the relationship between crossmodal retardation and degree of uncertainty could consist of several Certain and Uncertain blocks of trials, including different numbers of intensities of (light and sound) stimuli. If magnitude of crossmodal retardation is related to degree of uncertainty, then crossmodal retardation should increase from the Uncertain block including the least number of different stimuli (e.g., a light and a sound) to the Uncertain block including the most number of stimuli (e.g., 20 intensities of light and sound stimuli). Crossmodal retardation on Certain blocks should remain the same, since the current research showed no relationship between intensity and the magnitude of retardation.

Two points are worth repeating. First, there is no apparent relationship between degree of uncertainty and crossmodal retardation for normal control subjects. Mean crossmodal-ipsimodal difference scores for the normal control group of the current research, and of Spring's and Kriegel's experiments ranged from 0 msec to 9 msec (see Table 26).

Thus, in these experiments, the influence of uncertainty on retardation seems to be limited to the patient groups. Second, uncertainty, alone, cannot account for the significant modality shift differences between patients and controls, since both Kriegel (certain condition) and Spring found the modality shift effect when all uncertainty was removed.

One might speculate why a higher degree of uncertainty should result in a greater retardation in patients. Perhaps, by increasing the degree of uncertainty, physiological changes occur in the schizophrenic, and these changes further impair his ability to shift attention. For example, perhaps when the schizophrenic is placed in a "relatively more uncertain" situation, he becomes aroused, and increased arousal, in turn, exacerbates his attentional shift deficit. Note that this proposal suggests that degree of arousal, rather than degree of uncertainty, is a key factor in bolstering crossmodal retardation in patients. Thus, for example, if schizophrenic patients are injected with a central nervous system stimulant, crossmodal retardation should increase, regardless of whether the forthcoming stimulus is known, or not (i.e., regardless of whether these patients are tested on Certain blocks, or on Uncertain blocks). Conversely, crossmodal retardation should decrease on Uncertain (and Certain) blocks if patients are injected with a central nervous system depressant.

It has been noted that degree of uncertainty does not seem to influence the magnitude of crossmodal retardation in normals, and that uncertainty, alone, cannot account for the significant modality shift differences between normals and schizophrenics. These observations suggest that some process (unrelated to uncertainty) is responsible for

the modality shift effect, and that some other process (related to uncertainty) is differentially affected in patients, when degree of uncertainty is increased. However, if arousal is a key variable, then an alternative explanation is tenable. Perhaps the relationship between crossmodal retardation and arousal is described by a U-shaped function, and that schizophrenics and normals (in their "resting" states) occupy different positions on the U: normals, near the base, and schizophrenics, on the ascending leg. Thus, when patients and controls are tested in the usual crossmodal reaction time experiment, differences in crossmodal retardation are found. When degree of uncertainty (and, presumably, arousal) is increased, then crossmodal retardation should increase for both subject groups. However, if normals are located at the base (i.e., the flatter portion) of the U, then degree of uncertainty would have to be increased by a relatively greater amount before changes are exhibited in crossmodal retardation. This would explain why the data presented in Table 26 suggest that there is no relationship between uncertainty and retardation in normals: normals in these studies might not have been sufficiently uncertain.²⁴

The purpose of the present section was to point out one possible reason (degree of uncertainty) for the large magnitudes of crossmodal retardation exhibited by patients in the current research. The discussion of arousal and retardation admittedly represents a huge theoretical

²⁴The proposed relationship between arousal and retardation leads to an interesting prediction: by decreasing arousal, the magnitude of crossmodal retardation should decrease in patients, and increase in normals.

leap from the data reviewed. However, this discussion does illustrate how a yet-unexplored variable can be used to explain crossmodal retardation, in general, and the modality shift effect, in particular. Similar theories can be proposed (and tested) for variables other than arousal.

Crossmodal Retardation and Magnitude of Difference. A second possible reason for the large degrees of crossmodal retardation demonstrated by patients in the current research concerns the "magnitude of difference" hypothesis. According to this hypothesis, schizophrenic patients are disproportionately affected by degree of stimulus difference (whether within or across modalities) on successive trials, rather than by trial-to-trial changes in stimulus modality (see section on Magnitude of Difference Hypothesis, p. 27).

In the current research, 10 intensities of light and sound stimuli were employed. Since each stimulus preceded and followed every stimulus (including itself) exactly once, there was a single L_1S_1 trial sequence, a single L_2S_2 sequence, a single L_3S_3 sequence, etc. Most crossmodal trials reflected a relatively larger "magnitude of difference" with respect to sensation level (e.g., $L_{10}S_1$, L_9S_1 , ..., L_2S_1 are "more different" than L_1S_1). Spring (1977) and Kriegel (1967) used one light stimulus and one sound stimulus equated for sensation level. Thus, "magnitude of difference" was identical on all of their crossmodal trial sequences (either L_xS_x or S_xL_x). Perhaps, in the current research, the larger discrepancies between relative sensation levels of light and sound stimuli on crossmodal trials, bolstered crossmodal retardation magnitudes. More specifically, the magnitude of difference hypothesis

predicts a disproportionate slowing (even greater than that seen in most studies) of patients on, for example, an L_1S_{10} trial, since the stimulus of the previous trial (L_1) and the stimulus of the present trial (S_{10}) in this sequence, differ with respect to both modality (L-S) and to relative sensation level (1-10).

It can be argued that, although crossmodal trial sequences in the current research were "more different" than those found in experiments with only one light and one sound stimulus, ipsimodal (nonidentical) sequences were also "more different." Therefore, the influence of ipsimodal retardation should offset the impact of retardation on large-difference (e.g., L_1S_{10}), crossmodal trials. More explicitly, crossmodal retardation is represented by a difference score. If a constant is added to both the subtrahend and the minuend (i.e., if both ipsimodal and crossmodal reaction times are equally slowed by magnitude of difference), then the difference score (crossmodal retardation) should remain unchanged. However, the comparative influences of magnitudes of difference within and across stimulus modalities have never been systematically studied.

A systematic evaluation of the magnitude of difference hypothesis might include crossmodal and ipsimodal-nonidentical blocks of trials with two, randomly ordered stimuli differing in intensity (and in modality, for crossmodal blocks). For example, one block might contain S_1 and S_5 stimuli; another block, S_1 and S_{20} stimuli; a third block, L_5 and S_{10} stimuli; etc.²⁵ The magnitude of difference hypothesis would

²⁵Note that the proposed design would control for (i.e., hold constant) the degree of uncertainty, since only one of two stimuli could be presented on any single trial (in any block).

predict: (1) the degree of retardation would be largest in crossmodal blocks that include the "most different" stimuli (e.g., S_1-L_{20} and $S_{20}-L_1$ blocks), and smallest in ipsimodal-nonidentical blocks that include the "least different" stimuli (e.g., S_1-S_5 and S_5-S_1 blocks); (2) retardation would be larger for certain ipsimodal blocks (e.g., $L_{20}-L_1$) compared to certain crossmodal blocks (e.g., S_1-L_1), and; (3) the differentiation between patients and controls would be greatest for those blocks showing the largest degrees of retardation (i.e., patients would be disproportionately affected by magnitude of difference).

Neural Trace Theory, Magnitude of Difference, and Degree of Uncertainty

According to Zubin's neural trace theory (see p. 23 of the Introduction), with each presentation of a stimulus, facilitory and inhibitory traces remain within the organism's neuron pool, and have an effect on the processing of future stimuli. More specifically, the processing of similar stimuli is facilitated, and the processing of dissimilar stimuli is inhibited, by these traces. Zubin proposed that schizophrenics are disproportionately retarded on crossmodal trials because both facilitory and inhibitory traces persist longer in their nervous systems.

Although Zubin's neural trace theory was formulated to account for a specific performance anomaly demonstrated by schizophrenic patients (the "modality shift" effect), the theory is not incompatible with the magnitude of difference hypothesis. Stated differently, perhaps the processing of similar (or, relatively more similar) stimuli is facilitated, and the processing of dissimilar (crossmodal or ipsimodal-

nonidentical) stimuli is inhibited, by these lingering neural traces.

