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# Spatial selective attention in schizophrenic, affective disorder, and normal subjects

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**Abstract**

Schizophrenic, affective disorder, and normal subjects performed tasks involving exogenous (automatic) and endogenous (voluntary) attention. In the exogenous attention task, schizophrenic subjects demonstrated a greater benefit in response time than did normal subjects. In the endogenous attention task, however, schizophrenic subjects showed a smaller benefit in response time than did normal subjects. These results are consistent with a model of schizophrenia that predicts a deficit in voluntary (endogenous) control, and a disinhibition and therefore enhancement of the automatic (exogenous) processes of spatial selective attention. Affective disorder subjects did not demonstrate a greater benefit in response time than normal subjects in the exogenous attention task, but did show a smaller benefit in response time than normal subjects in the endogenous attention task. The somewhat similar pattern of behavior of schizophrenic and affective disorder subjects suggests that abnormal spatial selective attentional processes may not be specific to schizophrenia.

*Keywords:* Schizophrenia; Bipolar disorder; Automatic attention; Voluntary attention

## 1. Introduction

Although an impairment of attention has frequently been cited as a fundamental clinical symptom of schizophrenia (Kraepelin, 1919; Bleuler, 1911/1950; McGhie and Chapman, 1961) a clear characterization of the precise nature of this attentional dysfunction has not yet appeared. Subtle deficits in schizophrenia involving sensory, memory, and motor processes have often been attributed to an underlying attention dysfunction. Whatever the nature of the attentional impairment, however, it is more complex than the assertion that schizophrenic patients simply do not 'attend' to the task experimenters set before them. Cognitive psychologists have distinguished between an early reflexive, automatic component and a later voluntary, sustained component of selective attention. In this paper we examine these reflexive and voluntary components of spatial selective attention in schizophrenic patients.

### *1.1. Attention in schizophrenia–Kraepelin's view*

Kraepelin recognized that attention was not a single process, and he distinguished between

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*Auffassung* an automatic, almost reflexive apprehension of sensory material and *Aufmerksamkeit*, a voluntary, sustained, and directed alertness. Kraepelin, it appears, believed that schizophrenia involved a dysfunction of a voluntary or sustained attention (Aufmerksamkeit). Such a view is not inconsistent with recent formulations (cf. Knight, 1992).

### *1.2. Cognitive theories of attention*

Cognitive theorists, too, frequently distinguish between a reflexive and a voluntary form of selective attention. The distinction can be traced at least to William James' Principles of Psychology, where he distinguished between a 'passive, reflexive, non-voluntary, and effortless' and an 'active and voluntary' variety of attention (James, 1890/1983, p. 394). Contemporary theorists also distinguish two forms of attention, but they assign other labels to them: *exogenous* and *endogenous*, *preattentive* and *attentive*, *automatic* and *controlled*, *non-conscious* and *conscious*, *transient* and *sustained* (Kahneman and Treisman, 1984; Klein et al., 1992; Nakayama and Mackeben, 1989; Posner and Snyder, 1975; Shiffrin and Schneider, 1977; see also Johnston and Dark, 1986). In the present paper, we focus on spatial selective attention and use the terms *exogenous* and *endogenous* to refer to the reflexive and voluntary aspects of spatial selective attention, respectively.

Two of the most widely used experimental paradigms in spatial selective visual attention are tasks that orient attention with either a peripheral or central cue. These experimental paradigms were developed by Posner and his colleagues (Posner and Cohen, 1980; Posner et al., 1980) and the findings resulting from these and many other studies created the infrastructure upon which many cognitive theories of attention rely.

*Spatial selective attention (exogenous and endogenous mechanisms)*: Visuospatial attention is frequently inferred from the time taken to recognize a target at a particular location. It is well established that a person's ability to detect or discriminate a target at a location to which he or she is attending is enhanced compared with that at a non-attended to location. There are two common methods of directing attention to an upcoming target: one is to present a peripheral cue at the location of the upcoming target (Fig. 1A); another is to present a symbol (e.g., an arrow) that indicates the future location of the target (Fig. 1B).

A peripheral visual signal (i.e., a cue that is eccentric to the point of fixation) has a biphasic effect in both covert orienting (attentional movement, unaccompanied by eye movement) and overt orienting (saccadic eye movement to the signal). First, the appearance of the cue rapidly and automatically summons attention (from 50 to 150 ms), which facilitates detection at the location of the signal (Posner et al., 1982). In James' words, "We don't bestow it [attention], the object draws it from us" (James, 1890/1983, p. 425). Perhaps simultaneously, the cue also primes midbrain oculomotor centers to prepare a saccadic eye movement toward it (Posner and Cohen, 1980). These two facilitating effects are then followed by an inhibition (often referred to as 'inhibition of return') that slows detection at the cued location when attention is not maintained at this position and induces a bias against making a saccade toward the cued location (Posner and Cohen, 1984; Posner et al., 1985; Maylor, 1985; Maylor and Hockey, 1985; Tassinari et al., 1987).

In addition to examining these rapid reflexive or exogenous attentional effects that follow the appearance of a peripheral cue, many researchers have studied slower-acting voluntary or endogenous attentional effects that occur shortly after the initial reflexive, exogenous effects. In these investigations, a central symbolic cue indicates where the upcoming target is likely to appear. Two points need emphasis: the cue is symbolic (e.g., an arrow which does not actually appear in the position of the subsequent target), and it induces a *spatial expectation* about where the upcoming target is about to appear. As with an exogenous cue, an endogenous cue also facilitates detection or discrimination of the upcoming target when the target appears in the *expected* position. In comparison with the rapid attentional facilitation (from 50 to 150 ms) induced by an exogenous cue, however, endogenous facilitation occurs more slowly. Endogenous facilitation begins no sooner than about 200 ms after the cue onset, and is often less

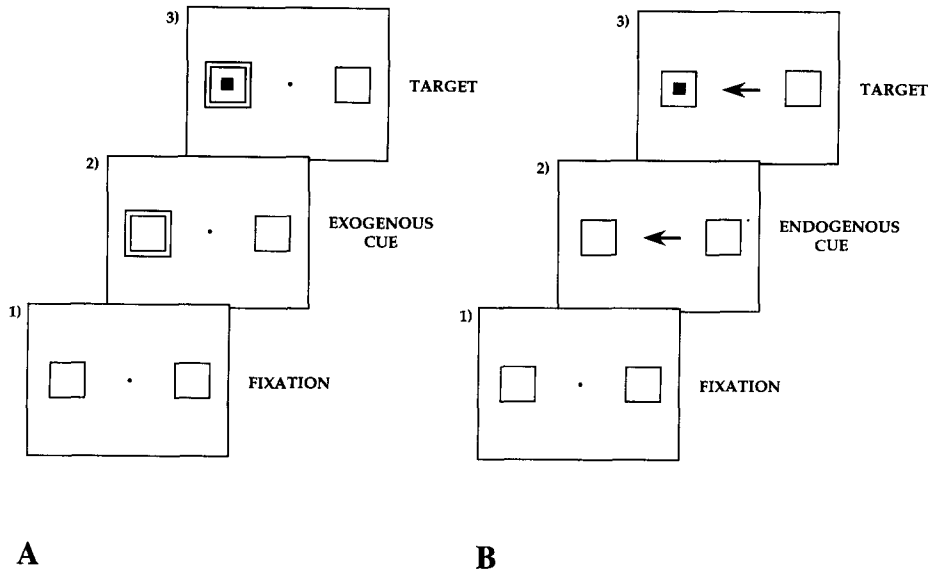


Fig. 1. Schematic diagrams of exogenous (A) and endogenous (B) covert orienting tasks. In exogenous tasks (A), a peripheral cue, such as the brightening of a box (indicated as a double box in step 2 of the figure), is in the actual position in which the upcoming target is about to appear. In endogenous tasks (B), a central symbol, such as an arrow (indicated in step 2 of the figure), signals the position where the upcoming target is about to appear.

robust than that seen following an exogenous cue (Klein et al., 1992; Jonides, 1981; Nakayama and Mackeben, 1989). In addition, when there is no saccade preparation, an endogenous cue does not produce inhibition of return (Rafal et al., 1989).

