Symptomatic and Neuropsychological Components of Defect States

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Abstract

The distinction between positive and negative symptoms has gained prominence in schizophrenia research, but the construct has not been unequivocally validated. The authors report preliminary findings of investigations in which symptomatic and neuropsychological assessments were conducted in a sample of 32 chronic schizophrenic inpatients. Three distinct clusters of symptoms were identified in correlative analyses. One cluster of symptoms (alogia, attentional impairment, positive formal thought disorder, and bizarre behavior) appeared to reflect primarily a disorganization of thought independent of current definitions of the positive/negative symptom construct. A second cluster of symptoms (affective flattening, avolition/apathy, and anhedonia) appeared to reflect predominantly blunting of affect and volition. A third cluster (delusions, hallucinations, and "breadth of psychosis") seemed to represent only the florid psychotic features. The first and (to a lesser extent) second clusters of symptoms were selectively associated with neuropsychological impairment. The pattern of neuropsychological deficits correlated with the first cluster of symptoms appeared to be consistent with a process characterized by failure in the development of a normal repertoire of cognitive abilities. It is suggested that the "defect state" may not be a monothetic construct, and that within the domain of "type II" schizophrenia, disturbances of thought may be distinguished from those of affect and motivation.

The clinical and social impact of chronic disabilities that may appear in the absence of florid psychosis has stimulated investigation of the schizophrenic "defect state." The florid or "positive" symptoms may have little prognostic significance (Jansson 1968; Strauss and Carpenter 1972) and "negative" symptoms, such as apathy and emotional blunting, have received renewed attention over recent years. Resemblance of these negative features to those found in other neurological disorders led to speculation that such symptoms could provide the focus for meaningful biological subtyping within the schizophrenia spectrum, and further our understanding of the pathophysiologies associated with schizophrenic disorders.

Simplifying assumptions may be necessary in the development of any conceptual framework; dichotomous formulations are often invoked to enable reliable hypothesis testing. The type I/type II distinction (Crow 1980) stands as a seminal contribution to the development of present research perspectives in schizophrenia. A body of literature has accumulated supporting the general utility of this construct, and several indices have been used to measure positive and/or negative symptoms (Johnstone et al. 1976; Abrams and Taylor 1978; Lewine, Fogg, and Meltzer 1983; Iager, Kirch, and Wyatt, in press). The problem of reliability in assessment of symptoms specifically relevant to the positive/negative distinction has been addressed by Andreasen and her colleagues. They defined domains of positive and negative symptoms, and developed a system for classifying patients into positive and negative groups (Andreasen 1981, 1982;

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Andreasen and Olsen 1982). The development of these standardized criteria has enabled objective evaluation of the validity of the positive/negative symptom construct.

Evaluation of the positive/negative symptom construct so far has involved:

- Assessment of consistency within the domain of positive and negative symptoms;
- Concurrent validity studies testing the effects of positive/negative symptom magnitude or group classification on other variables theoretically relevant to the more general type I/type II or positive/negative distinctions.

The original validation study, using the Scale for the Assessment of Negative Symptoms (SANS) (Andreasen 1981) and positive symptom variables derived from the Schedule for Affective Disorders and Schizophrenia (SADS) (Endicott and Spitzer 1978), demonstrated overall consistency within the set of negative symptoms and negative correlation between positive and negative symptoms (Andreasen and Olsen 1982). These results supported the suggestion that positive and negative symptoms might represent opposite extremes of a bipolar continuum.

However, other investigators using similar instrumentation have reported conflicting results. In one study, a nonsignificant correlation between positive and negative symptoms was found over all patients, with a significant positive correlation in a subset of patients (Rosen et al. 1984). Nonsignificant correlations between positive and negative symptom scales also were reported by Lewine, Fogg, and Meltzer (1983). These demonstrations that positive and negative symptoms are not necessarily inversely correlated, and in fact may covary directly, are inconsistent with the hypothesis of a bipolar positive/negative symptom dimension. Intercorrelations of individual symptoms were not presented in these reports, however, making it impossible to determine whether some alternative symptom clusters might have supported the positive/negative distinction.