Nor is the impact of uncertainty incompatible with a magnitude of difference (or a neural trace) explanation of the modality shift effect. Recall that, when all uncertainty is removed, the modality shift effect is still found (Kriegel, 1967, Certain condition; Spring, 1977).²⁶

Whether magnitude of difference, neural trace theory, and degree of uncertainty are all related to crossmodal retardation is an empirical question that can be tested, since the three are separable, experimentally. For example, according to neural trace theory, with the passage of time, the strength of trace residues will eventually dissipate. Thus, an inverse relationship between the duration of intertrial interval and the magnitude of crossmodal retardation is predicted. Therefore, intertrial interval can be systematically varied in a crossmodal reaction time experiment to evaluate the predictions made by neural trace theory, independent of the predictions made by the magnitude of difference hypothesis. Furthermore, experiments can be designed in which different theories predict opposite findings. For example, the probability of stimulus occurrence could be varied across blocks of trials (e.g., "In this block, a light or a sound will be presented on 25% of the trials."). If degree of uncertainty is related to the magnitude of crossmodal retardation, then retardation should increase with decreases in the probability of stimulus occurrence. However, neural trace theory predicts the opposite finding. The lower the percentage of stimuli in a block

²⁶Even if an arousal theory is proposed, neural trace theory is still a viable explanation of the modality shift effect. Perhaps increased arousal causes facilitory and inhibitory traces to persist longer in schizophrenics.

of trials, the higher the percentage of blank (i.e., no stimulus) trials, and the longer the period of time between one stimulus trial and the next. In essence, by decreasing the probability of stimulus occurrence, intertrial interval (or, more correctly, interstimulus interval) is increased. Thus, according to neural trace theory, the magnitude of crossmodal retardation should decrease with decreasing probability of stimulus occurrence since trace residues would be expected to dissipate.²⁷ In conclusion, properly designed experiments can answer several questions that might provide a better understanding of factors influencing the modality shift effect and crossmodal retardation.

Conclusion

The chronological history of crossmodal reaction time research in schizophrenia has followed a logical sequence of scientific inquiry. With each experiment, new information has been added to our knowledge of the modality shift effect-- certain hypotheses have been supported, artifactual explanations have been refuted, and past findings have been strengthened by replication. For example, studies have suggested that the modality shift effect is not limited to certain chronicity or category subtypes of schizophrenia: Sutton and his colleagues (1961) and Sutton and Zubin (1965, Experiment B) demonstrated the effect in chronic patients; Waldbaum et al. (1975) and Spring (1980) showed the effect in

²⁷A more systematic approach would be to group trials with respect to interstimulus interval in the data analyses.

acute patients; Sutton and Zubin (1965, Experiment A), in process and reactive patients, and; the current research showed no differences in crossmodal retardation between acute and chronic, or between paranoid and nonparanoid, schizophrenic patients. In addition, significant differences in crossmodal retardation have been found between schizophrenic and depressed patients (Spring, 1980), and between schizophrenic and alcoholic patients (Rist, Note 1), compared to normal control subjects. Also, results of the current research suggest that the modality shift effect is impervious to stimulus intensity changes: the magnitudes of crossmodal retardation did not differ significantly, across the 10 intensity values employed, and for each of the 10 intensity values, a significant modality shift effect was found. Furthermore, of the various explanations that have been proposed to account for the modality shift effect: psychotropic medication, patient-control differences in expectations of the forthcoming stimulus (Waldbaum et al., 1975), and psychometric artifact (the current research) have been refuted; whereas, the impact of different degrees of uncertainty, and of the magnitude of difference hypothesis (Rist, Note 1), warrant additional systematic investigation.

The disproportionate (i.e., greater-than-normal) slowing of schizophrenic patients on crossmodal (relative to ipsimodal) trials of a reaction time task is one of the few performance anomalies that has been repeatedly demonstrated (eight independent experiments) in the psychiatric research literature. Future investigators should take advantage of this fact by attempting to delineate the clinical significance (e.g., prognostic value, relationship to treatment response) as

well as the behavioral correlates (e.g., biochemical, psychophysiological) of the modality shift effect. Research in psychopathology is likely to be most beneficial (to both the scientist, and to the patient) if the relationship between consistent findings across areas of study is thoroughly investigated within a multidisciplinary framework.

APPENDIX A

Demographic Characteristics of Each Subject

This appendix includes the demographic characteristics of each of the 15 schizophrenic patients, and each of the 50 normal control subjects who participated in the present experiment. Information regarding age, education, and ethnicity is presented in Table 27 for patients, and in Table 28 for control subjects.

Table 27

Demographic Characteristics of Each Schizophrenic Patient

Subject Number	Age (years)	Education (years)	Ethnicity
29	48	8	Black
30	26	12	White
31	26	12	Hispanic
34	40	12	White
35	39	16	White
36	25	11	Black
40	20	8	Hispanic
41	30	10	Black
44	26	15	White
46	18	11	Hispanic
47	38	11	White
50	27	15	Hispanic
55	23	9	Hispanic
59	27	14	White
63	31	14	White

Table 28

Demographic Characteristics of Each Normal Control Subject

Subject Number	Age (years)	Education (years)	Ethnicity
01	18	12	White
02	21	16	White
03	40	9	White
04	45	16	White
05	25	16	White
06	30	14	White
07	33	14	White
08	23	13	White
09	31	14	Hispanic
10	21	12	Hispanic
11	30	16	White
12	31	13	White
13	27	12	White
14	36	17	White
15	33	15	White
16	29	12	White
17	27	19	White
18	18	12	White
19	31	12	White
20	19	10	Hispanic
21	20	11	Hispanic
22	26	10	White
23	23	12	White
24	49	12	White
25	28	16	White
26	30	12	White
27	36	19	White
28	32	14	White
32	18	10	Hispanic

Table 28 (continued)

Demographic Characteristics of Each Normal Control Subject

Subject Number	Age (years)	Education (years)	Ethnicity
33	19	11	Hispanic
37	36	17	White
38	32	9	Black
39	21	10	Black
42	37	12	Black
43	22	13	Black
45	41	10	Black
48	37	12	Hispanic
49	28	12	Black
51	27	11	Black
52	26	14	Hispanic
53	21	10	Hispanic
54	23	16	Hispanic
56	42	10	Hispanic
57	24	9	Hispanic
58	25	10	Black
60	31	12	Black
61	47	12	Black
62	37	14	Hispanic
64	26	10	White
65	31	12	White

APPENDIX B

RDC Criteria for Schizophrenia and its Subtypes

This appendix includes the criteria for Schizophrenia and its subtypes, according to the most recent edition of the Research Diagnostic Criteria (Third Edition, updated 6/80). Criteria for Schizophrenia are presented in Table 29. Criteria for category subtypes are presented in Table 30. Criteria for chronicity subtypes are presented in Table 31.

Criteria for Residual Schizophrenia are not presented in Table 30 since patients were not recruited for testing in the present experiment if they exhibited only residual symptoms.

RDC Criteria for Schizophrenia

A through C are required.

A. At least two of the following are required for Definite Schizophrenia:

- (1) Thought broadcasting, insertion, or withdrawal.
- (2) Control delusions, other bizarre delusions, or multiple delusions.
- (3) Somatic, grandiose, religious, nihilistic, or other delusions without persecutory or jealous content lasting at least one week.
- (4) Delusions of any type if accompanied by hallucinations of any type for at least one week.
- (5) Auditory hallucinations in which either a voice keeps up a running commentary on the subject's behaviors or thoughts as they occur, or two or more voices converse with each other.
- (6) Non-affective verbal hallucinations spoken to the subject.
- (7) Hallucinations of any type throughout the day for several days, or intermittently for at least one month.
- (8) Definite instances of marked formal thought disorder accompanied by either blunted or inappropriate affect, delusions or hallucinations of any type, or grossly disorganized behavior.

B. Signs of the illness have lasted at least two weeks from the onset of a noticeable change in the subject's usual condition.

C. At no time during the active period of illness did the subject meet the full criteria for either probable or definite manic or depressive syndrome to such a degree that it was a prominent part of the illness. (Underlines included in the RDC.)

RDC Criteria for Category Subtypes of Schizophrenia

(1) Paranoid

Throughout the active period of the episode of illness the clinical picture is dominated by the relative persistence of, or pre-occupation with, one or more of the following:

1. Persecutory delusions
2. Grandiose delusions
3. Delusions of jealousy
4. Hallucinations with a persecutory or grandiose content

(2) Disorganized (Hebephrenic): A through C are required.

A. Marked formal thought disorder

B. Either 1 or 2:

1. Affect which is shallow, incongruous, or silly
2. Fragmentary delusions or hallucinations

C. Not associated with marked emotional turmoil except during exacerbation

(3) Catatonic

Throughout the active period of the episode of illness the clinical picture is dominated by any of the following catatonic symptoms:

1. Catatonic stupor
2. Catatonic rigidity
3. Waxy flexibility
4. Catatonic excitement
5. Catatonic posturing

(4) Undifferentiated: Meets the criteria for more than one of the previous subtypes, or for none of them.

RDC Criteria for Chronicity Subtypes of Schizophrenia

(1) Acute Schizophrenia: A through C are required.

A. Sudden onset-- less than three months from first signs of increasing psychopathology to any of the core symptoms (see Criterion A, Table 29).

B. Short course-- continuously ill with significant signs of Schizophrenia (e.g., delusions, hallucinations, extreme social withdrawal) for less than three months.

C. Full recovery from any previous episode.

(2) Subacute Schizophrenia

Course is closer to that of Acute, than to that of Chronic: e.g., first episode with fairly rapid onset, and duration of five months; second episode with onset for this episode over a period of six months, and full recovery from first episode.

