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Effects of Psychotropic Medication on Selected Cognitive and Perceptual Measures

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The effects of antipsychotic drugs on selected cognitive tests were assessed in schizophrenic and in depressed patients. Two hospital experimental groups (schizophrenias and affective disorders), two hospital comparison groups (schizophrenias and affective disorders), and a normal control group were tested on two occasions. The experimental schizophrenic group (n = 34) and the experimental affective disorder group (n = 26) were removed from all medication for 3 weeks and were then tested; they were tested again 4 weeks after being placed on their prescribed therapeutic drugs. The control schizophrenic group (n = 13) and control affective disorder group (n = 6) were tested twice, 4 weeks apart, and medicated on both occasions. The normal, drug-free control group (n = 26) was tested twice, 4 weeks apart. Results showed that (a) during drug-free periods there were significant differences between drug-free patients and drug-treated patients, attributable most likely to generalized deficits; (b) medication does not affect performance on these tests in any manner; (c) presence or absence of clinical improvement does not account for performance on these measures; and (d) the test performance of patients already on drugs changes in ways that are very similar to those of patients who are first off drugs and then put on drugs.

In efforts to understand the psychological processes that accompany schizophrenic pathology, investigators have tried to map characteristic cognitive and perceptual dysfunctions—an undertaking that can claim equal importance with recent advances in tracing the biological aspects of major mental disorders (e.g., Kety, 1959; Snyder, Banerjee, Yamamuro, & Greenberg, 1974). Both approaches must eventually converge in a consistent schema that illuminates the nature of schizophrenia. As a result of these psychological investigations, data on attention (Kornetsky, 1972; Neale & Cromwell, 1970), perception (Holzman, 1969), memory (Koh, Marusarz, & Rosen, 1980), sensation and reception (Venables, 1964), and spatial orientation (Witkin, Lewis, Hertzman, & Machover, 1954) have accumulated, from which several theories of the schizophrenic psychological dysfunction have been adduced (cf. Broen & Storms, 1967; Cromwell, 1968; Mirsky & Kornetsky, 1964; Shakow, 1962, 1963; Silverman, 1964; Venables, 1964).

In spite of some consistent findings—for example, the schizophrenics' poor performance on the Continuous Performance test and on span of apprehension, and longer reaction time—many empirical investigations have yielded generally ambiguous or inconsistent results, and failures to replicate have not been uncommon. In studies of size-estimation performance, for example, investigators have reported chronic schizophrenics to be less constant than a control group (e.g., Cooper, 1960; Weckowicz, 1957). Others have reported a restitutive effect (e.g., Johannesen, Friedman, & Liccione, 1964), and some have found that early paranoid schizophrenics were no different from normal subjects, whereas others have reported overconstancy (e.g., Rausch, 1952).

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One reason for such inconsistent results could be methodological, as several critics have noted (e.g., Chapman & Chapman, 1973; Holzman, 1969). Thus, faulty subject and control group selection, diagnostic unclarities, contaminating variables of hospitalization and treatment, all could obscure basic psychological processes of psychosis. Another reason may lie in the strategy of these studies: Researchers have directed their efforts not to discerning the basic psychological processes involved in disorganization but have studied patients' responses to certain standard, available, and much-utilized laboratory tests without careful regard for the relevance of these tests for exploring selected dimensions of psychosis. The links between a schizophrenic patient's disorientation and over constancy or under constancy, for example, is generally left unexamined.

In an earlier publication, Holzman (1969) noted that tasks like size estimation or constancy determinations do not primarily assess the functioning perceptual act. That is, we do not see brightnesses, or slants, or retinal sizes; rather, we see objects that are mediated by brightness, slant, and size. "Performance differences of mediators of things may not necessarily result in defective perception of the *thing*" (Holzman, 1969, p. 160). Individual performance variations in these mediational processes partly reflect differences in the way people organize sense data; that is, they reflect enduring cognitive and perceptual organizing preferences of subjects, styles of reality contact that Klein and his associates (Gardner, Holzman, Klein, Linton, & Spence, 1959) called *cognitive control principles.* Inasmuch as cognitive controls are alleged to remain relatively stable over time for each person (Gardner & Long, 1960; Witkin et al., 1954), it follows that in employing these measures with schizophrenic subjects, many of the ambiguous results may reflect the more or less enduring characteristics of the people being tested rather than of the essential psychotic process or of a defective perception of the thing itself. In this view, psychotic disorganization does not make all of its victims alike in all respects.

Otteson and Holzman (1976) administered 10 tests that are usually employed in the assessment of cognitive controls (rod-and-frame test, RFT; Concealed Figures Test; size estimation; the Stroop Color–Word Interference Test; Schematizing Test; Spatial Orientation Memory Test, SOMT; and four subscales of the Wechsler Adult Intelligence Scale, WAIS) to schizophrenic patients, psychotic nonschizophrenic patients, and normal control subjects. They reported that the factor structure derived from the correlation matrix was clearly interpretable in terms of previously described cognitive controls derived from normal subjects (e.g., field articulation, scanning, and leveling–sharpening). That is, the same cognitive controls that characterize normal subjects are also characteristic of the clinical population. Psychotic subjects, however, were more inefficient in the SOMT, Stroop Color–Word Interference Test, size estimation constant error, and Concealed Figures Test. But both schizophrenic and nonschizophrenic psychotic patients were inferior to normal subjects on these tasks, with neither group showing specific performance dysfunctions. Otteson and Holzman suggested that general organismic impairments (generalized deficit, in Chapman & Chapman's, 1973, terms) and not a specific schizophrenic impairment could have accounted for these defective performances.