A peripheral cue automatically and rapidly draws attention to its spatial location, and, in most experiments, this location is the one in which the target will most likely appear. Thus, in addition to exogenous attentional effects, there is an endogenous attentional expectation that accrues for this position. In a spatial attention task, the terms exogenous and endogenous describe whether the spatial expectation is derived from an external cue (i.e., exogenous) that appears in the attended position or from a symbolic or internal cue (i.e., endogenous) that appears in some location separate from the intended focus of attention, but that indicates where such a location should be. As mentioned above, some work has also defined a temporal distinction between these two forms of attention: exogenous facilitation occurs rapidly and transiently whereas endogenous facilitation occurs more slowly and can be sustained. In a recent set of experiments examining the effect of the time interval between cue and target, i.e., stimulus onset asynchrony (SOA), on exogenous and endogenous facilitation, Nakayama and Mackeben (1989) repeatedly showed robust exogenous attentional effects peaking around a 100 ms SOA whereas they showed endogenous effects beginning only around a 200 ms SOA and becoming more robust around a 350 ms SOA. Hence, we chose intervals close to a 100 ms SOA and close to 500 ms in order to try to obtain optimal exogenous and endogenous facilitation effects. For the endogenous task, we chose a fairly long SOA (500 ms) to give patients a long enough time to allocate attention allocated, but not so long that they would not be able to sustain their attention perhaps due to an increased susceptibility to distraction.

In the experiments reported here we attempt to separate the exogenous and endogenous components of attention in two ways: first by manipulating whether the target appears in the position of the cue or in a separate position symbolically indicated by the cue; and second, by manipulating the SOA, that is, the time separating the cue and target (expecting that a short ISI will capture

exogenous attention alone and a longer ISI, endogenous attention alone). We examine the integrity of these two components of attention in schizophrenic patients.

### *1.3. Physiological model of spatial selective attention*

The striking differences in character and temporal course of the attentional facilitation following a peripheral or symbolic cue suggest that these attentional benefits are mediated by physiologically distinct processes. Specifically, we propose that differences reported between exogenous and endogenous attention reflect a distinction between a reflexive orienting system and a voluntary orienting system. However, within each system, we believe that both covert (attention) and overt (eye movement) orienting are handled by common or closely related physiological structures (Sereno, 1992). We suggest that the superior colliculus and related brainstem structures play a crucial role in the generation of exogenous attentional facilitation, whereas the prefrontal cortex may be intimately involved in the generation of endogenous attentional facilitation. There is some available evidence to support such a claim (Posner et al., 1982, 1985; Petersen et al., 1991).

It is known that in overt orienting (saccadic eye movements), prefrontal cortex plays a controlling and inhibiting role. In particular, evidence suggests that prefrontal cortex is crucial to both the *generation* of *voluntary* saccadic eye movements and the *inhibition* of *reflexive* saccadic eye movements (Guitton et al., 1982, Guitton et al., 1985; Schiller et al., 1987). Thus, in covert orienting, we expect the prefrontal cortex to play a similar role with respect to endogenous (voluntary) and exogenous (reflexive) attention. Accordingly, prefrontal cortex may be crucial both to the generation of endogenous attention and to the inhibition of exogenous attention.

### *1.4. Proposed hypothesis*

Schizophrenia has often been related to prefrontal cortical dysfunction (for review, see Levin, 1984a, b; Goldman-Rakic, 1987). According to our model, such a dysfunction would lead to two behavioral effects: (1) loss or impairment of proper function of endogenous orienting, and (2) disinhibition of exogenous orienting (Sereno, 1992). Such a pattern of performance in schizophrenic patients has been demonstrated for overt orienting (i.e., reflexive and voluntary saccadic eye movements; Sereno and Holzman, 1991, 1993, 1995). The present study examines exogenous and endogenous orienting in schizophrenic patients; we predict that schizophrenic patients, compared with normal individuals, will demonstrate a reduced endogenous attentional facilitation and a disinhibited (more robust) exogenous attentional facilitation.

## 2. Subjects and methods

### *2.1. Subjects*

Three subject populations were tested: (a) a schizophrenic group ($n=17$); (b) a psychiatric comparison group (predominantly patients with bipolar affective disorder) ($n=12$); and (c) a normal control group ($n=14$). Subjects were recruited for the study only if they met the following requirements: (1) less than 50 years of age; (2) no evidence of mental retardation (WAIS-R Verbal IQ $>85$); and (3) no evidence of organic brain pathology (as indicated by neurological exam noted in the medical charts). Patients were recruited if their condition had been diagnosed as either schizophrenia or major affective disorder by the hospital psychiatrist. The diagnoses were independently verified by information gathered from the Structured Clinical Interview for DSM-III-R (SCID), administered by an experienced interviewer. A comprehensive chart review and consultations with the patient's primary clinician provided supplementary information for the diagnostic decision. Sixteen patients (11 schizophrenic and 4 affective disorder) were recruited from Medfield State Mental Hospital in Medfield, Massachusetts, and 13 patients (6 schizophrenic and 7 affective disorder) were recruited from McLean Hospital in Belmont, Massachusetts, a private psychiatric hospital. All schizophrenic patients showed both positive and negative symp-

toms. Three of the patients in the schizophrenic group and 3 of the patients in the affective disorder group were outpatients. There were no distinguishable differences on any of the measures between the patients from the two hospitals. Neither were there distinguishable differences between the 6 outpatients and the 22 inpatients.

During independent diagnostic evaluation (SCID), four patients with a hospital diagnosis of schizophrenia met diagnostic criteria for schizoaffective disorder, considered to be related to the schizophrenia spectrum of disorders (DSM-III-R). The 4 schizoaffective patients were indistinguishable from the 12 other schizophrenics on all measures. The affective disorder group consisted of 8 patients with bipolar disorder and three patients with major depression (one with an additional diagnosis of panic disorder, one with an additional diagnosis of general anxiety disorder, and one with an additional diagnosis of borderline personality disorder). We excluded one patient whose admission diagnosis was bipolar affective disorder, but whose SCID diagnosis was borderline personality disorder. Normal subjects were screened for any serious (axis I) mental or neurological disorders in themselves and in their first degree relatives.

Table 1 presents the demographic characteristics of the subjects. There were no significant differences between the groups in age, years of education, IQ, gender or handedness. The patient groups did not differ with respect to age at onset or duration of illness.