There have been some attempts to assess the concurrent validity of the positive/negative symptom distinction. Interpretation of these studies necessarily depends on accepting the validity of the concurrent or criterion measures. Thus, positive and negative symptoms have been "tested" for relationships with other features theoretically relevant to an expanded type I/II construct, such as computed tomographic (CT) findings, neuropsychological test results, response to drugs, and premorbid social adjustment.

The validity of the positive/negative symptom distinction in this context was supported by several studies. In contrast to positive symptoms, negative symptoms were more often associated with ventricular enlargement (Johnstone et al. 1976; Andreasen and Olsen 1982; Andreasen et al. 1982); impaired performance on brief cognitive or mental state exams (Johnstone et al. 1978*b*; Andreasen and Olsen 1982); poor premorbid social adjustment (Prentky, Watt, and Fryer 1979; Andreasen and Olsen 1982); lack of response to neuroleptic medication (Johnstone et al. 1978*a*); and absence of exacerbation in response to amphetamine-like drugs (Kornetsky 1976; Angrist, Rotrosen, and Gershon 1980).

Some of these supportive findings were not very robust. Other studies have failed to support the validity of the positive/negative distinction. Two different CT-scan studies have shown no clear relationship between symptoms defined according to the positive/negative dichotomy and ventricular enlargement (Bishop et al. 1983; Nasrallah et al. 1983). Furthermore, in a recent study, patients selected for "Kraepelinian" features did not exhibit more negative symptoms than acutely exacerbated patients and, among patients who were studied in both stable and acutely exacerbated states, negative symptom scores were found to increase during exacerbation (Rosen et al. 1984).

These conflicting results are not easy to reconcile within a simple positive/negative symptom construct. Sample differences and variation in the definitions of both symptomatic and criterial measures may account for some of the discrepancies. Nevertheless, the basic tenets of the type I/II distinction appear supported, although perhaps not across all features that have been proposed as important to it.

It is possible, however, that there is some inherent "noise" in the present definitions of positive and negative symptoms; perhaps more than one or two dimensions are being measured. It is often the case that initially unitary or dichotomous constructs require further subdivision into more homogeneous ones, and that the revised constructs enable better prediction.

We present here some of the preliminary results from an ongoing multidisciplinary investigation of brain-behavior relationships in schizophrenic disorders which includes historical, symptomatic, neuropsychological, biochemical, cerebrometabolic, and neuroradiographic components. The focus of this article is on positive and negative symptoms, as defined by Andreasen and Olsen (1982), and the

relationships of such symptoms to neuropsychological performance.

Methods

Patients. The study was conducted on the Columbia-Creedmoor Special Treatment Unit (STU) at Creedmoor Psychiatric Center (CPC). Patients signed informed consent for participation in the assessment procedures. Diagnosis according to Research Diagnostic Criteria (RDC) (Spitzer, Endicott, and Robins 1977, 1978) was based on a structured interview using the Schedule for Affective Disorders and Schizophrenia (SADS) (Endicott and Spitzer 1978) and additional information from patients' records and from family members when available. Exclusion criteria were history of alcoholism or drug abuse, known neurological disease or mental retardation, or electroconvulsive therapy within the previous 6 months. Since our multidisciplinary research involves radiological procedures (CT scans and regional cerebral blood flow studies), subjects with previous major exposure to radiation (> 6 rads within the previous year) and females with positive pregnancy tests were also excluded. The RDC diagnoses for the episode leading to present hospitalization were chronic schizophrenic disorder = 30 (paranoid = 13; undifferentiated = 8; disorganized = 6; catatonic = 2; mixed = 1) and chronic schizoaffective disorder, mainly schizophrenic subtype = 2.

Subject characteristics for this sample are shown in table 1. Patients admitted to the STU for participation in the protocol were all from the CPC catchment area, which is a primarily middle class community. Twenty-two patients were white, eight were black, and one each were of Asian or Hispanic descent.