Because all of the psychotic subjects in the Otteson and Holzman (1976) study were medicated, those therapeutic drugs could have dampened any real differences between schizophrenic and other psychotic groups. Indeed, because antipsychotic drug treatment is so widely used, it may have obscured the special psychological dysfunctions in schizophrenic and other psychotic patients and thus have hidden the ways in which a schizophrenic disorder uniquely affects cognitive and perceptual behavior. Some investigators have been concerned that psychotropic medication affects learning and cognition, although to date there has not been a comprehensive study of this area. Hartlage (1965), for example, speculated that the use of antipsychotic compounds has dampening effects on learning.

The study of the effects of antipsychotic medication on cognitive and perceptual test performance commands a special status, both because of the widespread use of many of these tests by experimental psychopathologists and because any effects on test performance that

these therapeutic compounds may have can illuminate the psychological concomitants of schizophrenic pathology, as Kornetsky (1972) and Spohn, Lacoursiere, Thompson, and Coyne (1977) have already argued.

Despite the large volume of work devoted to perceptual and cognitive aspects of schizophrenia and depression, the effects of medication on tests of perception and cognition have been evaluated systematically in only a few previous studies (e.g., Kornetsky, Pettit, Wynne, & Evarts, 1959; Orzack, Kornetsky, & Freeman, 1967; Spohn et al., 1977; Wynne & Kornetsky, 1960). Perhaps this is due to the difficulties involved in withdrawing patients from medication. Furthermore, those patients who can be removed from drugs are those who the hospital staff feels are well enough to be drug free, and therefore they make up a special sample that may not be representative of the wider clinical population.

Kornetsky et al. (1959) found no significant effects of chronic administration of 400 mg of chlorpromazine (cpz) on (a) the Digit Symbol subscale of the WAIS, (b) pursuit rotor performance, and (c) the tachistoscopic recognition threshold, although acute dosages of 100 and 200 mg of cpz and secobarbital in both chronic and acute administrations did impair performance. Wynne and Kornetsky (1960) reported no effects of chronic cpz administration on visual reaction time, and Orzack et al. (1967) found that chronic administration of 400 mg of carphenazine, a piperazine phenothiazine, improved performance on the Continuous Performance Test, a test of vigilance, but had no measurable effect on the Digit Symbol test. In their systematic study of phenothiazine effects on psychological functioning, Spohn et al. (1977) compared the performance of 20 chronic schizophrenics who received chlorpromazine for 8 weeks with the performance of 20 chronic schizophrenics who received a placebo for 8 weeks. All of their subjects had been hospitalized for long periods. They found significant ameliorating drug effects on attentional functioning, information processing, and autonomic reactivity. Yet they found no such effects on cognitive test responses that call for abstract reasoning or on tasks that can elicit concrete and autistic verbalizations—such as the Gorham Proverbs, a test of conceptual breadth—and on ratings of cognitive dysfunction.

We report here a study of the effects of antipsychotic medication on performance of several cognitive and perceptual tests of patients with schizophrenic and affective disorders who have been hospitalized less than one year. This sample is clearly a less chronic one than the sample studied by Kornetsky et al. (1959); Orzack et al. (1967); and Spohn et al. (1977) and, as such, can reveal effects of medication on patients who are expected to improve clinically in relatively short time spans. The measures tap cognitive domains frequently assessed in psychotic patients. The principal purpose is to provide data about drug effects on these commonly used cognitive and perceptual tests to determine if the Otteson and Holzman (1976) data can be attributed to the normalizing effects of drugs and to explore the significance of the presence or the absence of any such effects for a theory of schizophrenia. There are two principal hypotheses, both stated in null form:

1. Cognitive control measures are not affected by medication.
2. Performance on selected cognitive and perceptual tests remains stable, regardless of clinical response to antipsychotic medication.

Method

Subjects

There were two hospitalized experimental groups and two hospitalized control or comparison groups. These patients were consecutive admissions to an active psychiatric treatment facility in Chicago. The two hospital experimental groups consisted of 34 schizophrenic and 26 depressed patients who had been removed from all medication and kept drug free for 3 weeks. They were first tested at the end of the 3-week washout period (T1) and then retested 4 weeks after having been placed on appropriate therapeutic drugs (T2). The hospitalized control groups consisted of 13 schizophrenic and 6 depressed patients who were medicated during both test occasions, 4 weeks apart. All of the hospitalized experimental schizophrenic patients received antipsychotic drugs (e.g., phenothiazines or butyrophenones), and all of the hospitalized experimental depressed patients received mood-active drugs (e.g., tricyclics) after the 3-week washout period.

There were 26 normal, drug-free control subjects tested on two occasions, 4 weeks apart. These subjects were at low risk for schizophrenia and for depression. The control subjects consisted of subjects, recruited through local

newspaper advertisements, who had never been hospitalized for a psychiatric illness and who were matched to the patients' means for socioeconomic status, ethnicity, and sex. The Minnesota Multiphasic Personality Inventory (MMPI) was used to assess low risk for schizophrenia and depression, and those subjects who had any MMPI subscore over 70 on the Hypochondriasis (Hs), Depression (D), Psychasthenia (Pt), Paranoia (Pa), and Schizophrenia (Sc) scales were eliminated from the normal control group. Exclusionary criteria for all of the subjects included organic brain disorder, substance abuse, and WAIS IQs below 70.

Diagnostic Criteria

A diagnosis for each patient's condition was assigned by the patient's attending physician in consultation with the entire ward staff. This diagnosis was obtained from a standardized diagnostic interview, the Schedule for Affective Disorders and Schizophrenia, Lifetime Version (SADS-L) conducted by trained staff physicians. Diagnoses conformed to the Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1975).