Table 1
Summary of demographic variables for the three subject groups

| Variable (mean ± SD) | Subject group | | |
|---|---|---|---|
| | Schizophrenic ($n=17$) | Affective ($n=11$) | Normal ($n=14$) |
| Age (years) | 33.2 ± 5.4 | 30.5 ± 8.4 | 32.3 ± 5.2 |
| Years of education | 13.4 ± 2.3 | 13.1 ± 1.8 | 14.7 ± 1.8 |
| IQ | 108 ± 17.7 | 107 ± 10.7 | 114 ± 17.7 |
| Gender (% female) | 24% | 36% | 29% |
| Handedness (% left-handers) | 12% | 0% | 29% |
| Onset of illness (years) | 22.5 ± 5.3 | 20.8 ± 9.1 | |
| Duration of illness (years) | 10.2 ± 4.3 | 10.1 ± 7.5 | |

All 17 schizophrenic and 10 of the 11 affective disorder patients were taking psychotropic medication. Only one affective disorder patient was not on medication at the time of the testing. All of the schizophrenic patients and 82% of the affective disorder patients were receiving neuroleptic drugs. In addition, other therapeutic agents were administered to both groups of patients, including lithium, anti-convulsants and anxiolytics. The usual strategies for testing the effects of medication could not be employed in this study. Removing patients from medication was therapeutically contraindicated; testing unmedicated patients at their first hospitalization requires a recruiting strategy different from the one used in this study; and use of covariance provides only statistical estimates, that could be useful if only one compound had been used. Our position in this study is to leave unspecified the effects of medication on these results and to follow up with systematic studies that are specifically designed to test medication effects.

### 2.2. Methods

#### 2.2.1. Stimuli

In order to separate the exogenous effects of a peripheral cue from endogenous expectation effects, the present experiments employ a peripheral cue that acts as a symbolic cue indicating that the most likely appearance of the target will be at the position *opposite* to the peripheral cue. Validity is defined with respect to the induced expectation. Therefore, valid trials are trials in which the target appeared in the position opposite the cue, and invalid trials are trials in which the target appeared in the position of the cue. Eighty percent of the trials were valid trials; twenty percent of the trials were invalid trials. Hence, a target that appears, only occasionally, in the peripheral cue position (invalid trials) benefits from exogenous attentional effects alone, and a target that appears in the position opposite to the peripheral cue (valid trials) benefits from endogenous attentional effects alone. Further, exogenous effects arise within a short interval between onset of the cue and onset of the target and are transient,

whereas endogenous effects take longer to appear and are sustained. Hence, with a short interval between the cue onset and target onset (on the order of 100 ms), we expect to find exogenous attentional effects on invalid trials, whereas with a long interval between the cue onset and target onset (on the order of 500 ms), we expect to find endogenous attentional effects on valid trials.

*Exogenous task.* The visual display for the exogenous task was generated on a Macintosh II screen. The cue and target appeared in one of eight possible positions. All positions were arrayed along the circumference of an imaginary circle with radius 7.2° from a black fixation point (0.2° diameter). The cue was a black horizontal bar which briefly appeared upon a gray background. The target was a white horizontal line with a short vertical line either at the left or right end of the horizontal segment. Each trial consisted of the following sequence (see Fig. 2A and B): (1) a fixation point screen, which was experimenter-terminated by a click of the mouse, followed by a timed fixation point screen (510 ms); (2) a timed cue screen (30 ms); (3) a timed interstimulus interval (between the cue and target screens; 75 ms) that was identical to the fixation point screen; (4) a timed target screen (150 ms) in which the target and seven other lines appeared (the seven distractors appeared in other possible target positions which were to be ignored by the subjects); and, finally (5) a timed blank screen (210 ms).

*Endogenous task.* The endogenous task was identical to the exogenous task, except that the duration of the interstimulus interval (event No. 3 above) was 510 ms.

The subjects were instructed in both attention tasks to indicate, by a key press, whether the target had a vertical line on its left or right.

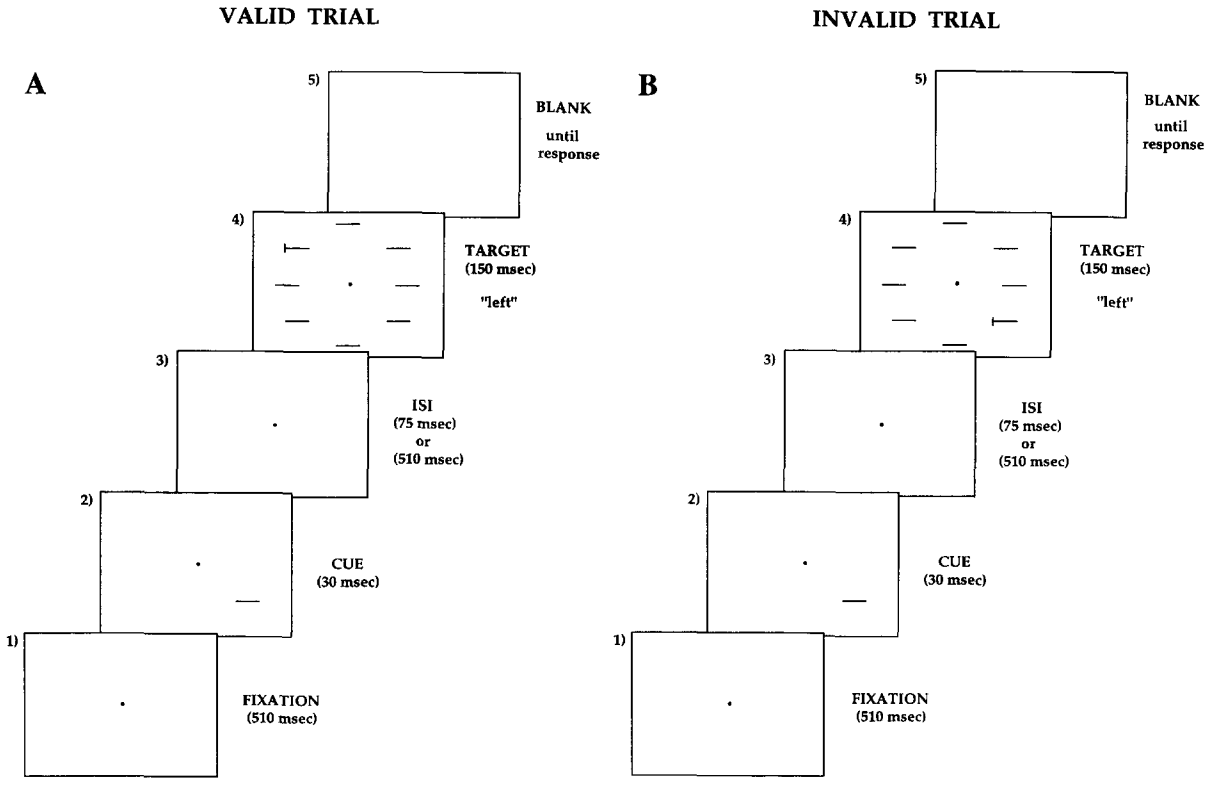


Fig. 2. Schematic diagrams of valid (A) and invalid (B) trials where the correct response was 'left'. The duration of the ISI (event 3 above) was 75 ms in the exogenous attention task and 510 ms in the endogenous attention task.