(3) Subchronic Schizophrenia

Course is closer to that of Chronic, than to that of Acute: e.g., significant signs of Schizophrenia more or less continuously present for at least the past year; second period following a previous period from which he did not fully recover.

(4) Chronic Schizophrenia

Significant signs of Schizophrenia more or less continuously present for at least the last two years.

APPENDIX C

Diagnostic, Drug, and Hospitalization Details of Each Schizophrenic Patient

This appendix includes the RDC category and chronicity subtypes, number of previous hospitalizations, duration of hospitalization, and type and dosage of medication for each of the 15 schizophrenic patients who participated in the present experiment.

Duration of hospitalization refers to date of testing minus date of admission. In some cases, subjects were not initially admitted to the New York State Psychiatric Institute, but rather were transferred to the Psychiatric Institute from another facility. For these cases, initial admission date was used to calculate Duration of Hospitalization.

For ease of inspection, brand names are entered under Name of Medication. Generic names are as follows: haloperidol (Haldol); fluphenazine (Prolixin); chlorpromazine (Thorazine); thiothixene (Navane).

Total daily medication dosages were translated to equivalent dosages of Thorazine in accordance with a comparative dosage table presented in Davis (1976, p. 859).

Table 32

Diagnostic, Drug, and Hospitalization Details of Each Schizophrenic Patient

Subject Number	RDC Category Subtype	RDC Chronicity Subtype	Number of Previous Hospitalizations	Duration of Hospitalization (days)	Name of Medication	Equivalent Daily Medication Dosage (mg/kg body weight)
29	Paranoid	Chronic	30	24	Haldol	16.8
30	Disorganized	Subacute	5	11	Prolixin	7.3
31	Paranoid	Acute	2	3	Thorazine	18.0
34	Paranoid	Acute	1	4	Haldol	16.0
35	Undifferentiated	Chronic	7	270 (9 months)	Prolixin	7.7
36	Disorganized	Subchronic	4	213 (7 months)	Haldol	49.9
40	Paranoid	Acute	2	3	Haldol	26.9
41	Paranoid	Subacute	10	211 (7 months)	Haldol	18.9
44	Undifferentiated	Subchronic	4	465 (1 year 3 months)	Navane	46.3
46	Undifferentiated	Acute	0	74	Haldol	20.7
47	Disorganized	Chronic	1	3650 (10 years)	Navane	10.0

Table 32 (continued)

Diagnostic, Drug, and Hospitalization Details of Each Schizophrenic Patient

Subject Number	RDC Category Subtype	RDC Chronicity Subtype	Number of Previous Hospitalizations	Duration of Hospitalization (days)	Name of Medication	Equivalent Daily Medication Dosage (mg/kg body weight)
50	Undifferentiated	Subchronic	4	2	Haldol	9.7
55	Paranoid	Subchronic	2	2	Haldol	9.8
59	Undifferentiated	Subacute	4	4	Haldol	17.5
63	Paranoid	Subacute	17	3	Thorazine	15.5

APPENDIX D

The SADS Summary Scales

This appendix includes the item content of eight summary scales that have been developed for analyzing data obtained from the Current section (Part I) of the Schedule for Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978). Each scale represents a descriptive dimension of psychopathology. Scale scores are obtained by adding the ratings of each item contributing to a particular scale. The eight scales are not mutually exclusive: i.e., certain SADS items are included in more than one scale. According to Endicott and Spitzer:

The item assignment was based on knowledge of factor analytic work of scales with similar item content, consideration of the major clinical distinctions usually made in research studies of affective and schizophrenic disorders, and a desire to have a smaller number of clinically meaningful measures on which to compare individual subjects over time or groups of subjects with each other. (p. 842)

Number of items contributing to each scale, range of scale scores, and scale composition are shown in Table 33.

Table 33
Item Content of the SADS Summary Scales

Scale Number and Name	Number of Items	Scale Score Range	SADS Items
1 Depressive Mood and Ideation	5	5 - 31	Depressed mood, Brooding, Feelings of self-reproach, Negative self-evaluation, Discouragement
2 Endogeneous Features	13	11 - 62	Distinct quality of mood, Lack of specific concern, Feelings of self-reproach, Insomnia (middle, terminal), Anorexia, Weight loss, Loss of interest, Agitation, Retardation, Reactivity, Morning depression
3 Depressive- Associative Features	17	14 - 88	Suicidal tendencies, Insomnia (initial, middle, termi- nal), Hypersomnia, Anergia, Anorexia, Weight loss, Appe- tite increase, Weight gain, Indecisiveness, Concentra- tion difficulties, Loss of interest, Agitation, Retard- ation, Depressed Appearance
4 Suicidal Ideation and Behavior	4	1 - 28	Suicidal tendencies, Number of attempts, Seriousness of intent, Medical threat to life
5 Anxiety	5	4 - 30	Panic attacks, Number of attacks, Somatic anxiety, Psy- chic anxiety, Phobias

Table 33 (continued)

Item Content of the SADS Summary Scales

Scale Number and Name	Number of Items	Scale Score Range	SADS Items
6 Manic Syndrome	18	10 - 65	Elevated mood, Less need for sleep, Energetic, Increased activity, Grandiosity, Irritability, Hyperactivity, Accelerated speech, Speeded thinking, Intrusiveness, Public display, Financial indiscretion, Assumes responsibility, Antisocial, Sexual excesses, Drunk, Grossly bizarre, Poor judgment
7 Delusions and Hallucinations	20	20 - 69	Reference delusions, Control delusions, Delusions of mind read, Thought broadcasting, Persecutory delusions, Delusions of guilt, Grandiose delusions, Somatic delusions, Severity of delusions, Bizarreness of delusions, Multiple delusions, Auditory hallucinations, Voice commentary, Two or more voices converse, Non-affective verbal hallucinations, Visual hallucinations, Olfactory hallucinations, Somatic hallucinations, Grandiose or persecutory hallucinations, Severity of hallucinations
8 Formal Thought Disorder	6	6 - 33	Flight of ideas, Incoherence, Loosening of associations, Illogical thinking, Poverty of content, Neologisms

APPENDIX E

SADS Summary Scale Scores for Each Schizophrenic Patient

This appendix includes the SADS Summary Scale scores for each of the 15 schizophrenic patients who participated in the present experiment. Table 34 shows the score for each scale and subject, expressed in terms of Percent Maximum Possible Scores: i.e., the scale score divided by the range for that scale. Each score ranges from 0 (i.e., every item contributing to the designated scale was scored as absent) to 100 (i.e., every item contributing to the designated scale was given the maximum, or most extreme, score).

The full title of each scale is:

- Scale 1: Depressive Mood and Ideation
- Scale 2: Endogenous Features
- Scale 3: Depressive-Associative Features
- Scale 4: Suicidal Ideation and Behavior
- Scale 5: Anxiety
- Scale 6: Manic Syndrome
- Scale 7: Delusions and Hallucinations
- Scale 8: Formal Thought Disorder

Table 34

Percent Maximum Possible Scores on SADS Summary Scales for Each Schizophrenic Patient

Subject Number	Scale 1 Depressive Mood/Idea.	Scale 2 Endogenous Features	Scale 3 Associated Features	Scale 4 Suicidal Idea/Behav	Scale 5 Anxiety	Scale 6 Manic Syndrome	Scale 7 Delusions/ Hall.	Scale 8 Thought Disorder
29	11.5	5.9	4.1	0.0	0.0	14.5	44.9	7.4
30	15.4	11.8	9.5	0.0	7.7	9.1	26.5	33.3
31	15.4	7.8	13.5	51.9	0.0	0.0	49.0	0.0
34	11.5	2.0	12.2	48.2	0.0	9.1	38.8	11.1
35	34.6	15.7	20.3	63.0	7.7	0.0	51.0	3.7
36	0.0	9.8	6.8	0.0	0.0	16.4	46.9	33.3
40	34.6	23.5	17.6	0.0	19.2	0.0	49.0	0.0
41	3.8	5.9	4.1	0.0	3.8	0.0	32.7	0.0
44	7.7	15.7	10.8	0.0	15.4	16.4	51.0	0.0
46	38.5	25.5	14.9	0.0	23.1	0.0	49.0	0.0
47	0.0	0.0	0.0	0.0	0.0	7.3	59.2	40.8
50	0.0	0.0	1.4	0.0	0.0	7.3	30.6	0.0
55	53.8	21.6	27.0	51.9	42.3	0.0	42.9	0.0
59	23.1	17.6	18.9	0.0	15.4	0.0	40.8	0.0
63	34.6	17.6	12.2	0.0	0.0	12.7	40.8	0.0

APPENDIX F

Consent Form for Patients

This appendix includes the consent form that was signed by each schizophrenic patient before testing commenced. In accordance with the New York State Psychiatric Institute-Columbia University Department of Psychiatry Institutional Review Board requirements, a copy of the consent form was given to each patient.