Table 1. Sample characteristics (n = 32; 16 males, 16 females)

Variable	Mean	SD	Range
Age (years)	32.5	7.6	20-48
Educational level[1]	4.0	1.3	1-7
Age at onset (years)	19.8	4.1	14-29
Illness duration (years)	12.5	7.4	2-26
Full Scale IQ (WAIS-R)	81.2	11.2	67-112
Verbal IQ (WAIS-R)	88.4	15.4	70-133
Performance IQ (WAIS-R)	75.4	7.8	61-96

[1] Hollingshead/Redlich (1958): "0" = no formal education, "4" = completed high school, "7" = completed graduate or professional school.

Means of Verbal and Full Scale IQ scores were in the low average range, while Performance IQ was in the borderline range of intellectual functioning. On tests widely used as screening instruments for the identification of brain damage (Purdue Pegboard, Trail-Making Test, Finger-Tapping Test), 70 to 96 percent of the sample was classified as brain-damaged (Bilder 1984) according to standard cutoff scores (Halstead 1947; Goldberg and Smith 1976; Reitan 1979).

All patients were treated by staff psychiatrists during their participation in the study. During the assessment period, all but one of the patients (who was receiving no medication) were receiving neuroleptic medications; one patient received imipramine (25 mg/day) in addition to haloperidol; four received benztropine mesylate and one diphenhydramine HCl in addition to neuroleptics.

Assessments. The data presented here are based on psychiatric and neuropsychological assessments. The assessments for a given subject were performed within a 2-week period; each assessment was performed independently and without knowledge of the results of other examinations.

The *psychiatric assessments* included several published interview schedules and rating scales. The measures of positive and negative symptoms were derived from the SADS and the SANS (Andreasen 1981).

No widely accepted scale for measuring positive symptoms has been developed. We followed the definitions presented in the classification criteria of Andreasen and Olsen (1982) in selecting measures of delusions, hallucinations, bizarre behavior, and positive formal thought disorder to represent positive symptoms. The first three of these were measured as single items from the SADS (severity of delusions, severity of hallucinations, and bizarre behavior). A measure of positive formal thought disorder was formed by summing raw scores on three items from the SADS (impaired understandability due to thought disorder, loosening of associations, and illogical thinking). A fifth measure was constructed to reflect the *range* of positive symptoms independent of the severity of individual symptoms. This "breadth of psychosis" scale was calculated as the sum of scores on 15 types of

delusions or hallucinations (each of which was rated on a scale from 0 to 3, from *absent* to *definitely present*). The items included in this scale were delusions of reference; delusions of being controlled or influenced; delusions of mind being read; thought broadcasting; thought insertion; thought withdrawal; persecutory delusions; delusions of jealousy, delusions of guilt or sin; grandiose delusions; somatic delusions; auditory hallucinations; visual hallucinations; somatic or tactile hallucinations; and olfactory hallucinations.

Negative symptoms were rated using the SANS, a 30-item scale composed of five subscales: affective flattening, anhedonia, avolition/apathy, alogia, and attentional impairment. The subscale scores are reported here; each score is the sum of objective ratings on the items comprising that subscale.

The *neuropsychological assessments* included over 35 tests administered in two batteries. The *standard* battery, which was usually administered in one session, comprised the Wechsler Adult Intelligence Scale, Revised Edition (WAIS-R); Raven Standard Progressive Matrices; Wechsler Memory Scale, Russell Revision; Purdue Pegboard; and other tests not discussed here. The *specific* battery, including 30 tests selected for their relevance to discrete neuropsychological systems, was usually administered in a separate session. To avoid the effects of fatigue on a given function or test, tests within the specific battery were arranged in four different counterbalanced administration sequences, maximizing the temporal separation of tests with similar task demands or difficulty. The four counterbalanced orders were assigned sequentially to consecutive protocol admissions. Total testing time ranged from 6 to 20 hours per patient.

Linear combinations of neuropsychological test variables were used to form seven *functional* scales: motor, somatosensory, perceptual, language, memory, attentional, and executive. A *global* scale was also constructed. A detailed rationale for the selection of tests and assignment of variables to scales is provided elsewhere (Bilder 1984, pp. 64–79). The domain of neuropsychological functional systems (Luria 1980) was selected first, and tests were selected from existing batteries and the experimental literature to represent these systems. The assignment of variables to each scale is shown in the Appendix. Raw scores on each of the test variables were standardized (to mean = 0; SD = 1) before inclusion in scales.