*2.2.2. Apparatus*

Subjects were seated 42 cm from a Macintosh II screen with their head on a chin support and their forehead against a restraint. An infrared light source was directed at the right eye. A video camera was also focused on the same eye. The output of the camera was sent to a Pupil/Corneal Reflection Tracking System (RTS), manufactured by ISCAN of Cambridge, Massachusetts. The RTS locates the subject's pupil and corneal reflection of infrared rays. Using the difference between location of the pupil and cornea, the ISCAN equipment calculates eye position taking into account any small head movements. The RTS was connected to a Macintosh II via a data acquisition board. As the experiment progressed, custom software on the computer presented stimuli on the screen, monitored eye movements, and recorded data on accuracy and timing.

The video camera was attached to a small monitor that displayed the right eye of the subject. Thus, the experimenter could see where the subject was looking in real time. The experiments were conducted in an isolated room under dim illumination.

*2.2.3. Procedure*

*Calibration.* At the beginning of each experiment, the subject was asked to look at five points on the screen – a center point and four points near the corners of the screen. An Auto-Calibrator, also developed by ISCAN, read the eye position at those points. Using the 5 points as references, it then calculated, on-line, the $x$- and $y$-axis screen coordinates corresponding to the current pupil position. These coordinates were updated 60 times per second. Calibration was successful if the eye position error radius was less than 1.5° from the fixation and reference points.

*Subject preparation.* After written informed consent was obtained from the subjects or their guardians, a first testing session was arranged, in which subjects were given a brief introduction to the equipment. Subjects were then properly positioned in front of the apparatus. After successful calibration, subjects were told that they were to decide whether the target had a short vertical line on its left or right. They were then shown sample trials. They were warned that the target would appear for only a very brief period of time and that it was important that they respond as fast as possible without sacrificing accuracy. All subjects responded with the index and middle fingers of their right hand. They were told to press the key labeled 'L' if the target had a line on the left and the key labeled 'R' if the target had a line on the right. The 'L' response key was always to the left of the 'R' response key. A trial was initiated when the experimenter clicked the mouse. Each subject was told that the cue indicated where the target was likely to appear and that paying attention to the cue could facilitate their ability to discriminate the target and thus make a faster response.

The instructions emphasized that the cue validly predicts the position of the target (i.e., in the position opposite to the cue) on the majority (80%) of the trials and they were reminded to respond as fast as possible without sacrificing accuracy. On the remaining trials (20%), the target appeared in the same position as the cue, contrary to the expectation induced by the instructions.

The subjects were instructed to focus on the fixation point at the beginning of each trial and continue fixating on it throughout the trial. They were told that if they moved their eyes from the fixation point at any time after the mouse click or before the target and distractors disappeared, the computer would automatically cancel the trial and re-present it later. The computer provided immediate feedback (a beep for an error). After the experimenter answered any questions about the procedure, each subject was allowed to complete the experiment.

There were four testing sessions. In session 1, subjects received 40 practice trials and then a set of 80 additional trials each for the exogenous and endogenous attention tasks. All 200 trials in session 1 were considered practice and were not included in the data analysis. Conditions were counterbalanced, such that half of the subjects within each group received the exogenous (short ISI) attention task as their first 40 practice trials, whereas the other half of the subjects received the endogenous (long ISI) attention task as the first 40 practice

trials. After the 40 practice trials, subjects were informed there would be two sets of experimental trials: a 'fast' (short ISI, exogenous) and a 'slow' (long ISI, endogenous) version. Each subject was then given the set of 80 experimental trials that matched the ISI of the practice trials they had just received and then the other set of 80 experimental trials.

The order of the experimental sessions in session 1 determined the order of attention experiments in sessions 2 and 3. That is, the subjects who, in session 1, had received the exogenous (short ISI) task first, were tested on the exogenous task in session 2, and the endogenous task in session 3. Subjects who had received the endogenous (long ISI) task first, however, were tested on the endogenous task in session 2, and the exogenous task in session 3. For both sessions 2 and 3, subjects received 40 practice trials and then 320 experimental trials. In session 4, each subject was briefly interviewed and then given the Vocabulary subtest of the Wechsler Adult Intelligence Scale. Table 2 shows the design of the experiments with the counterbalancing.

Table 2
Summary of the experimental procedures

| 50% of subjects | 50% of subjects |
|---|---|
| **Session 1** (all trials considered practice) | |
| 40 Practice trials; Short ISI | 40 Practice trials; Long ISI |
| 80 Trials; Short ISI | 80 Trials; Long ISI |
| 80 Trials; Long ISI | 80 Trials; Short ISI |
| **Session 2** | |
| 40 Practice trials; Short ISI | 40 Practice trials; Long ISI |
| 320 Test trials; Short ISI | 320 Test trials; Long ISI |
| **Session 3** | |
| 40 Practice trials; Long ISI | 40 Practice trials; Short ISI |
| 320 Test trials; Long ISI | 320 Test trials; Short ISI |
| **Session 4** | |
| WAIS-R vocabulary subtest | WAIS-R vocabulary subtest |

Dependent variables: RT for Valid and Invalid Trials, Percent Correct for Valid and Invalid Trials.
80% of the trials are Valid Trials.
Valid Trials: target appears in position opposite the cue.
Invalid Trials: target appears in position of the cue.

*Dependent measures*. There were two dependent variables: response time (RT) and accuracy. Eye movement deviations from the fixation point that exceeded 2.3° during the timed fixation point (i.e., the fixation point screen after the click of the mouse but before the appearance of the cue), or during the cue, the ISI, or the target screens automatically canceled a trial and the computer presented it again later. The computer also provided immediate feedback (a beep for an error), recorded the response key pressed, and the latency of the press for each trial.

## 3. Results

*Scoring*: There were 320 observations per subject for each of the attention tasks. RTs from incorrect responses were eliminated prior to analysis, removing 6.6%, 5.3% and 3.6% of the data for schizophrenic, bipolar, and normal subjects, respectively. All data, however, were included in the error analysis. The median RT and accuracy in each condition (ISI and attentional facilitation) were calculated for each subject. We separately analyzed RTs and accuracy. For both RT and accuracy, we first performed an overall ANOVA on the data including all three subject groups. We then computed attentional ratio scores for each subject and used these ratio scores to make specific comparisons between groups ($t$-tests).