I voluntarily agree to participate as a subject in the Crossmodal Reaction Time test procedure. I realize that the Crossmodal Reaction Time task will require about an hour, and that I will be expected to lift my finger from a key when either a light or a sound is presented. I understand that this task is part of a research project whose purpose is to increase scientific understanding of mental functioning. I have been informed that this research will be of no direct benefit to me.

All aspects of the procedure have been explained to me and I understand that the Crossmodal Reaction Time procedure is neither physically nor psychologically harmful or discomforting. I have also been assured that I may withdraw from the study at any time.

(FOR PATIENTS) I understand that not participating or discontinuing participation in the study will not result in any loss of benefits to which I am otherwise entitled. I have been given a copy of this consent form to keep.

I have been told that the results of this procedure will be confidential and will be seen only by specialists in this research. I have been assured that all data will be kept in files at the Department of Psychophysiology, that information concerning subject identification will be coded, and that reports of the findings will not identify any individual tested.

The New York State Psychiatric Institute-Columbia University Department of Psychiatry Institutional Review Board has approved recruitment of subjects for this study. Dr. Samuel Sutton (212 927-8827) is the Principal Investigator of the study and he can be contacted for further information about the research. The Psychiatric Institute Review Board (212 694-7310) can be contacted for information concerning my rights as a research subject.

Signature

Date

Witness to Signature

APPENDIX G

Consent Form for Normal Control Subjects

This appendix includes the consent form that was signed by each normal control subject before testing commenced. In accordance with the New York State Psychiatric Institute-Columbia University Department of Psychiatry Institutional Review Board requirements, a copy of the consent form was given to each subject.

I voluntarily agree to participate as a subject in the Crossmodal Reaction Time test procedure. I realize that the Crossmodal Reaction Time task will require about an hour, and that I will be expected to lift my finger from a key when either a light or a sound is presented. I understand that this task is part of a research project whose purpose is to increase scientific understanding of mental functioning. I have been informed that this research will be of no direct benefit to me.

All aspects of the procedure have been explained to me and I understand that the Crossmodal Reaction Time procedure is neither physically nor psychologically harmful or discomforting. I have also been assured that I may withdraw from the study at any time.

(FOR CONTROL SUBJECTS) I understand that I will receive five dollars per hour (\$5.00/hour) for my participation in this task. I have been given a copy of this consent form to keep.

I have been told that the results of this procedure will be confidential and will be seen only by specialists in this research. I have been assured that all data will be kept in files at the Department of Psychophysiology, that information concerning subject identification will be coded, and that reports of the findings will not identify any individual tested.

The New York State Psychiatric Institute-Columbia University Department of Psychiatry Institutional Review Board has approved recruitment of subjects for this study. Dr. Samuel Sutton (212 927-8827) is the Principal Investigator of the study and he can be contacted for further information about the research. The Psychiatric Institute Review Board (212 694-7310) can be contacted for information concerning my rights as a research subject.

Signature

Date

Witness to Signature

APPENDIX H

Geometric Mean Reaction Time Data for Each Subject

This appendix includes the geometric mean reaction time data for each of the 15 schizophrenic patients, and each of the 50 normal control subjects who participated in the present experiment. For all tables in this appendix, reaction times (milliseconds) are listed by Subject Number (abbreviated "No.").

The geometric means (based on 10 reaction time trials) for each intensity value (1 through 10) and modality shift condition (C = crossmodal; I = ipsimodal) are presented in Tables 35 through 38. Each patient's reactions to light stimuli are shown in Table 35, and to sound stimuli in Table 36. Each control subject's reactions to light stimuli are shown in Table 37, and to sound stimuli in Table 38. "Slow Normal" control subjects are designated by an asterisk following the subject number in Tables 37 and 38.

Means and standard deviations for each modality shift condition, averaged across intensity values, are presented in Table 39 for patients, and in Table 40 for control subjects. Each reaction time and standard deviation is based on 10 values. For example, a Crossmodal/Light entry represents the arithmetic mean of the 10 geometric means (one for each intensity) for that subject, and the standard deviation reflects the variability of these 10 measures.

Table 35

Geometric Mean Light Reaction Times of Each Patient
for Each Intensity Value and Modality Shift Condition

No.		Intensity Value									
		1	2	3	4	5	6	7	8	9	10
29	C:	486	439	419	442	447	421	413	455	439	428
	I:	379	387	398	380	350	332	361	329	327	384
30	C:	559	511	499	485	450	449	434	475	493	430
	I:	501	495	435	420	361	390	375	442	369	388
31	C:	571	489	456	515	432	477	460	439	524	510
	I:	504	441	479	455	449	449	441	455	378	430
34	C:	393	399	417	426	372	398	348	358	360	373
	I:	380	379	371	374	359	376	366	355	380	363
35	C:	700	650	640	664	638	630	564	564	599	626
	I:	532	461	445	460	474	384	386	414	382	395
36	C:	501	501	484	465	474	495	476	492	444	446
	I:	365	325	325	313	325	303	300	298	298	305
40	C:	577	591	548	581	518	494	484	542	519	534
	I:	433	393	358	372	356	337	349	328	328	315
41	C:	456	424	482	411	455	505	412	417	465	501
	I:	436	429	453	476	439	436	488	436	402	444
44	C:	481	486	489	515	456	467	478	459	509	510
	I:	446	375	483	484	459	403	482	433	378	430
46	C:	350	360	374	360	313	319	340	318	330	379
	I:	323	362	341	318	336	331	346	315	330	320
47	C:	631	516	586	518	563	452	548	581	564	460
	I:	371	380	350	425	369	379	377	377	345	353
50	C:	359	366	364	347	370	317	344	353	316	311
	I:	366	319	314	317	342	339	349	290	331	305

Table 35 (continued)

Geometric Mean Light Reaction Times of Each Patient
for Each Intensity Value and Modality Shift Condition

No.		Intensity Value									
		1	2	3	4	5	6	7	8	9	10
55	C:	504	529	443	463	480	487	440	423	438	507
	I:	440	405	371	424	368	325	334	331	315	420
59	C:	666	587	573	556	545	572	476	554	515	527
	I:	591	575	512	509	473	486	545	487	455	435
63	C:	483	493	454	471	474	481	427	473	410	446
	I:	386	351	306	318	321	315	332	321	331	308

Table 36

**Geometric Mean Sound Reaction Times of Each Patient
for Each Intensity Value and Modality Shift Condition**

No.		Intensity Value									
		1	2	3	4	5	6	7	8	9	10
29	C:	550	462	503	430	462	462	416	524	427	446
	I:	392	391	349	381	334	349	370	384	325	348
30	C:	416	398	336	341	352	389	366	321	331	339
	I:	372	342	331	327	322	353	306	310	291	295
31	C:	422	486	530	510	380	455	501	401	389	400
	I:	458	431	410	367	367	382	411	374	389	388
34	C:	472	531	545	440	508	446	420	428	549	426
	I:	463	431	379	352	377	363	353	328	326	354
35	C:	590	569	567	602	594	542	608	608	582	593
	I:	409	407	402	380	431	437	406	401	465	410
36	C:	413	403	373	421	411	389	378	401	407	354
	I:	266	275	257	278	282	256	256	285	254	259
40	C:	491	492	540	484	502	545	424	499	491	457
	I:	336	297	325	308	373	315	301	330	284	296
41	C:	458	582	513	479	519	502	463	453	451	446
	I:	529	526	442	497	504	433	369	408	344	427
44	C:	473	352	481	504	560	489	445	460	546	594
	I:	458	504	477	413	517	498	449	472	448	456
46	C:	334	339	349	328	327	320	354	339	304	340
	I:	314	308	288	299	280	306	275	280	285	304
47	C:	568	537	485	520	507	453	546	490	502	492
	I:	317	370	405	330	376	357	353	389	386	332
50	C:	351	362	350	406	313	342	279	344	329	322
	I:	315	274	318	298	260	334	335	283	299	246

Table 36 (continued)

Geometric Mean Sound Reaction Times of Each Patient
for Each Intensity Value and Modality Shift Condition

		Intensity Value									
No.		1	2	3	4	5	6	7	8	9	10
55	C:	499	545	504	477	452	459	488	473	410	448
	I:	444	379	375	365	361	399	371	392	359	369
59	C:	476	569	437	417	451	458	501	475	466	491
	I:	492	420	422	424	429	442	436	369	463	425
63	C:	463	484	393	431	421	500	427	425	469	372
	I:	337	307	331	296	342	364	317	309	317	295