In the experimental schizophrenic group, 23 patients were suffering from acute psychotic episodes in a generally chronic disorder and 11 showed a typically chronic course; 18 patients had prominent paranoid symptoms and 16 had undifferentiated schizophrenic syndromes. Among the control schizophrenic patients, 7 were acutely psychotic and 6 were more typically chronic; 7 were paranoid and 6, undifferentiated.

Among the experimental depressed subjects, there were 21 unipolar and 5 bipolar patients. The depressed control group consisted of 6 unipolar patients. There were no significant differences among these four groups with respect to socioeconomic status (Hollingshead, 1957) and to mean number of hospitalizations. Table 1 presents demographic information about the subject groups concerning sex, age, Verbal IQ, socioeconomic status, and total number of hospitalizations.

Procedure

All subjects were tested twice. The 34 schizophrenic and 26 depressed experimental psychiatric inpatients began a drug washout period on the first day of hospitalization and were tested initially (T1) after they completed a 3-week drug-free period (Week 3). They were then placed on medication for at least 4 weeks (Week 7) before being retested (T2). The three comparison groups, 13 medicated schizophrenic inpatients, 6 medicated depressed patients, and 26 normal nonhospitalized patients, were also tested twice at 4-week intervals. The schizophrenic and depressed inpatients who composed the comparison subjects were medicated during both test occasions.

Eleven tests yielding 17 measures were administered to all of the subjects. To test each subject took about 3 hours and two to three sessions, depending on the subject's capacities and tolerance. The tests and their measures follow. (Asterisks indicate tests that were also used by Otteson and Holzman, 1976.)

1. *Block Design.** Score: standard WAIS score.
2. *Digit Symbol.** Score: standard WAIS score.
3. *Object Assembly.** Score: standard WAIS score.
4. *Picture Completion.** Score: standard WAIS score.
5. *RFT.** The apparatus used was a portable version of Witkin's original test (Oltman, 1968). In this test, the subject must determine the true vertical position of the rod and ignore the potentially misleading frame. After the instructions were given to the subject, he or she was given eight trials to adjust the rod to the true vertical position. Each trial was begun with both the rod and the frame tilted 28° in the same or in opposite directions. Score: mean error for the eight judgments.
6. *Embedded Figures Test (EFT).* This test explicitly requires that subjects disentangle one part of the field from its context. A simple figure is hidden in the pattern of a larger figure that has various patterns of color and line. The stimuli for this test were identical to those used by Witkin et al. (1954): 24 complex and 8 simple figures. Form A with complex Figures 1–12 were used at T1, and

Table 1
Demographic Characteristics of Subjects

Group	Sex		Age		Verbal IQ		Number of hospitalizations		Socioeconomic status	
	Male (*n*)	Female (*n*)	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Schizophrenic										
Experimental[a]	17	17	24.3	6.4	90	23	2.4	1.9	3.9	1.2
Control[b]	8	5	24.1	3.1	103	23	3.3	2.9	3.9	0.8
Depressed										
Experimental[a]	12	14	37.1	12.7	109	37	3.6	2.6	3.7	1.3
Control[b]	4	2	26.3	6.3	105	32	3.3	2.9	3.8	1.6
Normal control	13	13	28.8	8.2	116	21	0.0	0.0	3.7	1.5
Total	54	51	28.7	8.1	104	26	–	–	3.8	1.3

[a] Patients were off drugs at Time 1 and on drugs at Time 2. [b] Patients were on drugs at both testing times.

Form B with the complex Figures 13–24 were used at T2. Score: mean time in seconds taken to find simple figures in the complex ones.

7. *Stroop Color–Word Interference Test.** This test is in three parts. Subjects must read as fast as possible (a) a list of color names (e.g., red, green, yellow, and blue) printed in black ink, (b) the colors of asterisks printed in the four colors, and (c) a list of color names printed in disparate colors (e.g., the word *red* might be printed in yellow; the subject must ignore the word *red* and name the color *yellow*). A fast reading of the color names on the third part of the test is assumed to reflect a capacity to attend selectively to the relevant stimulus and simultaneously to inhibit actively the more readily accessible but overlearned response to the disparate names of colors. Scores: (1) a color–word interference score (difference between reading times on (a) and (b) above, and (2) the total number of errors in reading the color–word card.

8. *Size Estimation test.** Each subject was asked to adjust a circle of light to be equal in size to that of a hand-held disk. Four trials, two ascending and two descending were required. In the present study, two disks were administered to each subject: a light grey disk (10 g), and a heavy grey disk (65 g), both 40 mm in diameter. Scores: (1) the mean number of looks at the standard disk; (2) the mean time for each judgment; (3) the mean starting-point effect (SPE) computed as the difference between the ascending (A) and the descending (D) trials for each disk; and (4) the constant error, computed as the mean error of each of the four judgments.

9. *Schematizing test.** The subject is asked to judge 90 successively presented squares that increase gradually in size. Scores: (1) percentage ranking accuracy, which consists of the actual percentage of correct ranks made by the subject on the 90 squares. This score reflects the degree to which subjects make accurate discriminations between successively appearing squares; (2) absolute increment error, which represents extent to which subjects keep track of the increasing size of the squares; and (3) a lag score, which reflects the degree to which subjects lag behind the progressive increase in the sizes of the squares throughout the total test sequence.

10. *SOMT.** Each subject was shown a standard geometric figure for 5 s, after which the subject was shown another sheet containing four or five identical figures that were rotated in different directions. The subject was told to select the figure that is in the orientation identical to the standard. Score: the total number of correct identifications out of 20 items.