Statistical Analyses. For certain patients not all data could be obtained due to lack of cooperation, and data were excluded in cases where the reliability of ratings or test performance was questioned by the rater/examiner. Complete symptom ratings were not available for two patients. Two patients refused all neuropsychological testing, and all results for one other patient were rejected. Only 22 patients completed all neuropsychological tests. The total *n* for analyses involving neuropsychological scales thus ranges from 22 to 29. Calculations were performed on all nonmissing data. All variables (psychiatric scale scores, neuropsychological scale scores) were standardized (to mean = 0; SD = 1) before entry into subsequent analyses.

All correlations reported are Pearson product-moment coefficients. No a priori hypotheses were made concerning the direction of correlations among symptoms or between symptoms and the neuropsychological test scores; therefore, only two-tailed probability levels are reported. One exception is seen in tests for the difference between correlated coefficients of correlation, for which one-tailed tests are implicit (Guilford and Fruchter 1978).

Results

Positive and Negative Symptoms. Correlations within the set of positive symptoms are shown in table 2. The correlation between bizarre behavior and positive formal thought disorder was significantly

Table 2. Correlations within the set of positive symptoms (n = 30)

Symptom measure	2	3	4	5
1. Delusions	.51[1]	.51[1]	.40	.50[1]
2. Hallucinations	—	.51[1]	.35	.46[1]
3. Bizarre behavior		—	.82[2]	.05
4. Positive FTD[3]			—	.07
5. Breadth of psychosis				—

[1] $p < .01$, 2-tailed.
[2] $p < .0001$, 2-tailed.
[3] FTD stands for formal thought disorder. See text for variable definitions.

greater than the correlations of either variable with delusions, hallucinations, or the breadth of psychosis scale (the smallest t statistic for the difference between correlated coefficients of correlation was $t_{dr} = 2.71$, $p < .001$).

Inspection of the correlations within the set of negative symptoms (table 3) shows strong correlations: (1) between alogia and attentional impairment; and (2) between affective flattening and avolition/apathy. These correlations were significantly greater than any other intraset correlations (minimum $t_{dr} = 1.71$, $p < .05$). Although anhedonia appeared to relate more strongly to avolition/apathy or affective flattening than to alogia, the differences between these correlation coefficients were not significant ($p > .05$).

Correlations between the positive and negative sets of symptoms are shown in table 4. Noteworthy are the large positive correlations of bizarre behavior and positive formal thought disorder from the positive symptom set with alogia and attentional impairment from the negative symptom set. There were no significant ($p < .05$) negative correlations between positive and negative symptoms.

Table 3. Correlations within the set of negative symptoms ($n = 30$)[1]

Symptom measure	2	3	4	5
1. Affective flattening	.43	.78[2]	.60[3]	.47[4]
2. Alogia	—	.36	.41	.79[2]
3. Avolition/apathy		—	.61[3]	.40
4. Anhedonia			—	.57[3]
5. Attentional impairment				—

[1] See Andreasen (1981, 1982) for variable definitions.
[2] $p < .0001$, 2-tailed.
[3] $p < .001$, 2-tailed.
[4] $p < .01$, 2-tailed.

To illustrate the pattern of correlations among the positive and negative symptoms, the results of principal components analysis are shown in table 5. The initial solution produced three principal components with Eigenvalues greater than 1.0, which together accounted for 78 percent of the variance. The rotated (Varimax) factor pattern is also shown in table 5. Factor 1, with high loadings on positive formal thought disorder, bizarre behavior, alogia, and attentional impairment, appears to represent a disturbance of thought, attention, and the organization of behavior which is not definable as either a positive or a negative symptom set. Factor 2 had highest loadings on those negative symptoms (affective flattening, avolition/apathy, and anhedonia) that were found to be independent of other negative symptoms in the within-set correlative analysis, and independent of positive symptoms in the between-sets analysis. This factor appears to reflect primarily blunting of affect and volition. Factor 3, with highest loadings on delusions, hallucinations, and the breadth of psychosis scale, could be considered a reflection of florid psychotic phenomena.