*Ratio scores*: Ratio scores were used to compare the relative difference (percent difference) in performance of each subject on the attention tasks. These measures attempt to control for possible baseline differences in order to compare the amount of attentional facilitation across subjects and groups. In terms of RT, median RT on invalid trials was divided by median RT on valid trials (I/V) for each subject. This ratio score was used as a measure of attentional facilitation. In the exogenous task (short ISI), I/V ratios less than 1 reflect an exogenous facilitation (i.e., subjects were faster to respond on invalid trials than on valid trials). The smaller the ratio, the greater the effect of exogenous attention. In the endogenous task (long ISI), I/V ratios larger than 1 reflect an endogenous facilitation (i.e., subjects were faster

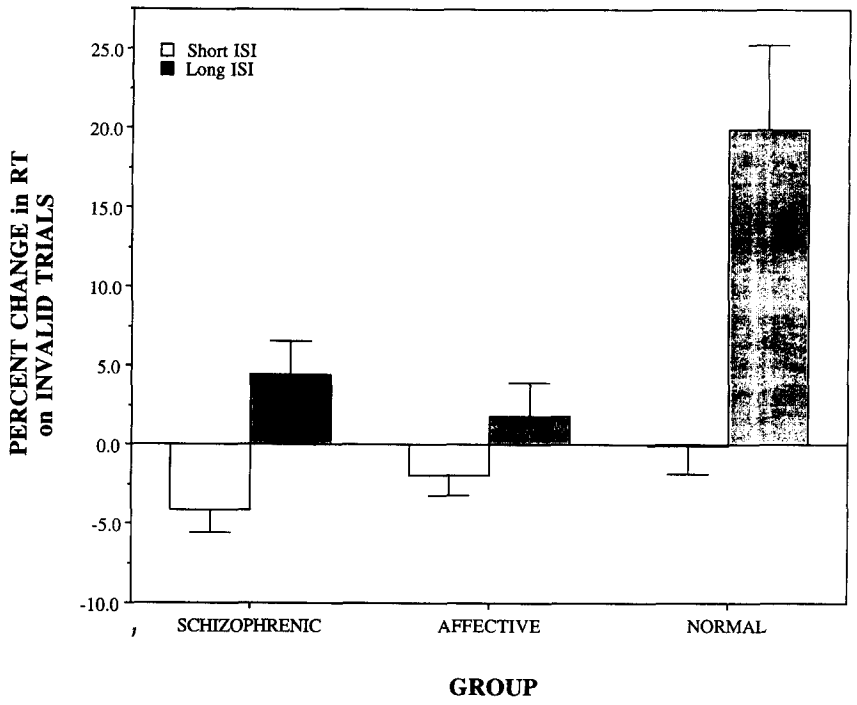


Fig. 3. Percent change in RT performance on invalid trials with respect to valid trials in the short and long ISI experiments across the different subject groups. That is, (ratio score $-1$)$*100\%$. Error bars are SEMs of the ratio scores for each task and group. At the short ISI, percent changes were expected to be negative for an exogenous attentional benefit.

to respond on valid trials than on invalid trials). The greater the ratio (larger than 1), the greater the effect of endogenous attention. Specific statistical comparisons between groups were performed on these RT and accuracy ratio scores. These ratio scores merely represent percent change (or a relative difference) in performance. If one subtracts 1.0 from the ratio and multiplies by 100, the results represent the percent change (or relative difference) in performance on invalid trials compared with valid trials. It is this percent difference in performance that is represented in Fig. 3.

*3.1.1. Response time*

Table 3 contains the results for all subject groups. It presents the group mean RT (of the individual subjects' medians) on valid and invalid trials as well as the attentional facilitation ratio scores for both short and long ISI experiments.

An analysis of variance was performed on the median RT data, which included groups (schizophrenic, bipolar and normal) as the between subject factor, and interstimulus interval (short and long) and attentional facilitation (yes and no) as within subject factors. For the short ISI experiment, invalid trials (i.e., cued but unexpected position) were considered trials with attentional facilitation, whereas for the long ISI experiment, valid trials (i.e., uncued but expected position) were considered trials with attentional facilitation. This analysis confirmed that there was a main attentional facilitation effect, $F(1,39)=22.67$, $p<0.0001$, such that subjects responded faster on trials with attentional facilitation. There were also significant main effects of group (normal subjects responded fastest and schizophrenic subjects slowest, $F(2,39)=8.24$, $p<0.001$) and ISI (faster response occurred with the short ISI compared with the long ISI, $F(1,39)=6.27$, $p<0.02$). Subjects showed a significantly larger endogenous attentional facilitation (faster on valid trials at the

Table 3
Group mean saccadic response time (RT) and percent correct (PC) for the three subject groups on both short and long ISI tasks

| Group | Schizophrenic | Affective | Normal |
|---|---|---|---|
| No. Ss | 17 | 11 | 14 |
| **Exogenous attention (mean ± SEM) (Short ISI)** | | | |
| RT (ms) | | | |
| Invalid-yes[a] | 491 ± 31 | 470 ± 51 | 354 ± 11 |
| Valid-no | 504 ± 29 | 474 ± 49 | 354 ± 10 |
| Ratio (I/V) | 0.9583 ± 0.0140 | 0.9805 ± 0.0126 | 0.9993 ± 0.0184 |
| PC (%) | | | |
| Invalid-yes | 93.0 ± 1.5 | 93.2 ± 2.1 | 95.2 ± 1.5 |
| Valid-no | 93.3 ± 1.5 | 94.8 ± 1.4 | 96.5 ± 1.0 |
| Ratio (I/V) | 0.9978 ± 0.0098 | 0.9818 ± 0.0108 | 0.9857 ± 0.0088 |
| **Endogenous attention (mean ± SEM) (Long ISI)** | | | |
| RT (ms) | | | |
| Invalid-no | 538 ± 33 | 498 ± 38 | 380 ± 9 |
| Valid-yes | 524 ± 32 | 497 ± 41 | 348 ± 13 |
| Ratio (I/V) | 1.0442 ± 0.0208 | 1.0179 ± 0.0215 | 1.1992 ± 0.0535 |
| PC (%) | | | |
| Invalid-no | 93.6 ± 1.5 | 94.7 ± 1.9 | 96.5 ± 1.4 |
| Valid-yes | 93.7 ± 1.6 | 96.0 ± 1.3 | 97.5 ± 0.8 |
| Ratio (I/V) | 1.0005 ± 0.0116 | 0.9863 ± 0.0122 | 0.9897 ± 0.0090 |

[a]Each condition is labelled both in terms of validity (valid/invalid) and attentional facilitation (yes/no).

long ISI) compared with a minimal exogenous attentional facilitation (faster on invalid trials at the short ISI), as evidenced by a significant interaction between ISI and attentional facilitation, $F(1,39)=4.35$, $p<0.044$. There was a tendency for affective disorder patients to show a small attentional facilitation (3 ms) across the tasks, whereas schizophrenic patients and normal subjects showed larger attentional facilitation effects, 13 and 16 ms, respectively, as evidenced by a marginally significant interaction between group and attentional facilitation, $F(1,39)=2.90$, $p<0.068$.

We had predicted that schizophrenic patients would show a reduced endogenous component of attention and an enhanced exogenous component of attention compared with normal subjects. We therefore expected that there would be a significant interaction between ISI, attentional facilitation, and group, such that, at the short ISI, *schizophrenic* subjects would show a greater attentional facilitation (i.e., attentional facilitation on invalid trials as a measure of exogenous attention) and, at the long ISI, *normal* subjects would show a greater attentional facilitation (i.e., attentional facilitation on valid trials as a measure of endogenous attention). There was, as predicted, a significant interaction between ISI, attentional facilitation, and group, $F(2,39)=5.44$, $p<0.0083$. This interaction, however, does not specifically test the proposed hypotheses, since the analysis includes all three of the subject groups. Thus, a series of planned, unpaired, one-tailed $t$-tests were performed to compare the attentional effects (using the ratio scores) between diagnostic groups at the two ISIs.