Table 37

Geometric Mean Light Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition

No.		Intensity Value									
		1	2	3	4	5	6	7	8	9	10
01*	C:	386	364	352	359	344	353	375	350	342	352
	I:	377	356	361	357	332	321	329	343	318	326
02*	C:	474	485	436	473	441	439	422	427	464	414
	I:	512	492	424	442	377	384	366	448	357	394
03	C:	329	307	338	312	306	322	300	305	330	351
	I:	325	306	307	318	336	322	301	309	297	310
04	C:	377	356	361	352	349	336	320	347	321	361
	I:	326	326	347	322	323	324	343	328	321	295
05	C:	279	290	259	239	244	253	253	253	253	250
	I:	261	270	245	245	254	225	246	265	235	246
06	C:	335	315	289	302	323	295	297	300	271	282
	I:	326	340	292	301	305	293	288	298	262	279
07	C:	316	314	305	317	327	319	324	305	306	300
	I:	322	311	324	318	325	314	301	286	294	307
08	C:	273	250	281	252	266	239	253	247	231	234
	I:	270	253	259	264	241	245	248	240	239	249
09	C:	363	317	353	337	345	335	293	324	300	283
	I:	328	350	345	311	311	321	314	298	317	297
10	C:	336	319	293	308	302	309	296	314	308	299
	I:	318	297	330	316	289	328	285	310	282	281
11*	C:	388	383	360	354	343	346	300	328	305	322
	I:	419	351	356	316	314	361	325	343	314	318
12	C:	329	322	310	315	319	270	283	294	281	284
	I:	321	345	313	291	276	279	278	289	255	260

**Geometric Mean Light Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition**

No.		Intensity Value									
		1	2	3	4	5	6	7	8	9	10
13	C:	279	303	290	244	252	265	275	259	249	234
	I:	302	250	290	298	296	272	254	270	264	253
14*	C:	435	410	411	357	398	335	372	345	390	327
	I:	396	353	405	341	362	334	306	330	309	296
15	C:	311	329	294	320	328	283	314	304	314	289
	I:	304	286	299	273	280	282	255	266	259	283
16	C:	299	316	278	264	267	260	248	262	252	275
	I:	293	284	288	291	293	275	266	269	253	251
17	C:	370	322	323	344	329	310	298	281	311	285
	I:	316	323	315	303	313	288	288	267	273	273
18	C:	270	312	280	303	284	295	270	297	276	303
	I:	294	261	266	282	270	252	282	256	254	267
19	C:	271	298	306	269	273	280	283	255	288	274
	I:	296	301	253	297	265	263	274	260	278	249
20	C:	277	270	261	264	268	264	255	251	253	244
	I:	277	278	266	279	252	259	257	262	242	255
21	C:	374	345	346	343	310	332	304	309	314	333
	I:	365	340	338	332	350	334	321	300	308	319
22	C:	258	263	251	277	283	252	245	259	238	224
	I:	272	265	239	285	250	244	255	249	233	246
23	C:	283	281	272	270	270	275	286	262	269	276
	I:	280	277	298	257	260	264	262	251	257	276
24	C:	268	261	275	241	238	245	238	263	234	234
	I:	300	291	245	277	261	249	273	235	235	239

Table 37 (continued)

Geometric Mean Light Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition

		Intensity Value									
No.		1	2	3	4	5	6	7	8	9	10
25*	C:	388	366	331	357	319	297	293	389	298	310
	I:	595	592	563	538	544	529	494	531	451	537
26*	C:	351	342	316	332	320	334	318	311	294	313
	I:	566	536	554	487	509	482	479	469	448	479
27	C:	249	257	267	246	243	251	237	241	240	245
	I:	266	246	240	252	242	244	225	218	224	226
28	C:	274	282	261	271	253	270	267	250	261	257
	I:	268	273	268	251	258	258	266	248	227	247
32	C:	334	349	409	336	371	338	353	302	346	341
	I:	335	300	344	320	315	292	310	330	308	287
33	C:	388	359	364	332	288	319	272	312	309	296
	I:	322	287	305	298	293	268	265	256	281	289
37	C:	277	256	270	261	237	251	263	251	241	240
	I:	259	265	246	241	259	256	259	249	235	223
38*	C:	388	388	423	394	380	383	378	373	395	349
	I:	411	407	374	378	370	369	348	358	306	381
39*	C:	431	393	373	371	370	347	373	347	368	376
	I:	399	331	335	356	389	364	329	399	314	346
42	C:	287	295	269	287	262	261	239	264	266	269
	I:	279	265	271	247	236	253	251	290	243	245
43	C:	371	364	372	312	342	315	358	345	322	359
	I:	333	326	355	327	316	318	297	325	325	324
45*	C:	535	546	478	451	570	514	487	552	462	494
	I:	501	416	507	492	468	428	368	450	432	465

Table 37 (continued)

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Geometric Mean Light Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition

No.		Intensity Value									
		1	2	3	4	5	6	7	8	9	10
48	C:	362	354	340	367	332	327	338	344	329	346
	I:	354	324	360	307	317	301	323	317	316	287
49	C:	253	258	253	238	261	246	224	261	250	243
	I:	247	231	239	237	235	213	235	221	244	228
51	C:	356	338	352	348	333	311	326	320	318	323
	I:	338	321	319	330	305	308	318	322	315	305
52*	C:	361	357	362	390	363	309	334	342	308	341
	I:	347	334	345	327	340	332	356	335	319	306
53*	C:	463	487	434	438	414	416	399	413	395	431
	I:	425	399	409	416	390	399	400	376	403	355
54*	C:	407	428	380	418	379	364	404	412	379	364
	I:	367	361	352	352	372	386	354	354	374	335
56*	C:	436	431	472	392	464	452	416	481	445	427
	I:	454	455	441	470	419	406	394	393	456	459
57*	C:	426	407	415	416	394	385	389	390	394	374
	I:	406	371	403	365	371	362	380	371	364	338
58	C:	379	354	325	357	344	343	324	372	346	352
	I:	341	329	336	351	352	315	352	303	311	335
60	C:	354	374	348	360	346	362	329	325	349	355
	I:	310	294	318	299	297	312	301	329	279	302
61	C:	352	359	356	356	334	328	379	309	335	376
	I:	357	342	338	350	330	324	317	313	314	313
62*	C:	421	456	393	429	403	422	410	396	409	410
	I:	415	412	435	391	421	407	396	388	378	401

Geometric Mean Light Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition

No.		Intensity Value									
		1	2	3	4	5	6	7	8	9	10
64	C:	330	322	332	339	316	305	324	299	309	322
	I:	315	316	319	312	311	296	301	311	321	296
65	C:	243	256	269	231	230	237	225	246	222	239
	I:	244	252	256	235	253	233	232	230	239	225

Geometric Mean Sound Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition

		Intensity Value									
No.		1	2	3	4	5	6	7	8	9	10
01*	C:	405	409	357	348	374	339	367	382	347	384
	I:	339	342	337	358	354	353	322	358	323	327
02*	C:	356	380	306	321	339	336	359	297	334	331
	I:	352	322	238	319	315	365	298	309	277	287
03	C:	305	303	294	299	275	280	283	272	271	274
	I:	275	283	295	291	283	265	267	271	264	272
04*	C:	379	389	340	355	364	361	343	357	321	307
	I:	357	339	340	319	343	367	320	311	309	326
05	C:	286	251	244	246	264	231	259	233	262	254
	I:	246	229	252	243	230	244	237	229	244	248
06	C:	298	336	275	282	265	286	305	276	255	289
	I:	282	293	283	299	273	245	275	263	274	280
07	C:	284	290	276	283	271	276	281	293	276	288
	I:	273	278	284	271	270	284	262	292	278	263
08	C:	232	235	238	219	214	240	243	213	201	219
	I:	215	228	216	213	219	229	216	204	239	222
09	C:	311	280	292	284	277	277	301	268	270	277
	I:	272	275	302	324	290	275	253	260	234	251
10	C:	274	323	267	294	252	246	278	262	257	255
	I:	276	272	261	233	267	253	256	231	243	244
11*	C:	342	374	332	325	317	340	344	349	331	325
	I:	331	318	323	313	344	299	317	332	311	330
12	C:	257	266	252	250	257	259	266	237	250	254
	I:	268	264	246	244	247	255	261	272	211	250

Table 38 (continued)

Geometric Mean Sound Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition

No.		Intensity Value									
		1	2	3	4	5	6	7	8	9	10
13	C:	267	251	236	244	247	252	260	227	232	234
	I:	230	250	236	235	253	237	239	244	240	230
14	C:	321	408	336	357	319	290	346	379	352	367
	I:	275	290	263	277	283	262	266	276	270	304
15	C:	308	290	279	305	304	283	271	275	273	290
	I:	268	283	278	277	279	283	267	254	246	262
16	C:	214	217	197	211	195	231	207	222	196	227
	I:	223	237	233	227	200	198	219	206	233	220
17	C:	284	321	264	270	284	280	278	271	274	263
	I:	290	289	277	283	261	266	243	266	262	303
18	C:	266	262	251	251	246	240	247	240	229	224
	I:	235	264	265	241	243	235	241	238	235	242
19	C:	283	265	270	259	289	275	259	277	250	255
	I:	247	241	232	249	249	224	220	258	251	235
20	C:	274	262	254	259	265	242	254	218	258	239
	I:	269	240	251	262	237	268	227	250	245	234
21	C:	296	288	291	266	274	292	284	281	265	271
	I:	262	282	293	296	267	262	270	269	257	267
22	C:	224	238	241	230	268	221	213	224	206	219
	I:	224	220	201	238	221	211	193	211	221	229
23	C:	250	240	230	224	238	227	220	222	240	218
	I:	244	246	205	238	233	257	235	233	231	230
24	C:	256	259	236	248	251	233	217	207	221	226
	I:	246	257	231	240	239	227	193	220	241	226