Neuropsychological Correlates. Scores on the rotated factors were computed for each subject and correlated with neuropsychological scale scores. The correlations of factors 1, 2, and 3 with each of the neuro-

Table 4. Correlations between positive and negative symptoms ($n = 30$)

	Positive symptoms				
Negative symptoms	Delusions	Hallucinations	Bizarre behavior	Positive formal thought disorder	Breadth of psychosis
Affective flattening	.25	.06	.12	.13	−.14
Alogia	.10	−.02	.44[1]	.59[2]	−.19
Avolition/apathy	.32	.19	.15	.14	−.07
Anhedonia	.24	.27	.18	.12	−.03
Attentional impairment	.26	.29	.59[2]	.62[3]	−.15

[1] $p < .05$, 2-tailed.
[2] $p < .01$, 2-tailed.
[3] $p < .001$, 2-tailed.

Table 5. Principal components analysis of positive and negative symptoms[1]

Initial solution	EV[2]	Pr[3]	Cum[4]
Factor 1	4.16	.416	.416
Factor 2	2.18	.218	.633
Factor 3	1.50	.150	.783
Rotated factor pattern			
	Factor		
Symptom measure	1	2	3
Positive			
Delusions	.25	.24	.77
Hallucinations	.26	.10	.76
Bizarre behavior	.80	.01	.45
Thought disorder	.88	−.02	.36
Breadth of psychosis	.17	−.12	.79
Negative			
Affective flattening	.13	.90	−.01
Alogia	.80	.36	−.27
Avolition/apathy	.08	.90	.07
Anhedonia	.19	.81	.07
Attentional impairment	.81	.45	−.02
Variance explained	2.93	2.68	2.22

[1] See text for scale definitions. Rotation method was Varimax (Statistical Analysis System 1982).
[2] Eigenvalue.
[3] Proportion of variance explained.
[4] Cumulative proportion of variance accounted for.

Table 6. Correlations between symptom factors and neuropsychological scale scores

		Factor			
Scale	***n***	1	2	3	**Differences[1]**
Motor	24	−.08	−.05	−.31	
Somatosensory	24	−.27	−.01	.03	
Perceptual	24	−.33	.29	.02	1 > 2
Language	24	−.58[2]	−.06	−.04	1 > 2,3
Memory	22	−.61[2]	−.11	.04	1 > 2,3
Attentional	22	−.35	−.35	.17	1,2 > 3
Executive	23	−.25	−.17	.31	1 > 3
Global	26	−.41[3]	−.10	.23	1 > 3

[1] The inequalities indicate which factors were more correlated with impairment on neuropsychological scales; assessed by *t* tests for the difference between correlated coefficients of correlation, $p < .05$.
[2] $p < .005$, 2-tailed.
[3] $p < .05$, 2-tailed.

psychological scales are shown in table 6. The neuropsychological scales were all computed so that higher scores indicate better performance; thus, negative correlations suggest associations of higher symptom factor scores with poorer neuropsychological performance. Differences between the correlations of each neuropsychological scale with factors 1, 2, and 3 were assessed using *t* tests for differences between correlated coefficients of correlation. Factor 1 was more highly correlated than factor 3 with poor performance on the language, memory, attentional, executive, and global scales; factor 1 was more highly correlated than factor 2 with poor performance on the perceptual, language, and memory scales; and factor 2 was more highly correlated than factor 3 with poor performance on the attentional scale.

Comment

This preliminary report raises certain questions about the validity of the positive/negative symptom construct as it is now widely interpreted. Support for the construct would have been provided if both positive and negative sets of symptoms were internally homogeneous, and if positive and negative symptoms were negatively correlated. Instead, the results suggest heterogeneity within both positive and negative domains of symptoms. Furthermore, negative correlations were not detected between positive and negative symptoms; in fact, significant positive correlations were found between positive and negative symptom sets. This study thus fails to support the hypothesis that positive and negative symptoms define either a dichotomy or opposite ends of a positive/negative symptom continuum. Such findings are at variance with those of Andreasen

and Olsen (1982) but are consistent with results recently reported by others (Lewine, Fogg, and Meltzer 1983; Rosen et al. 1984).