*Effects of exogenous attention (short ISI)*: As expected, at the short ISI, schizophrenic patients had a significantly larger attentional benefit on invalid trials (I/V ratio = 0.958) than did normal subjects (I/V ratio = 0.999), $t(29)=1.81$, $p<0.04$. Schizophrenic patients were 13 ms faster on invalid trials at the short ISI whereas normal subjects were no faster on invalid trials (354.3 ms) than valid trials (354.5 ms).

Affective disorder patients (I/V ratio = 0.981) and normal subjects (I/V ratio = 0.999) did not have significantly different I/V ratios at the short ISI for invalid trials, $t(23) = 0.80$, $p > 0.21$. Schizophrenic patients, however, did not differ from affective disorder patients at the short ISI, $t(26) = 1.10$, $p > 0.14$. These findings are illustrated in Fig. 3, which depicts mean RT ratio scores for the three groups in terms of percent difference.

*Effects of endogenous attention (long ISI)*: Also illustrated in Fig. 3, schizophrenic patients showed a smaller attentional benefit (I/V ratio = 1.044) at the long ISI for valid trials than did normal subjects (I/V ratio = 1.199), $t(29) = 2.89$, $p < 0.004$. Affective disorder patients had a significantly smaller attentional benefit (I/V ratio = 1.018) at the long ISI for valid trials than did normal subjects (I/V ratio = 1.199), $t(23) = 2.85$, $p < 0.005$. Schizophrenic patients did not differ from affective disorder patients at the long ISI, $t(26) = 0.84$, $p > 0.20$.

### 3.1.2. Accuracy

The mean percent accuracy was quite high across all subject groups (93.4%, 94.7% and 96.4% for schizophrenic, affective disorder, and normal subjects, respectively). Analyses were performed to see if there were any significant differences or speed-accuracy tradeoffs for the findings reported in the RT analyses. An analysis of variance was performed on the mean percent correct data, which included group (schizophrenic, affective disorder, and normal) as the between subject factor, and ISI (short and long) and attentional facilitation (yes and no) as within subject factors. For the short ISI experiment, invalid trials (i.e., cued but unexpected position) were considered trials with attentional facilitation, whereas for the long ISI experiment, valid trials (i.e., expected but uncued position) were considered trials with attentional facilitation. Although subjects were significantly faster (24 ms) to respond at the short ISI, the analysis showed that they were 0.9% less accurate (94.3% vs. 95.2% for short and long ISIs, respectively), as indicated by a significant main effect of ISI, $F(1,39) = 6.86$, $p < 0.013$. There was only one marginally significant finding in the accuracy analysis: an interaction between ISI and attentional facilitation. For both long and short ISIs, subjects showed a slight advantage in accuracy (0.7% and 1.0%, respectively) for valid trials, even though at the short ISI, invalid trials were exogenously facilitated, $F(1,39) = 3.76$, $p < 0.06$. With a short ISI between the cue and target, it is possible that although an exogenous cue leads to automatic orienting to its position thus reducing RT to a target at this position, it also produces a slight decrement in accuracy possibly due to interference with processing of the ensuing target (a possible forward masking effect). As was the case for the RT data analysis, a series of planned, unpaired, one-tailed $t$-tests for the effect of diagnostic groups was performed on the mean percent correct ratio scores. Table 3 presents group mean percent correct responses on valid and invalid trials as well as the ratio scores for both short and long ISI experiments. Three two-group comparisons were performed at each ISI.

*Effects of exogenous attention (short ISI)*: There were no differences between groups (schizophrenic vs. normal, affective disorder vs. normal, and schizophrenic vs. affective disorder) as reflected in accuracy ratio scores (see Table 3).

*Effects of endogenous attention (long ISI)*: There were no differences between groups (schizophrenic vs. normal, affective disorder vs. normal, and schizophrenic vs. affective disorder) as reflected in accuracy ratio scores (see Table 3).

### 3.1.3. Ratio vs. difference scores

As we stated above, ratio scores compare the relative change (percent change) in performance of each subject on the attention tasks. These measures, unlike difference scores, attempt to control for possible baseline differences in order to compare the amount of attentional facilitation across subjects and groups. In case this method affected the findings, we performed the specific comparisons ($t$-tests) in the main analyses above using difference scores instead of ratio scores. The use of difference scores, however, did not change any of the findings that we have reported. We use ratio scores in the specialized analysis below concerning lateralization effects.

### 3.2. Lateralization effects

Posner et al. (1988) were the first to examine schizophrenic patients with an exogenous attention task. They reported asymmetries in the hemispheric control of attention and suggested that the findings were similar to those of patients in previous studies who had unilateral left hemisphere lesions. The present experiments differed in several respects from the Posner et al. (1988) study. Nevertheless, most of the data we collected were lateralized and, hence, we examined whether or not any of the findings we reported interacted with visual field. A trial was considered to be a left visual field (LVF) trial when the target appeared in the LVF (regardless of what VF the cue appeared in), and a right visual field (RVF) trial when the target appeared in the RVF.

#### 3.2.1. Response time

An analysis of variance was performed on the median lateralized RT data, which included group (schizophrenic, bipolar and normal) as the between subject factor, and interstimulus interval (short and long), attentional facilitation (yes and no), and visual field (LVF, RVF) as within subject factors. The visual field location of the target proved to be an important factor. Subjects were faster to respond when the target appeared in the RVF, $F(1,39)=8.49$, $p<0.006$. This RVF advantage for the target, however, was true only for schizophrenic and normal subjects, as evidenced by a significant interaction between visual field and group, $F(2,39)=9.49$, $p<0.0005$. There were no other significant interactions involving visual field and group, suggesting no significant asymmetries in attention between groups. There was one higher order interaction with visual field location of the target, however, involving visual field of the target, ISI, and attentional facilitation, $F(1,39)=4.61$, $p<0.04$. It is important to note that visual field presentation of the target did not interact with ISI, attentional facilitation, and group, the finding of the main analysis, $F(2,39)=1.87$, $p>0.16$.

#### 3.2.2. Accuracy

An analysis of variance was performed on the mean percent correct data with factors identical to those for the RT analysis. The visual field location of the target was also an important factor in the accuracy analysis. Subjects were more accurate when the target appeared in the RVF, $F(1,39)=5.92$, $p<0.02$. There were no other significant or marginally significant interactions involving visual field.

## 4. Discussion

### 4.1. General findings

When compared with normal subjects and affective disorder patients, schizophrenic patients showed faster response times on invalid trials relative to their speed on valid trials on the short ISI trials. On the face of it, this relative augmentation of the schizophrenic patients' speed on invalid versus valid trials suggests an enhanced exogenous (reflexive) component of attention. With respect to the long ISI trials, both schizophrenic and affective disorder patients, compared with normal subjects, showed slower RTs on valid trials relative to their speed on invalid trials. This pattern suggests a reduced functioning of the endogenous component of attention for both groups of psychotic patients. Although we will enumerate below several cautions in this interpretation, the results suggest that the reflexive component of attention may be overactive in schizophrenia, whereas the voluntary component may be comparatively sluggish in both schizophrenic and affective disorder patients. Such an interpretation, if correct, would offer a modification of Kraepelin's observation that in schizophrenia the reflexive attentional component functions normally, but it is only the voluntary component that shows deficits.