A fuller description of the above 10 tests is found in Otteson & Holzman (1976).

11. *Critical Flicker Fusion.* The subject was asked to judge which of two lights was flickering: (a) the right light, (b) the left light, or (c) neither. There were a total of eight steps varying from 21.3 Hz to 36.7 Hz, and there was a total of six trials for each step. Score: a threshold score computed from the paired comparison model (Woodworth, 1938).

Statistical Design

In this repeated measures study, the 11 tests produced 17 quantitative measures at each time period. We transformed the responses at the two time points into change scores and proceeded under general multivariate assumptions. The major hypotheses concern the interaction between diagnostic groups and the change between predrug and postdrug conditions. We also assessed the role of sex, Verbal IQ, and clinical response, variables that may mediate the relationship between the independent variables and the response measures, and for this purpose we used a multivariate analysis of covariance (MANCOVA). These computations were carried out using the multivariance computer program, MESA 99 (Finn, 1972).

The data were thus analyzed as follows. First, we tested between-group differences on the predrug occasion (T1) for schizophrenic, depressed subjects and for normal control subjects. The scores were subjected to a MANCOVA that was adjusted for sex and verbal intelligence. Second, to test the hypothesis that the dependent measures are unaffected by psychotropic drugs, a multivariate analysis of variance (MANOVA) was performed to assess differences based on change scores (T1–T2). Third, to determine whether clinical improvement affects changes in these test scores, a MANCOVA was conducted to test the null hypothesis of no difference between groups once the effect of clinical improvement on the scores was eliminated. A subsequent analysis was performed to evaluate the between-group differences in change scores for normal subjects and only those inpatients who showed clinical improvement on psychotropic medication. Fourth, a series of univariate ANOVAs compared the experimental schizophrenic patients who were drug free and then medicated with control schizophrenic patients who were medicated on both occasions.

Results

Table 2 contains the means and the standard deviations for all scores for all groups on both testing occasions, as well as the difference scores.

Effects of Sex and Verbal IQ

Sex and Verbal IQ were two covariates used in the present study, and their contribution was analyzed prior to adjusting for their effects. Both sex and Verbal IQ were significantly associated with the dependent variables.

The overall multivariate F ratio for sex was significant ($p < .0002$), with 7 univariate and 4 step-down tests out of 19 significant at least at the .05 level. Digit Symbol, Picture Completion, SOMT, RFT, Critical Flicker Fusion, and two of the three scores of the Schematizing test showed significant sex differences. Males performed better on the Digit Symbol, RFT, and absolute increment error of the Schematizing test, whereas females performed better on Picture Completion, SOMT, Critical Flicker Fusion, and percentage accuracy of the Schematizing test. These significant effects were considered to be specific sample artifacts,

inasmuch as previous studies had established consistent sex differences only on the RFT and the EFT (Gardner, Jackson, & Messick, 1960; Gardner et al., 1959; Otteson & Holzman, 1976; Witkin et al., 1954, 1962).

The overall multivariate F ratio for Verbal IQ was significant ($p < .0005$), with 8 univariate and 5 step-down tests out of 17 significant at the .05 level, or better. Digit Symbol, Picture Completion, Object Assembly, Size Estimation constant error, RFT, Schematizing test absolute increment error, and two of the Stroop Color–Word Interference Test scores showed significant differences on IQ. On all eight of these dependent scores, high Verbal IQ was related to better performance. One would, of course, expect intelligence subscale scores to be related to Verbal IQ; for the other tests, no previous consistent relationship had been reported and we assume that these differences represent the effects of sampling.

Group Differences at T1

In an analysis that compared the schizophrenic experimental, the depressed experimental, and the normal subjects at T1, the overall multivariate F ratio was significant, $F(38, 126) = 2.29$, $p < .0004$. Six univariate tests of the 17 reached conventional levels of significance: Digit Symbol, $F = 7.67$, $p < .0009$; Picture Completion, $F = 4.89$, $p < .01$; SOMT, $F = 8.14$, $p < .0007$; Stroop errors, $F = 10.76$, $p < .0001$; Stroop Color–Word Interference Test, $F = 5.69$, $p < .005$; and Schematizing test percentage accuracy, $F = 5.78$, $p < .005$. The schizophrenic patients had significantly poorer scores than did the normal subjects on all six of these tests, whereas depressed patients did significantly worse than did normal subjects only on Digit Symbol and the Stroop Color–Word Interference Test. Otteson & Holzman (1976) reported significant effects of general psychosis on three of these six tests: the Concealed Figures Test (a version of the present EFT), the Schematizing test percentage accuracy, and the Digit Symbol test.

Effects of Drugs

The principal statistical analysis in this study concerns the differences among groups with respect to the change scores. A MANOVA was performed on change scores for the schizophrenic and depressed experimental groups from drug-free (T1) to medicated (T2) occasions, and for the control subjects tested on two occasions. The results show no significant group differences, $F(38, 126) = 1.23$, $p < .20$. All groups showed an improvement in most of the scores or no significant change from T1 to T2, with the patients showing the same degree of change as did the normal subjects.

Effects of Clinical Improvement

Not all patients show therapeutic clinical response to psychotropic drugs (Davis, Schaffer, Killian, Kinard, & Chan, 1980) and therefore it is possible to argue that drug effects on the dependent variables considered in this study were not demonstrable, because many of the patients in the experimental groups did not improve clinically on the medication. To examine this possibility, further analyses were conducted on those subjects who did show a drug response as measured by clinical improvement. This was accomplished in the following way.