Table 38 (continued)

Geometric Mean Sound Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition

		Intensity Value									
No.		1	2	3	4	5	6	7	8	9	10
25*	C:	396	335	323	276	281	308	343	276	289	300
	I:	587	575	577	533	489	563	519	512	526	511
26*	C:	376	350	343	353	306	324	303	305	280	331
	I:	527	575	524	505	459	508	489	491	479	495
27	C:	248	284	254	215	227	223	220	210	218	201
	I:	229	228	233	210	227	205	217	211	227	199
28	C:	271	315	269	254	257	275	306	232	289	261
	I:	216	236	223	225	226	216	211	221	221	222
32*	C:	404	379	347	359	336	358	351	302	308	318
	I:	336	344	314	317	310	296	319	313	312	265
33	C:	287	302	253	278	259	275	315	267	265	266
	I:	294	276	287	239	235	230	244	228	257	256
37	C:	256	253	237	240	213	230	240	227	228	244
	I:	238	231	243	226	226	223	209	219	219	224
38	C:	330	316	312	283	282	297	305	306	280	291
	I:	312	312	295	298	275	292	284	305	287	278
39*	C:	391	380	468	399	406	398	420	310	397	414
	I:	347	354	347	360	358	407	362	406	366	316
42	C:	239	255	267	279	246	242	233	263	261	232
	I:	222	244	222	236	226	241	225	218	221	229
43*	C:	433	416	359	331	359	334	435	341	337	342
	I:	367	355	347	366	355	349	357	337	328	347
45*	C:	527	448	489	512	484	472	446	510	431	434
	I:	503	495	470	440	462	367	446	456	407	386

Geometric Mean Sound Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition

No.		Intensity Value									
		1	2	3	4	5	6	7	8	9	10
48	C:	346	308	309	299	293	335	297	320	297	282
	I:	322	329	335	285	298	275	318	299	301	289
49	C:	271	235	244	226	266	230	241	227	247	227
	I:	219	199	211	198	212	219	197	197	233	197
51	C:	337	378	356	335	357	329	355	315	331	336
	I:	317	302	295	292	333	308	289	291	311	296
52	C:	347	322	303	293	275	288	280	288	282	249
	I:	318	289	281	273	298	296	252	268	252	272
53*	C:	414	478	416	390	387	371	427	429	370	382
	I:	380	431	391	378	343	411	398	335	363	348
54	C:	322	332	321	326	337	314	314	324	319	304
	I:	291	314	309	299	307	303	299	284	296	269
56*	C:	481	473	508	483	481	506	487	433	460	481
	I:	473	472	452	430	413	439	496	469	401	513
57*	C:	377	358	381	358	371	369	363	393	328	320
	I:	367	335	342	347	321	320	337	335	367	318
58*	C:	390	359	395	403	320	346	324	318	311	340
	I:	327	356	336	314	340	307	307	347	321	320
60	C:	310	277	294	272	250	286	285	302	285	253
	I:	251	276	268	271	271	257	259	259	272	243
61	C:	347	343	317	309	327	312	320	303	285	315
	I:	323	327	307	291	295	297	310	294	291	282
62*	C:	443	490	474	456	467	496	435	444	480	440
	I:	436	437	391	427	403	415	401	458	418	414

Geometric Mean Sound Reaction Times of Each Normal Control Subject
for Each Intensity Value and Modality Shift Condition

		Intensity Value									
No.		1	2	3	4	5	6	7	8	9	10
64	C:	310	331	306	275	302	295	278	282	283	296
	I:	282	286	287	275	296	273	273	278	276	292
65	C:	207	208	210	189	209	197	202	191	192	194
	I:	185	198	207	185	182	180	176	181	183	194

Average Crossmodal and Ipsimodal Geometric Mean Reaction Times
of Each Patient to Light and Sound Stimuli

No.	Condition	Light		Sound	
		Mean	<u>SD</u>	Mean	<u>SD</u>
29	Crossmodal:	439	21.2	468	44.1
	Ipsimodal:	363	26.6	362	24.3
30	Crossmodal:	478	39.8	359	32.1
	Ipsimodal:	418	50.4	325	25.8
31	Crossmodal:	487	43.3	447	55.9
	Ipsimodal:	448	32.5	398	29.7
34	Crossmodal:	384	26.2	476	51.9
	Ipsimodal:	370	9.1	373	43.5
35	Crossmodal:	628	42.5	586	21.0
	Ipsimodal:	433	49.6	415	23.7
36	Crossmodal:	478	20.9	395	21.1
	Ipsimodal:	316	20.7	267	12.1
40	Crossmodal:	539	36.4	492	35.2
	Ipsimodal:	357	35.3	316	25.8
41	Crossmodal:	453	35.9	487	42.9
	Ipsimodal:	444	24.1	448	64.6
44	Crossmodal:	485	21.1	490	67.8
	Ipsimodal:	437	41.4	469	31.1
46	Crossmodal:	344	23.9	333	14.5
	Ipsimodal:	332	14.6	294	13.9
47	Crossmodal:	542	56.2	510	33.6
	Ipsimodal:	373	22.5	362	28.8
50	Crossmodal:	345	22.3	340	33.3
	Ipsimodal:	327	22.5	296	30.3

Table 39 (continued)

Average Crossmodal and Ipsimodal Geometric Mean Reaction Times
of Each Patient to Light and Sound Stimuli

No.	Condition	Light		Sound	
		Mean	<u>SD</u>	Mean	<u>SD</u>
55	Crossmodal:	471	35.4	476	36.9
	Ipsimodal:	373	46.3	381	25.5
59	Crossmodal:	557	50.2	474	41.5
	Ipsimodal:	507	50.6	432	31.6
63	Crossmodal:	461	26.6	438	40.2
	Ipsimodal:	329	23.9	322	21.9

Average Crossmodal and Ipsimodal Geometric Mean Reaction Times
of Each Normal Control Subject to Light and Sound Stimuli

No.	Condition	Light		Sound	
		Mean	<u>SD</u>	Mean	<u>SD</u>
01	Crossmodal:	358	13.8	371	24.1
	Ipsimodal:	342	19.9	341	14.1
02	Crossmodal:	448	24.7	336	24.8
	Ipsimodal:	420	53.4	317	27.1
03	Crossmodal:	320	16.7	286	13.4
	Ipsimodal:	313	12.0	277	10.9
04	Crossmodal	348	18.0	352	24.9
	Ipsimodal	326	13.9	333	19.4
05	Crossmodal:	257	15.6	253	16.1
	Ipsimodal:	249	13.7	240	8.4
06	Crossmodal:	301	19.1	287	22.7
	Ipsimodal:	298	22.2	277	15.1
07	Crossmodal:	313	9.0	282	7.1
	Ipsimodal:	310	13.2	276	9.6
08	Crossmodal:	253	16.5	225	14.1
	Ipsimodal:	251	10.6	220	9.8
09	Crossmodal:	325	26.6	284	13.7
	Ipsimodal:	319	17.6	274	26.5
10	Crossmodal:	308	12.6	271	23.2
	Ipsimodal:	304	19.1	254	15.7
11	Crossmodal:	343	29.8	338	16.1
	Ipsimodal:	342	32.8	322	12.9
12	Crossmodal:	301	20.7	255	8.5
	Ipsimodal:	291	28.1	252	17.3

Average Crossmodal and Ipsimodal Geometric Mean Reaction Times
of Each Normal Control Subject to Light and Sound Stimuli

No.	Condition	Light		Sound	
		Mean	<u>SD</u>	Mean	<u>SD</u>
13	Crossmodal:	265	21.7	245	12.9
	Ipsimodal:	275	20.1	239	7.7
14	Crossmodal:	378	36.4	348	33.5
	Ipsimodal:	343	36.7	277	13.0
15	Crossmodal:	309	15.8	288	13.9
	Ipsimodal:	279	15.9	270	12.6
16	Crossmodal:	272	21.1	212	13.0
	Ipsimodal:	276	16.0	220	14.0
17	Crossmodal:	317	26.9	279	16.6
	Ipsimodal:	296	20.7	274	17.7
18	Crossmodal:	289	15.0	246	13.1
	Ipsimodal:	268	13.8	244	11.3
19	Crossmodal:	280	14.8	268	12.8
	Ipsimodal:	274	18.9	241	12.5
20	Crossmodal:	261	10.0	252	15.9
	Ipsimodal:	263	12.3	248	14.5
21	Crossmodal:	331	22.0	281	11.2
	Ipsimodal:	331	19.4	272	13.3
22	Crossmodal:	255	17.4	228	17.5
	Ipsimodal:	254	15.9	217	13.2
23	Crossmodal:	274	7.3	231	10.6
	Ipsimodal:	268	14.3	235	13.5
24	Crossmodal:	250	15.4	235	17.7
	Ipsimodal:	260	23.7	232	17.5