The findings that schizophrenic patients exhibit a reduced benefit of endogenous attention compared with normal subjects agree qualitatively with Posner et al. (1988) who found only a small attentional facilitation at long ISI intervals for schizophrenic patients. Similarily, Nestor et al. (1992) reported in their first experiment a smaller relative benefit for valid cues for schizophrenic patients (6%) compared with normal control subjects (9%). This difference was qualitative and not

significant. Further, this calculation of relative benefit was computed after collapsing across short and long ISI conditions. We would expect the reduction in benefit to be occurring only at the long ISI. In the present study, there were no significant differences with respect to these variables in the error analyses, suggesting that there were no speed-accuracy tradeoffs.

### *4.2. Lateralization effects*

The findings we report (enhanced exogenous, reduced endogenous attention in schizophrenia) do not interact with visual field in our study. We do, nevertheless, find that schizophrenic and normal subjects, but not affective disorder subjects, were faster to respond when the target appeared in the RVF. In addition, in the long ISI experiment, subjects showed a reduced attentional benefit for valid targets in the LVF after a RVF cue. This is in agreement with other studies (Nestor et al., 1992; Posner et al., 1988; Coppola and Gold, 1990). In our study, however, this was true for both schizophrenic and normal subjects, as there was no significant interaction with group.

The present study reports no evidence of differential cuing effects with respect to visual field presentation of the target between schizophrenic and normal subjects. This is in agreement with several recent reports that either report a lack of performance asymmetry in schizophrenic patients (Strauss et al., 1991) or very limited asymmetries (Gold et al., 1992; Nestor et al., 1992). Several explanations have been proposed to account for the discrepancies between studies. Strauss et al. (1991) suggested that differences in clinical state may be responsible for the different findings.

### *4.3. Decreased exogenous attention in an 'anti-attention' paradigm*

Extinguishing the exogenous component of attention in an 'anti-attention' task similar to ours, in which instructions pitted the endogenous and exogenous components of attention against each other, was first demonstrated by Posner et al. (1982). We suggest that the Posner et al. (1982) study and that by Warner et al. (1990) help to explain why the 'automatic' exogenous component of attention was so small in our experiment. The Posner et al. (1982) study used a peripheral cue that overlapped the target. Those authors reported that whether or not the cue indicated the expected position of the subsequent target, there was an 'initial advantage for the cued side.' They do not report which differences are significant, but from their Fig. 2, it appears that the facilitation is present at both 50 and 100 ms SOAs. By 200 ms SOA, however, there is an advantage for the opposite or expected position (i.e., the uncued position). The crossover in their study occurs somewhere between 100 and 200 ms SOA, and thus they report that exogenous facilitation already becomes 'inhibition' by 200 ms SOA in an anti-attention paradigm. Posner et al. (1982) do not suggest that this reversal requires any practice.

Warner et al. (1990) also report that in an anti-attention paradigm, subjects, even at short ISIs, showed an ability to suppress an otherwise reflexive exogenous component of attention in the cued position and attended instead to the expected position. Warner et al. (1990) report 2 experiments. The results of their Experiment 1 differ from our findings: those authors report a significant advantage in the cued but invalid condition with SOA of 100 ms. However, the results of the low practice condition of their Experiment 2 agree with our findings: the authors report no difference between the cued (invalid) and uncued (valid) positions with an SOA of 100 ms. Warner et al. (1990, p. 250) suggest that 'the resolution of the apparent incongruity' in their experiments could be a practice effect because Experiment 1 had only 288 trials, whereas Experiment 2 had 2 sessions of 576 trials. However, the data for at least 1 of their 4 subjects in Experiment 2 came from data collected in Experiment 1. Hence, for this subject, performance in Experiment 2 was based on only 288 trials (see comment in Warner et al., 1990, p. 248). Furthermore, Warner et al. (1990) did not actually analyze the data in their Experiment 2 to show that such a practice effect did occur in the 3 remaining subjects. The practice effect they do demonstrate in their Experiment 2 is a change from benefit to cost for cued but invalid positions with a 50 ms SOA after from 3456 to 4608

additional trials. With a 100 ms SOA, they demonstrate only that there is a change from no difference to a cost. In our experiment, with a 100 SOA, we also see no difference for normal subjects.

In summary, we believe there is ample evidence that exogenous attention is reduced in an anti-attention task, even when subjects have not had any practice. Furthermore, we believe that Warner et al. (1990) have shown that large amounts of practice can further reduce exogenous facilitation (at SOAs as low as 50 ms). It remains unclear whether one should see exogenous facilitation with a 100 ms SOA in an anti-attention paradigm when the subject has not had extensive practice. As mentioned above, Posner et al. (1982) report a crossover from facilitation to inhibition somewhere between 100 and 200 ms SOA. Warner et al. (1990) show a significant exogenous facilitation in Experiment 1 with an SOA of 100 ms. However, in the lowest practice condition of Experiment 2 there was no difference between facilitation and inhibition at 100 ms SOA. In our study, all subjects first performed an anti-saccade task prior to entering the anti-attention task. It is possible that this anti-saccade task could have served as extra practice in the anti-attention paradigm.

We offer the explanation that the extinguishing of the exogenous component of attention is the result of an increase in the amount of inhibition that prefrontal cortex exerts on the superior colliculus because of the specific task demands that are imposed – in this case, requiring subjects to try to ignore the peripheral onset of the cue as a true indicator of spatial position of the target. We have suggested that schizophrenia may involve a dysfunction of prefrontal cortex and an impairment in its ability to inhibit the colliculus. This functional pathway is, nevertheless, still present and schizophrenic patients, we expect, are still able to make some limited changes in the amount of inhibition.

Despite the dampening of this exogenous facilitation due to the nature of our task, we found a difference between the schizophrenic and normal subjects, with schizophrenic subjects demonstrating an enhanced or more resilient component of exogenous attention compared with normal subjects.

### *4.4. Masking effects*

We found a slight (0.9%) but significant reduction in accuracy in the short ISI experiment, suggesting that there was some interference between the cue and target in the present study. Because the invalid target in the short SOA experiment occurs in the same location at and a short duration after the onset of the cue, there is a possibility that some masking has occurred. First, it is possible that the target served as a backward mask of the peripheral cue in the short ISI experiment, thereby reducing its effectiveness. Saccuzzo et al. (1974) first demonstrated that schizophrenic subjects showed an increased vulnerability in a backward masking paradigm. It is unlikely in the present experiment, however, that backward masking with a 105 ms SOA would destroy the spatial location information of the cue. Nevertheless, even if schizophrenic subjects were more susceptible to backward masking of the cue by the target, we would argue that the cue would therefore be less effective for them. In the present study, however, schizophrenic subjects show a more robust or resilient component of exogenous attention than do normal subjects. Second, it is possible that the cue could act as a partial forward mask of the invalid target, making the target harder to discriminate and thereby increasing the RT of the invalid judgment. Unlike the situation in backward masking, however, schizophrenic subjects do not show excessive vulnerability to forward masking (Schuch and Lee, 1989). Forward masking of the target by the cue, then, could not easily explain the differential performance of schizophrenic and normal subjects at the short ISI. For these reasons, we do not think a masking effect explains the present findings.