Patients were rated for clinical improvement each week. The ratings were conducted by mental health personnel who were trained raters and who were kept unaware of the purposes of the study and of the medication status of the patients. All of the depressed patients were rated on the Hamilton Rating Scale (Hamilton, 1960), the Global Assessment Scale (Endicott, Spitzer, Fleiss, & Cohen, 1976), and the Itil-Katz Rating Scale (Katz & Itil, 1974). All schizophrenics were rated on the New Haven Schizophrenia Index (Astrachan et al., 1972), the Brief Psychiatric Rating Scale (Overall & Gorham, 1960), and the Global Assessment Scale (Endicott et al., 1976). The time periods chosen for ascertainment of clinical improvement ratings corresponded to the two times the patients were tested on the cognitive control measures.

Depressed patients were considered to have a drug response if any two of the following conditions held: (a) there was an improvement of 15 points or more on the Global Assessment Scale, (b) there was an improvement of 10 points or more on the Hamilton Rating Scale, or (c) there was an improvement of two points or more on the Itil-Katz Rating Scale.

Table 2
Means, Change Scores, and Standard Deviations for All Groups at Time 1 and Time 2

	Group									
	Schizophrenic				Depressed				Normal control	
	Experimental (n = 34)		Control (n = 13)		Experimental (n = 26)		Control (n = 6)		(n = 26)	
Test	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Picture Completion										
T1	7.5	2.0	9.0	2.1	9.8	3.1	10.5	3.0	10.0	2.3
T2	8.2	2.2	10.1	1.6	10.8	3.2	12.3	3.7	10.5	2.4
Δ	0.65	1.2	1.10	1.9	0.96	1.5	1.83	1.9	0.50	1.5
Block Design										
T1	8.5	2.8	9.5	3.1	10.3	3.2	12.6	2.4	10.4	3.6
T2	9.1	2.9	10.5	3.2	11.4	3.5	13.6	3.0	11.7	3.3
Δ	0.65	2.4	1.00	2.0	1.15	1.7	1.00	2.0	1.39	1.7
Object Assembly										
T1	7.4	2.9	8.7	4.0	9.4	3.2	11.0	2.7	10.1	3.3
T2	9.5	3.1	10.1	3.7	11.5	3.5	12.8	1.9	11.9	3.6
Δ	2.1	2.3	1.3	3.7	2.1	2.7	1.8	1.0	1.8	2.1
Digit Symbol										
T1	7.9	2.9	7.3	3.0	8.9	2.4	9.3	1.8	11.6	3.5
T2	8.2	2.4	8.4	2.6	10.3	2.7	10.0	1.7	12.6	3.3
Δ	.26	2.3	1.07	2.1	1.35	1.2	0.67	2.9	1.0	1.7
Spatial Orientation Memory										
T1	57.2	17.7	70.8	18.6	72.1	15.1	81.7	14.0	75.6	13.4
T2	60.9	18.8	78.8	14.2	76.5	18.2	80.8	17.1	81.5	12.1
Δ	3.7	21.0	8.0	16.9	4.4	15.2	−0.9	15.6	5.9	8.7
Rod and Frame										
T1	10.3	9.0	5.7	7.2	8.3	8.4	5.3	3.4	7.1	7.7
T2	9.1	9.6	4.6	7.0	7.6	8.2	4.0	3.5	5.9	6.6
Δ	−1.25	3.2	−1.00	2.1	−0.65	3.1	−1.13	2.8	−1.23	4.1
Embedded Figures										
T1	118.8	52.6	107.2	60.5	82.3	46.4	48.0	29.6	72.8	55.5
T2	96.6	64.0	98.1	66.6	56.2	49.2	45.7	31.1	52.8	53.5
Δ	−22.2	37.1	−9.1	23.9	−26.1	47.8	−2.3	11.6	−20.0	23.2
Critical Flicker Fusion										
T1	29.9	2.8	30.5	3.0	29.1	2.4	29.4	3.3	28.3	6.1
T2	28.7	5.1	30.0	2.7	29.4	2.3	30.8	2.4	29.6	1.5
Δ	−1.18	5.9	−0.41	2.3	0.29	1.8	1.38	4.1	1.32	6.4
Size Estimation										
Constant error										
T1	3.7	2.7	3.0	2.3	4.3	2.7	2.5	2.5	4.1	2.6
T2	3.2	2.4	3.8	2.7	2.5	2.4	2.9	3.2	3.2	2.7
Δ	−0.46	2.2	0.83	2.5	−1.75	2.3	0.38	3.1	−0.92	1.7
Time										
T1	19.3	12.6	20.8	16.7	16.0	5.5	18.3	5.5	18.3	12.6
T2	16.2	9.0	14.2	3.1	14.3	5.0	15.6	5.6	17.6	9.9
Δ	−3.05	7.5	−6.60	15.1	−1.69	3.8	0.33	3.4	−0.73	13.9
Number of looks										
T1	5.2	2.8	5.6	5.1	4.8	1.7	4.2	1.9	7.8	9.2
T2	5.1	4.5	4.4	1.6	4.6	1.8	4.0	0.9	6.6	6.0
Δ	−0.11	5.3	−1.20	5.1	−0.23	1.6	−0.17	2.1	−1.23	10.6
Starting point effect										
T1	0.50	2.2	1.46	2.5	0.69	2.5	0.58	2.0	0.79	1.7
T2	−0.04	2.4	−0.12	2.2	−0.65	2.2	−1.05	3.0	−0.02	1.8
Δ	−0.54	2.5	−1.33	3.2	−1.35	3.3	−1.63	2.9	−0.82	1.7

Table 2
(*continued*)