Table 40 (continued)

Average Crossmodal and Ipsimodal Geometric Mean Reaction Times
of Each Normal Control Subject to Light and Sound Stimuli

No.	Condition	Light		Sound	
		Mean	<u>SD</u>	Mean	<u>SD</u>
25	Crossmodal:	325	34.6	313	37.9
	Ipsimodal:	537	42.8	539	33.7
26	Crossmodal:	323	16.7	327	29.1
	Ipsimodal:	501	39.0	505	31.8
27	Crossmodal:	248	9.0	230	24.9
	Ipsimodal:	238	14.9	219	11.8
28	Crossmodal:	265	10.0	273	24.9
	Ipsimodal:	256	13.7	222	6.8
32	Crossmodal:	348	27.7	346	31.7
	Ipsimodal:	314	18.5	313	21.5
33	Crossmodal:	324	36.8	277	19.5
	Ipsimodal:	286	19.9	255	23.9
37	Crossmodal:	255	13.3	237	12.9
	Ipsimodal:	249	13.1	226	9.8
38	Crossmodal:	385	18.7	300	16.5
	Ipsimodal:	370	29.8	294	13.1
39	Crossmodal:	375	23.9	398	39.2
	Ipsimodal:	356	30.7	362	27.1
42	Crossmodal:	270	16.2	252	15.7
	Ipsimodal:	258	17.5	228	9.0
43	Crossmodal:	346	22.7	369	42.3
	Ipsimodal:	325	14.5	351	12.1
45	Crossmodal:	509	40.7	475	34.6
	Ipsimodal:	453	43.2	443	44.6

Average Crossmodal and Ipsimodal Geometric Mean Reaction Times
of Each Normal Control Subject to Light and Sound Stimuli

No.	Condition	Light		Sound	
		Mean	<u>SD</u>	Mean	<u>SD</u>
48	Crossmodal:	344	13.6	309	19.8
	Ipsimodal:	321	22.2	305	20.0
49	Crossmodal:	249	11.5	241	16.1
	Ipsimodal:	233	10.3	208	12.6
51	Crossmodal:	332	15.5	343	18.3
	Ipsimodal:	318	10.6	303	14.0
52	Crossmodal:	347	25.4	293	26.8
	Ipsimodal:	334	14.4	280	20.9
53	Crossmodal:	429	28.6	406	33.4
	Ipsimodal:	397	20.0	378	31.0
54	Crossmodal:	394	23.0	321	9.4
	Ipsimodal:	361	14.5	297	13.2
56	Crossmodal:	442	27.0	479	21.5
	Ipsimodal:	435	29.0	456	35.6
57	Crossmodal:	399	16.3	362	22.7
	Ipsimodal:	373	19.8	339	17.6
58	Crossmodal:	350	17.6	351	34.6
	Ipsimodal:	332	17.8	328	16.8
60	Crossmodal:	350	14.7	281	19.3
	Ipsimodal:	304	13.9	263	10.6
61	Crossmodal:	348	21.8	318	18.2
	Ipsimodal:	330	16.2	302	14.7
62	Crossmodal:	415	18.4	462	22.0
	Ipsimodal:	404	17.1	420	20.0

Average Crossmodal and Ipsimodal Geometric Mean Reaction Times
of Each Normal Control Subject to Light and Sound Stimuli

No.	Condition	Light		Sound	
		Mean	<u>SD</u>	Mean	<u>SD</u>
64	Crossmodal:	320	12.6	296	17.3
	Ipsimodal:	310	9.1	282	8.2
65	Crossmodal:	240	14.5	200	8.2
	Ipsimodal:	240	10.8	187	9.6

APPENDIX I

Analyses of Variance for Effects of
Subject Group, Modality Shift, and Intensity
on Mean (Untransformed) Reaction Times

This appendix includes the results of three-factor mixed model analyses of variance on the mean (untransformed) reaction time data. For all analyses, subject group (patients, controls/slow normals) is a between factor, and modality shift (crossmodal, ipsimodal) and intensity (1 through 10) are repeated-measures factors.

Data from the 15 schizophrenic patients and the 50 normal control subjects who participated in the current research were included in the analyses shown in Table 41 (light) and Table 42 (sound). The results of corresponding analyses of the geometric mean (transformed) data are shown in Table 13 (page 108) and Table 14 (page 109) of the Results chapter.

Data from the 15 schizophrenic patients and the 15 control subjects in each slow normal subgroup (one for each modality) were included in the analyses shown in Table 43 (light) and Table 44 (sound). The results of corresponding analyses of the geometric mean (transformed) data are shown in Table 23 (page 135) and Table 24 (page 136) of the Results chapter.

Table 41

Analysis of Variance for Effects of
Group (Patients, Controls), Modality Shift, and Intensity
on Mean Reaction Times to Light Stimuli

Source	<u>SS</u>	<u>df</u>	<u>ms</u>	<u>F</u>	<u>p</u>
Total	10,109,273	1299	-----	-----	-----
Between Subjects	7,659,503	64	-----	-----	-----
Subject Groups (G)	2,959,101	1	2,959,101	39.66	<.001 ^a
Error _b	4,700,402	63	74,610	-----	-----
Within Subjects	2,449,770	1235	-----	-----	-----
Modality Shift (M)	483,599	1	483,599	37.71	<.001 ^b
Intensity (I)	210,256	9	23,362	47.10	<.001 ^c
G x M	363,634	1	363,634	28.36	<.001 ^d
G x I	14,196	9	1,577	3.18	<.001 ^e
M x I	13,427	9	1,492	3.18	<.001 ^f
G x M x I	9,272	9	1,030	2.19	<.05 ^g
Error ₁	807,867	63	12,823	-----	-----
Error ₂	281,243	567	496	-----	-----
Error ₃	266,276	567	470	-----	-----

^aepsilon adjustment not applicable. G is not a repeated-measures factor.

^bepsilon adjustment not applicable. df cannot be reduced further.

^cepsilon adjustment-- df = 1,63; p < .001

^depsilon adjustment not applicable. df cannot be reduced further.

^eepsilon adjustment-- df = 5,308; p < .01

^fepsilon adjustment-- df = 1,80; n.s.

^gepsilon adjustment-- df = 1,80; n.s.

Table 42
Analysis of Variance for Effects of
Group (Patients, Controls), Modality Shift, and Intensity
on Mean Reaction Times to Sound Stimuli

Source	<u>SS</u>	<u>df</u>	<u>ms</u>	<u>F</u>	<u>p</u>
Total	11,447,453	1299	-----	-----	-----
Between Subjects	8,987,297	64	-----	-----	-----
Subject Groups (G)	3,210,835	1	3,210,835	35.02	<.001 ^a
Error _b	5,776,462	63	91,690	-----	-----
Within Subjects	2,460,156	1235	-----	-----	-----
Modality Shift (M)	564,255	1	564,255	50.87	<.001 ^b
Intensity (I)	111,969	9	12,441	18.79	<.001 ^c
G x M	355,551	1	355,551	32.05	<.001 ^d
G x I	8,062	9	896	1.35	n.s.
M x I	6,802	9	756	1.30	n.s.
G x M x I	9,878	9	1,098	1.88	n.s.
Error ₁	698,835	63	11,093	-----	-----
Error ₂	375,415	567	662	-----	-----
Error ₃	329,389	567	581	-----	-----

^aepsilon adjustment not applicable. G is not a repeated-measures factor.

^bepsilon adjustment not applicable. df cannot be reduced further.

^cepsilon adjustment-- df = 1,63; p < .001

^depsilon adjustment not applicable. df cannot be reduced further.

Table 43

Analysis of Variance for Effects of
Group (Patients, Slow Normals), Modality Shift, and Intensity
on Mean Reaction Times to Light Stimuli

Source	<u>SS</u>	<u>df</u>	<u>ms</u>	<u>F</u>	<u>p</u>
Total	3,728,235	599	-----	-----	-----
Between Subjects	1,817,018	29	-----	-----	-----
Subject Groups (G)	232,657	1	232,657	4.10	n.s.
Error _b	1,584,361	28	56,584	-----	-----
Within Subjects	1,911,217	570	-----	-----	-----
Modality Shift (M)	228,111	1	228,111	8.44	<.01 ^a
Intensity (I)	188,930	9	20,992	26.61	<.001 ^b
G x M	324,012	1	324,012	11.98	<.002 ^c
G x I	4,147	9	461	.58	n.s.
M x I	9,196	9	1,022	1.36	n.s.
G x M x I	11,376	9	1,264	1.68	n.s.
Error ₁	757,076	28	27,038	-----	-----
Error ₂	198,808	252	789	-----	-----
Error ₃	189,561	252	752	-----	-----

^aepsilon adjustment not applicable. df cannot be reduced further.