### *4.5. General slowing effect*

Another alternative explanation of the present findings is that they demonstrate a general effect of slowing, a well-documented characteristic of schizophrenic patients (cf. Nuechterlein, 1977). That is, suppose the speed of shifting attention from the cued to the uncued side is a function of general speed. Perhaps schizophrenic subjects are

still attending to the cued side 105 ms after the cue onset, whereas normal subjects have already begun to shift their attention to the other side. By 540 ms the schizophrenic patients have still not yet started to attend to the opposite side, whereas normals may be showing a strong effect.

Several studies have now suggested that schizophrenic patients show an attentional facilitation equivalent to that of normal subjects in the position of a peripheral cue by 100 ms (e.g., Posner et al., 1988; Nestor et al., 1992). This suggests that within 100 ms schizophrenic patients are able to shift their attention to a peripheral cue. Further, Nestor et al. (1992) reported that schizophrenic patients were able to rapidly disengage attention from an invalid cue to a target in the opposite visual field unlike normal subjects, who incurred a cost. Given these findings, it seems unlikely that the present results reflect some sort of general slowing for schizophrenic patients such that they are unable to shift their attention to the opposite side of the cue within 540 ms.

### *4.6. Benefit or cost*

We have referred to the present findings as support for an enhanced component of exogenous attention. This enhanced component of exogenous attention, however, could be due to an enhanced benefit on invalid trials where the target appears in the position of the cue or due to a reduced cost on valid trials where the target appears in the position opposite the cue. Although Nestor et al. (1992) reported reduced cost in schizophrenic patients, Strauss et al. (1991) have not shown this effect. One difference between the experiments is that Nestor et al. (1992) collapsed their trials across SOA (100 and 800 ms), whereas Strauss et al. (1991) report only 100 SOA results.

In order to distinguish the cost-benefit effects, one needs some measure of a neutral condition. We chose not to include such a condition primarily because it reduces the percentage of trials in which the subject is given accurate information about exactly where the target is going to appear and we feared this might adversely affect how much effort they put into attending to the cue condition. Further, there are often several dimensions that can differ between a baseline and cue condition. For example, if we had included a no cue condition in the present study, would we have been controlling for the peripheral onset of a stimulus or the expectation that it induced about where the target was going to appear? Both of these variables may be crucial.

Our model does not distinguish between whether it is an attentional benefit or cost that results in an enhanced exogenous component of attention. One could easily imagine that either situation is possible to implement in a way that would be consistent with the proposed schematic model. A more detailed understanding of the connections and functional relations between the brain regions important in spatial selective attention (cf. Goldman-Rakic, 1987), as well as simple models mimicking such neural networks, might help us to sort out the different possible mechanisms and to characterize their outcomes.

Inhibition of return (IOR) is one well-described attentional cost that occurs in the position of a peripheral cue under conditions where attention is subsequently drawn away from this position. It is not likely to have influenced the short ISI experiment, since Posner et al. (1982) showed no evidence of IOR at a short ISI (100 ms). It is likely, however, that IOR increased the amount of cost in the long ISI experiment. It is possible, therefore, that if schizophrenic patients showed a deficit in the amount of IOR, this would also lead to the findings we reported in the long ISI experiment.

On the other hand, however, some evidence suggests that IOR is mediated by a midbrain mechanism involving the superior colliculus. Posner et al. (1985) demonstrated that neurologic patients with progressive supranuclear palsy involving peri-tectal degeneration had a deficit of inhibition of return in the same directions in which eye movements were most severely impaired. In addition, Rafal et al. (1989) showed a temporal hemifield dominance under monocular viewing conditions for inhibition of return; they suggested that it may be mediated by the retino-tectal pathway or midbrain pathways. If schizophrenia involves a disinhibition of the superior colliculus, it is likely that IOR is enhanced in schizophrenic patients. Hence, in the present experiments, it

would make the performance difference in the long ISI experiment between valid and invalid trials greater for schizophrenic patients. We have reported a smaller performance difference between these trials, which would argue that there was actually an even greater deficit in the endogenous component of attention in schizophrenic patients in order to compensate for an enhanced IOR. Of course, if there were no difference in IOR between schizophrenic and normal subjects, the present findings would also support the idea that schizophrenic patients show a reduced benefit of endogenous attention.

### *4.7. Affective disorder patients*

Our present results suggest that an abnormal pattern of attentional performance is not specific to schizophrenia, inasmuch as affective disorder patients demonstrate a similar pattern in the long ISI task. We therefore cannot rule out the possibility that performance on these tasks may be sensitive to state related factors, not only in the affective disorder group, but also in the schizophrenic group.

The non-specificity of this pattern of results is reminiscent of the history of smooth pursuit eye movement (SPEM) studies in schizophrenia, in which both schizophrenic and manic depressive patients show elevated rates of SPEM abnormalities. Examination of patients' first degree relatives, however, demonstrated that although SPEM abnormalities can occur in many conditions, only in schizophrenia do they represent trait-related abnormalities (Holzman et al., 1984). We are currently exploring visuospatial attention in first degree relatives of schizophrenic and affective disorder patients. We are also testing patients who are in a state of remission from their psychosis (outpatients) as another probe into the state related nature of these data.

## 5. Conclusions

The present results are consistent with the proposed hypothesis that schizophrenic patients show a disinhibition (resulting in enhanced performance) on a task of reflexive orienting (exogenous task) and a deficit on a task of voluntary orienting (endogenous task). We have reported a similar pattern of performance for reflexive and voluntary saccadic eye movements (Sereno and Holzman, 1991, 1993, 1995). In those experiments, schizophrenic patients demonstrated a greater decrease in saccadic response time than did normal controls in a reflexive (or express) saccade task (when the fixation point was turned off 150 ms before the target appeared). Furthermore, these schizophrenic subjects demonstrated a relation between smooth pursuit and saccadic eye movement performance, such that subjects with impaired smooth pursuit showed a larger decrease in saccadic response time in a gap saccade task than did schizophrenic patients with normal smooth pursuit. In addition, we and others (e.g., Fukushima et al., 1990) have reported that schizophrenic patients show greater difficulties in a voluntary (or antisaccade) saccade task. More specifically, schizophrenic patients demonstrated lower accuracy and greater delays in generating antisaccades than did normal controls. Again, there was a relation between smooth pursuit and saccadic performance, such that schizophrenic patients with impaired smooth pursuit tracking showed a significantly lower accuracy in the antisaccade task than did schizophrenic patients with normal pursuit.

We must qualify our conclusions by recognizing that in spite of its complexity, our design does not successfully tease apart the exogenous and endogenous components of attention. Understanding that the peripheral cue does not indicate the true position of the target may result in altered baseline brain activity. One could argue that this change in baseline brain activity increases the amount of tonic inhibition of the subcortical or reflexive system. Schizophrenic patients may have a dysfunction in the brain system that plays a role in the generation of this inhibitory control over the reflexive system, yet they may nevertheless show some ability to modulate it.

The present study does show that there are differences in attentional processes between schizophrenic and normal subjects, and suggests one model of how and why we see these differences. Although the model is consistent with the present

findings, more detailed examinations of these attentional processes are necessary in order to specify more completely the differences between normal and schizophrenic attentional processes.

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