Test	Group									
	Schizophrenic				Depressed				Normal control	
	Experimental (n = 34)		Control (n = 13)		Experimental (n = 26)		Control (n = 6)		(n = 26)	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Stroop Color-Word Errors										
T1	11.5	9.4	7.8	6.5	3.1	4.2	3.3	2.3	2.8	4.4
T2	8.2	9.4	7.5	7.9	2.7	2.3	1.8	1.7	2.0	2.4
Δ	−3.3	10.3	−0.4	9.5	−0.4	3.6	−1.5	3.1	−0.8	3.6
Interference										
T1	134.6	51.8	155.7	80.9	118.3	45.4	100.5	74.8	86.2	24.7
T2	120.4	54.2	136.6	58.2	95.5	46.2	75.1	18.1	76.6	19.2
Δ	−14.2	51.4	−19.1	52.0	−22.8	26.5	−35.4	74.7	−9.6	26.5
Schematizing Lag										
T1	−2.3	3.7	−1.7	2.2	−0.8	3.6	−0.1	0.8	−1.2	2.6
T2	−1.9	2.5	−1.4	2.4	−0.8	2.5	−0.1	2.2	−1.4	2.5
Δ	−0.42	2.6	−0.33	2.1	0.0	2.9	−0.3	2.3	−0.20	2.1
Percentage accuracy										
T1	46.4	16.7	56.4	17.6	56.0	19.6	50.3	14.7	63.7	15.8
T2	46.5	16.7	56.8	19.5	55.1	18.9	51.3	21.6	60.8	19.6
Δ	0.10	15.1	.45	18.4	−0.94	14.1	1.00	6.3	−2.88	15.2
Increment error										
T1	45.1	25.9	27.6	16.2	46.9	45.8	39.5	26.5	30.6	16.9
T2	34.2	18.0	29.2	19.1	28.5	24.6	30.4	19.8	27.9	17.8
Δ	−10.9	25.0	1.6	23.5	−18.4	33.4	−9.1	14.8	−2.7	21.4

Note. T1 = Time 1; T2 = Time 2; Δ = change scores.

Schizophrenic patients were considered to show a drug response if any two of the following conditions held: (a) there was an improvement of 15 points or more on the Global Assessment Scale, (b) there was a change of 10 points or more on the New Haven Schizophrenia Index, or (c) there was a change of 10 points or more on the Brief Psychiatric Rating Scale.

Of the 26 depressed patients, 2 were eliminated because of a lack of information. Of the remaining 24 patients, 12 were rated as demonstrating clear clinical improvement after drug treatment. Out of the 34 schizophrenic patients, 18 were rated as drug responders.

To determine whether the initial level of severity was homogeneous for schizophrenic and depressed patients off medication, a t test was performed using the Global Assessment Scale at T1 as a dependent measure. The t test was not significant, $t(48) = .55$, $p = .59$, indicating no difference in the initial level of severity for the schizophrenic and the depressed patients. To test the differences in severity levels between the schizophrenic and the depressed groups after 4 weeks of pharmacotherapy, a t test was performed on the two groups' scores on the Global Assessment Scale at T2. This test was also nonsignificant, $t(48) = .27$, $p = .79$, indicating no differences between the depressed and schizophrenic groups with respect to the amount of response to drug treatment.

We then compared change scores in drug responders and nonresponders. A series of ANOVAs revealed no differences with respect to the dependent measures between drug responders and nonresponders, drug responders and normal control subjects, and schizophrenic drug responders and depressed drug

responders. We then ran two series of analyses of covariance on these same data. The first used initial level of test scores at T1 as the covariate. The second used the GAS scores as the covariate. These analyses produced the same results as the ANOVAs of change scores in drug responders and nonresponders. Thus, the failure to find significant change in the dependent cognitive and perceptual measures cannot be attributed to insufficient clinical improvement by patients.

Comparison of Experimental and Control Patient Groups

To test whether there were sample differences between the experimental and the control groups, the psychiatric experimental groups were compared with the psychiatric control groups in a series of univariate comparisons. Sample differences between schizophrenic experimental and control groups existed at T1 on only three measures: Picture Completion, SOMT, and Schematizing increment error. At T2, there were significant differences on two of these: Picture Completion and SOMT. In all of these instances the control group was superior to the experimental group. The presence of 3 marginally significant differences out of 17 suggests that these groups were actually not significantly different from each other at both T1 and T2. That sampling error most likely accounts for these differences is supported by the computation of the actual probabilities on these 3 of 17 statistical tests that are nonsignificant. This computation is based on the Bonferroni inequality procedure (Bock, 1975, pp. 422–423), which sets more stringent error rates in multiple comparisons such as these.

This conclusion is strengthened by an examination of clinical changes from T1 to T2 by the two schizophrenic groups. At T1, the Global Assessment Scale was 37.3 (SD = 13) for the schizophrenic experimental group and 46.6 (SD = 13) for the schizophrenic control group. This difference is statistically significant ($t(48) = 2.01$, $p = .05$) and indicates that the experimental group was clinically worse than the control group at T1. This difference in clinical state probably reflects the effects of removing a group of psychotic patients from medication and leaving another group of patients on medication. At T2, however, the Global Assessment Scale was 51.8 (SD = 16.8) for the schizophrenic experimental group and 56.3 (SD = 15.1) for the schizophrenic control group, a nonsignificant difference. Thus, both groups showed higher Global Assessment Scale scores at T2 than at T1, with the experimental group increasing 14.5 points (an increase of about 40%) and the control group increasing 9.7 points (an increase of about 21%). Because the experimental group was tested at T2 about 4 weeks after having been placed on medication, and the control group at T2 had been on medication for at least 7 weeks, the baseline differences at T1 and the changes from T1 to T2 were most parsimoniously interpreted as drug-related improvement in clinical state.