^bepsilon adjustment— df = 1,28; p < .001

^cepsilon adjustment not applicable. df cannot be reduced further.

Table 44

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Analysis of Variance for Effects of
Group (Patients, Slow Normals), Modality Shift, and Intensity
on Mean Reaction Times to Sound Stimuli

Source	<u>SS</u>	<u>df</u>	<u>ms</u>	<u>F</u>	<u>p</u>
Total	3,919,006	599	-----	-----	-----
Between Subjects	2,026,387	29	-----	-----	-----
Subject Groups (G)	150,670	1	150,670	2.25	n.s.
Error _b	1,875,717	28	66,990	-----	-----
Within Subjects	1,892,619	570	-----	-----	-----
Modality Shift (M)	278,125	1	278,125	12.34	<.002 ^a
Intensity (I)	109,687	9	12,187	11.00	<.001 ^b
G x M	312,451	1	312,451	13.87	<.001 ^c
G x I	6,208	9	690	.62	n.s.
M x I	5,014	9	557	.53	n.s.
G x M x I	8,573	9	953	.91	n.s.
Error ₁	630,917	28	22,533	-----	-----
Error ₂	279,122	252	1,108	-----	-----
Error ₃	262,522	252	1,042	-----	-----

^aepsilon adjustment not applicable. df cannot be reduced further.

^bepsilon adjustment-- df = 1,28; p < .01

^cepsilon adjustment not applicable. df cannot be reduced further.

APPENDIX J

Crossmodal Retardation and Symptoms of Psychopathology

In this appendix, reference will be made to the eight SADS summary scales that were introduced in the Method chapter (see Symptomatology of the Patient Group, page 59). The item content of each SADS scale is shown in Appendix D. Average summary scale scores for the patient group are shown in Table 3 (page 60). Summary scale scores for each schizophrenic patient are shown in Appendix E.

Analyses with SADS Summary Scales

Delusions and Hallucinations. As indicated in the Method chapter (see Symptomatology of the Patient Group), of the eight SADS summary scales, the Delusions-Hallucinations scale yielded the largest mean score, and every patient was given a positive score on this scale. (The latter finding was predictable since a Definite RDC diagnosis of Schizophrenia requires that either delusions or hallucinations are present.) Pearson product-moment correlations between crossmodal retardation and patient scores on the Delusions-Hallucinations scale only approached significance: for light, $r(13) = .54$ ($p < .05$); for sound, $r(13) = .51$ ($p < .05$).

Initially, comparisons between the crossmodal retardation magnitudes of deluded vs. non-deluded patients, and of hallucinating vs. non-hallucinating patients were considered. However, inspection of the SADS data for each patient indicated that all patients exhibited

delusions, and all but three patients exhibited hallucinations.

Of the 20 SADS items that contribute to the Delusions-Hallucinations scale, 11 items indicate the presence or absence of delusions, and 9 items indicate the presence or absence of hallucinations. Ratings on these two groups of items were averaged separately, for each patient, and correlations between subscale averages and cross-modal retardation were computed. Results indicated that none of these correlations were significant: delusions subscales-- $r(13) = .14$ for light, $r(13) = .20$ for sound; hallucinations subscales-- $r(13) = .38$ for light, $r(13) = .29$ for sound.

Formal Thought Disorder. Six patients received positive scores on the Formal Thought Disorder SADS scale. However, inspection of the individual scores indicates that only three of these patients exhibited a marked formal thought disorder (e.g., neologisms, pervasive incoherence), whereas the remaining three patients exhibited only minimal signs (e.g., vague, digressive speech) that could be explained by heavy dosages of medication. Therefore, analyses were not conducted since only three of 15 patients clearly portrayed the symptom.

Depressive Symptomatology. Four SADS summary scales represent depressive symptomatology: Scale 1, Depressive Mood and Ideation; Scale 2, Endogenous Features of Depression; Scale 3, Depressive-Associative Features, and; Scale 4, Suicidal Ideation and Behavior. The number of patients who received positive scores on Scales 1 through 4 was 12, 13, 14, and 4, respectively. Pearson product-moment correlations between summary scale scores and crossmodal retardation were calculated for Scales 1, 2, and 3. None of these correlations were

significant: for light, correlations with Scales 1, 2, and 3 were .22, .21, and .17; for sound, correlations were .22, .05, and .14.

Mann-Whitney U tests were conducted to determine whether the crossmodal retardation magnitudes of those four patients who attempted suicide during their present episode of illness, were significantly different from the magnitudes of the 11 patients who did not attempt suicide. Neither of these comparisons was significant: for light, $U(4,11) = 21$; for sound, $U(4,11) = 28$.

In summary, there was no apparent relationship between crossmodal retardation and depressive symptomatology.

Anxiety Symptoms. Eight patients were given positive scores on the Anxiety SADS summary scale. Mann-Whitney U tests were conducted to determine whether crossmodal retardation differed for patients with, and without, anxiety symptoms. Neither of these comparisons was significant: for light, $U(7,8) = 27$; for sound, $U(7,8) = 16$.

Manic Symptoms. Eight patients were given positive scores on the Mania SADS summary scale. Mann-Whitney U tests were conducted to determine whether crossmodal retardation differed for patients with, and without, manic symptoms. Neither of these comparisons was significant: for light, $U(7,8) = 26$; for sound, $U(7,8) = 28$.

Summary and Conclusions

The current research failed to indicate a relationship between specific forms of psychotic symptomatology (more specifically, delusions, hallucinations, and formal thought disorder, as indexed by the SADS summary scales) and magnitude of crossmodal retardation. However, it is worth noting that the current research was not designed to assess

this relationship. Since all patients exhibited delusions, all but three patients exhibited hallucinations, and only three patients exhibited a marked formal thought disorder, several potentially fruitful dichotomous comparisons could not be made. Therefore, the relationship between psychotic symptomatology and crossmodal retardation is most correctly viewed as unexplored, and in need of systematic investigation.

The current research also failed to show any relationship between crossmodal retardation and depressive symptomatology. However, the number of depressive symptoms exhibited by each patient was selectively limited, since no patient exhibited a full affective syndrome: i.e., patients fulfilling the criteria for Schizoaffective Disorder were not recruited as subjects. Therefore, correlational analyses with depressive symptom scales only reflect the relationship between retardation and symptoms of depression in a carefully screened group of schizophrenic patients. It cannot be concluded that crossmodal retardation is unrelated to "depression," since the results of the current research might have been different if a depressed patient group were tested.

Finally, for the same reasons discussed above, although crossmodal retardation was not significantly different for patients with and without anxiety symptoms, and for patients with and without manic symptoms, these results do not imply that retardation is unrelated to patients with Anxiety and Manic Disorders.

APPENDIX K

Calculation of Coefficient Alpha

Coefficient alpha is a reliability estimate of the internal consistency of a test. The procedure for calculating coefficient alpha is shown in Table 45 (adapted from Cronbach, 1970, pp 159-161). For the current research, $K = 10$ (the number of intensity values), $n = 50$ (the number of normal control subjects), and the entry shown in each cell of Table 45 is a geometric mean based on 10 reaction time trials.

Table 45

Calculation of Coefficient Alpha
for a K-Item Test Given to n Subjects

Subject Number	Item Number							Sum
	<u>1</u>	<u>2</u>	<u>3</u>	.	.	.	<u>K</u>	
1	$\bar{x}_{11}$	$\bar{x}_{12}$	$\bar{x}_{13}$	.	.	.	$\bar{x}_{1K}$	$\underline{X}_{1x}$
2	$\bar{x}_{21}$	$\bar{x}_{22}$	.	.	.	.	$\bar{x}_{2K}$	$\underline{X}_{2x}$
3	$\bar{x}_{31}$	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.
<u>n</u>	$\bar{x}_{n1}$	.	.	.	.	.	$\bar{x}_{nK}$	$\underline{X}_{nx}$
Variance	$\frac{s^2}{\underline{x1}}$	$\frac{s^2}{\underline{x2}}$	.	.	.	.	$\frac{s^2}{\underline{xK}}$	$\frac{S^2}{\underline{X}}$

$$\begin{aligned}
 \text{Coefficient Alpha} &= \left(\frac{\underline{K}}{\underline{K} - 1} \right) \left(1 - \frac{\text{Sum of } \frac{s^2}{\underline{x1}}}{\frac{S^2}{\underline{X}}} \right) \\
 &= \left(\frac{\underline{K}}{\underline{K} - 1} \right) \left(1 - \frac{\frac{s^2}{\underline{x1}} + \frac{s^2}{\underline{x2}} + \dots + \frac{s^2}{\underline{xK}}}{\text{Variance } (\underline{X}_{1x}, \dots, \underline{X}_{nx})} \right)
 \end{aligned}$$

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