When, however, one compares the changes in cognitive control measures, it is apparent that the changes from T1 to T2 were no greater in the two schizophrenic groups than they were in the normal control group. Although the groups clinically improved—a shift probably attributable to drug treatment—the changes in the cognitive control variables seem to reflect no more than the psychometric effect of repeated testing. Furthermore, when the cognitive control scores were adjusted for Global Assessment Scale scores at T1, the results were essentially unchanged: Differences between the experimental and the control schizophrenic groups persist on Picture Completion (p = .05), SOMT (p = .05), and Schematizing test percentage accuracy (p = .02), indicating that clinical condition did not significantly account for these differences.

The depressed experimental and control groups differed at T1 on the Schematizing test increment error score and on the EFT. At T2, there were no significant differences between these two groups. These two groups also most likely differ from each other on a chance basis, and a computation of the actual probabilities based on the Bonferroni inequality procedure (Bock, 1975, pp. 422–423), which are nonsignificant, supports that supposition.

With respect to the change scores, for the two schizophrenic groups, neither multivariate nor any of the univariate comparisons were significant. Thus both the experimental and the control schizophrenic patients changed from T1 to T2 in parallel ways. With respect to the two depressed groups, the multivariate

comparison was not significant, and on the univariate comparisons only the EFT showed a significant change from T1 to T2, a finding that may warrant further investigation. There is, however, no support in the present data for a finding of a drug-related change in the scores of these tests in schizophrenic patients.

Discussion

This study demonstrated that (a) during drug-free periods there were significant group differences on six test scores, (b) medication does not affect performance on these measures in any specific manner, (c) clinical improvement does not account for performance on these measures, and (d) patients who are already on drugs change—with respect to their cognitive test scores—in ways similar to patients who are at first off drugs and who are then put on drugs.

With respect to the finding that at T1 there were group differences on six tests and that schizophrenics showed the worst performance on all six, we believe that the most reasonable explanation is in terms of general deficit performances of schizophrenic patients, as the Chapmans have argued (Chapman & Chapman, 1973). We compared the mean score of patients in the present sample with mean scores of patients in the study by Otteson and Holzman (1976) where identical tests could be compared. There were 9 tests (of the 11 in the present study) that were identical in both studies. The level of performance of the schizophrenic and of the depressed patients in both samples did not differ significantly on these nine tests. The performances of the two depressed groups in the present sample likewise did not differ significantly from those of the other psychotic group in the Otteson and Holzman (1976) sample.

One may assume that the Otteson and Holzman (1976) study reported test scores that represent the performance level of schizophrenic patients while on drugs. Keeping such patients drug-free for 3 weeks, as was done in the present study, produced no significant difference in level of test performance between the present sample and that of Otteson and Holzman (1976). Nevertheless, the drug-free schizophrenic group at T1 in the present sample did show conspicuous deficit performance on six of these tests: Digit Symbol, Picture Completion, SOMT, Stroop Color–Word Interference Test errors, Stroop Color–Word Interference Test score, and Schematizing test percentage accuracy. There was also clear performance improvement in this group at T2 when they had been on medication for 4 weeks. Yet the improvement did not exceed the general improvement that the other groups showed as a result of a second testing.

The six tests that showed some deficit performance are among those that seem to be vulnerable to impairments on the basis of interference with motivation, attention, and vigilance. Such a general but unsystematic lowering of performance at T1 in the schizophrenic experimental group suggests general performance dysfunction that is characteristic of schizophrenic subjects.

The general improvement of test scores in the schizophrenic group at T2, however, could not be attributed to specific ameliorating effects of the antipsychotic agents because the magnitude of change from T1 to T2 was the same for all groups, including the normal control group and the two patient control groups who were medicated at both testing times. The improvement in test scores seems to reflect the effects of repeated testing rather than therapeutic amelioration inasmuch as clinical change, as measured by the Global Assessment Scale, showed clear drug effects, and did not affect test score change. That is, the changes in clinical state from T1 to T2 were significantly greater for the schizophrenic experimental group (off drugs–on drugs) than for the schizophrenic control group (on drugs both times), a change that was not in evidence for the cognitive tests. The improvement in cognitive control test scores seen in the patient groups thus reflects the effects of repeated testing rather than therapeutic amelioration. A definitive test of this conclusion, however, would include groups of patients off drugs at both testing times, but such a design is highly improbable because it could represent the withholding of treatment from patients.

It may be argued that the 3-week drug-free period was not sufficient time for a release of drug control over psychotic behavior and therefore over the cognitive behavior that governs the tests under study here. This argument gains strength from the findings of Hogarty et

al. (1979) who report that relapse is cumulative over several months after withdrawal of antipsychotic drugs. Davis (1975) has confirmed that relapse is constant at about 10% to 15% of patients per month. Spohn (personal communication) also calls attention to his experience with drug-withdrawn schizophrenic patients who show low rates of relapse after only 3 weeks of drug withdrawal. On the other hand, it has been established that the 3-week period is more than sufficient for *pharmacologic* wash out and that the drug effects at the receptor sites are reduced to insignificance at 10 days. Studies of the half-life of neuroleptics show that one does not find evidence of neuroleptics or of their major metabolites in the blood after about 10 days (Davis, Ericksen, & Dekirmenjian, 1978).

A 3-week withdrawal is as long as is practicable in an active treatment hospital and is as long as or even longer than researchers typically use in this kind of a study. Yet it is also unmistakable that the *pharmacodynamic* effects do not wash out in 3 weeks (Davis, 1975; Davis et al., 1978). Side effects such as akathesia and dyskinesias remain, and relapse rates accumulate at a constant rate. Therefore, this study can claim generalizability only for the 3-week wash-out period. Why certain psychological effects should take longer to appear is quite unclear and unknown at this time, although several suggestions about structural or trophic changes in postsynaptic receptors and in dendritic branching have been proposed (Matthysse & Williams, 1981). Nor can one rule out the existence of a minor metabolite with a very long half-life. It is, however, unmistakable that it is not the *direct* pharmacologic effects of the antipsychotic drugs that are responsible for the test effects seen in this study. Should future studies demonstrate drug effects for the performances on these tests, such effects would probably reflect trophic and structural, but not direct, pharmacologic effects.

It may be fairly stated, moreover, that these antipsychotic drugs at the dosages used do not impair the performance in schizophrenic and in depressed patients on these cognitive and perceptual tests as, for example, Hartlage (1965) suggested that they might. The performance measures assessed here in schizophrenic and in depressed patients thus seem to be generally refractory to modification by the therapeutic pharmacologic agents used in this study.

Yet many controlled clinical studies have demonstrated the efficacy of neuroleptic and other antipsychotic drugs such as the antidepressants and lithium carbonate in reducing psychotic behavior (see review by Davis et al., 1980), and at least one study has demonstrated a noteworthy effect of a neuroleptic drug on quantity of thought disorder (Hurt, Holzman, & Davis, 1983). A reconciliation of these apparently disparate findings may elucidate some fundamental issues in the psychopathology and treatment of the schizophrenias.

Consider the following argument: In the psychological domain, schizophrenic dysfunction includes prominent thought disorder and impairment of attention and information processing as well as affective blunting and social withdrawal. Beyond these specific dysfunctions, general impairments in competence occur as a reflection of the psychosis and of its aftereffects. We suggest that neuroleptic agents do not affect the central or focal etiological features of schizophrenic pathology, although they have a demonstrated ameliorative effect on psychotic behavior and on the nonspecific deficits that accompany psychosis. These chemical compounds apparently derive their effects by making it possible for patients to attend to their environment, perhaps by dampening internal and external distractors and by aiding in reestablishing internal inhibitory controls over distraction. That is, they recruit possibilities for focussed and adaptive attention, but they do not repair the conditions that led to disinhibited and dysfunctional attentional processes.

Where a task includes efforts by the experimenter to inhibit such distractions by instructional sets and by diligent concern to ensure the patients' task involvement—as in the tests under scrutiny in this study—an increase, but not necessarily an optimum of attentional engagement, has been achieved. By the very nature of the examiner's efforts to bring out the best in the subjects, subject effectiveness is increased; the increased effectiveness, however, depends on the experimenter's continued exhortations and on other props that mobilize task involvement. One may assume that neuroleptics add nothing further to performance

adequacy than the experimenter's efforts have contributed; thus, at T2 only, the improvement due to the psychometric nature of the task becomes manifest.

Where, however, the task performance relies on the patients' display of their internal organization, with little effort by the experimenter to impose constraints or to maximize performance—as in general ward behavior, speech samples, autonomic nervous system responses—the neuroleptics will, for the most part, show effects, perhaps by affecting inhibition and by helping to dampen inappropriate behavior, loose thinking, and distracted attention. We would, therefore, agree with Orzack et al., (1967) and with Spohn et al. (1977) that phenothiazines (and neuroleptics in general) make possible greater attentional deployment; that is, they increase attention to environmental input by creating internal conditions that mute or reduce internal distractions.

It is noteworthy that in the studies of Kornetsky et al. (1959) and of Spohn et al. (1977) the tests of cognitive functioning showed no specific effects of the therapeutic drugs, and the present study confirms their findings. The patients in their samples were long-term chronic patients, whereas the patients in the present study were younger and less chronically hospitalized.

The present findings suggest that studies of medicated patients performing tests similar to the ones used in this study probably do not reflect drug artifacts and that results of such studies are not a manifestation of drug treatment. This study, moreover, suggests that, although the general antipsychotic effectiveness of neuroleptic medication is not questioned by the present data, these therapeutic agents are limited in the domain of their therapeutic effects. We propose that they do not appear to alter the perceptual and cognitive deficits of schizophrenia, although they appear to normalize other behaviors like thought disorder, autonomic reactivity, and ward behavior. It is, however, questionable whether these drugs affect the central pathogenic and pathotrophic (Holzman, 1977) factors involved in the schizophrenias.

One final interpretative conclusion: These tests do not show specific patterns of schizophrenic pathology beyond a general but unsystematic lowering of scores; the repeated testing shows that neuroleptic agents do not uniquely affect these test scores. Even with the general lowering of schizophrenic subjects' scores on these 11 tests, the results have not been so extreme or bizarre as to cast doubt on the general efficiency of their performance. The usefulness of these tests for studying schizophrenic patients seems to be in their promise to yield information about general cognitive and perceptual processes that might be involved in the schizophrenic disorders. From the present data, it seems, however, that these tests are not the best procedures we have for assessing such processes; they are only rough assessments of such functions as focussed attention, inhibition of distraction, or perceptual apprehension of things. Nor do they permit the partitioning of performance into phases of the input–processing network that could shed light on the aspects or phases of cognition and perception that may be dysfunctional. These tests thus seem to lack the required focussed relevance either for the experimental psychopathologist or for the practicing clinician.

We would therefore propose that psychological studies of schizophrenia be directed to processes and functions that may be clearly implicated in the disorder and that psychopathologists abandon the use of tests and laboratory procedures that are recommended only by their availability and familiarity.

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