The exploratory correlational analyses revealed distinct clusters of symptoms within predefined positive and negative sets. Within the positive set of symptoms, positive formal thought disorder and bizarre behavior were distinguished from delusions and hallucinations. Within the negative set of symptoms, attentional impairment and alogia were differentiated from affective flattening, avolition/apathy, and anhedonia. It appeared that within each set of symptoms, a cluster reflecting a disturbance of thought, attention, and the organization of behavior could be separated from other symptoms unique to positive or negative domains. This interpretation was supported by the between-sets analysis, showing that the positive and negative symptoms of disorganization were positively intercorrelated. Such findings, if replicated, would indicate that a process independent of the positive/negative distinction may be important in understanding the symptomatic presentations in chronic schizophrenic illnesses.

The results of correlational analyses were summarized in a principal components analysis. The rotated factor pattern concisely illustrates the separate symptom clusters obtained in this sample. Factor 1 had high and unique loadings on the symptoms of disorganization drawn from both positive and negative symptom sets; factor 2 loaded highly only on symptoms from the negative set; and factor 3 loaded highly only on symptoms from the positive set. Thus, considering only factors 2 and 3, support for a revised positive/negative distinction could be suggested, if positive symptoms were represented primarily by delusions and hallucinations, and negative symptoms were represented by the blunting of affect and volition. The symptoms loading on factor 1 would be seen as orthogonal to both positive and negative symptoms. Such a scheme implies that the differentiation between positive and negative forms of thought disorder may be irrelevant. It is noteworthy in this context that Lewine, Fogg, and Meltzer (1983) found bizarre behavior and loosening of associations to be the items with poorest fit to scales of positive and negative symptoms developed with Rasch models. Their results are thus consistent with ours and support the idea that these symptoms may be relatively independent of the positive/negative distinction.

Poor performance on brief tests of cognition and mental state has been reported to be associated with the "negative" symptom profile (Johnstone et al. 1978*b*; Andreasen and Olsen 1982). In this study, neuropsychological deficits were most strongly correlated with factor 1 scores. In support of Crow's (1980) hypothesis that type I patients should show less cognitive impairment, factor 3 scores (i.e., delusions and hallucinations) were nonsignificantly correlated with neuropsychological test performance. Factor 1 and (to a lesser extent) factor 2 were selectively associated with neuropsychological compromise. These results are consistent with the results of previous studies, but suggest that a process independent of the positive/negative distinction may be a more valid predictor of neuropsychological impairment.

Perhaps stronger relationships between symptom profiles and neuropsychological performance would have been found in other studies if different clusters of symptoms had been identified. The results of the present study suggest that the symptoms of factor 1 shared the most variance with neuropsychological defects. Had we simply correlated the predefined positive and negative symptom summary scores with the neuropsychological test results, the differences between symptom clusters would have been obscured (Rieder et al. 1984).

It is possible that overall levels of impairment may play a contributory role in the production of different symptom clusters and their correlations with neuropsychological measures. Patients in this sample were severely impaired. Many had been institutionalized for the greater part of their illnesses. Overall levels of intelligence were quite low, and conspicuous neuropsychological deficits were prevalent. It is conceivable that the pattern we observed, with a strong representation of disorganized features on factor 1, is due to the inclusion of more severely impaired patients. Studies of larger samples with a wide range of social and intellectual impairments are needed to address this question.

The profile of neuropsychological impairments correlated with factor 1 prompts speculation about the process of cognitive compromise that could lead to such an association. Highest correlations were found with the language, memory, and global neuropsychological scales; in previous analyses (Bilder 1984), these scales were found to correlate highly with estimates of premorbid intellectual functioning and educational achievement, and to be relatively independent from other scales (attentional, executive) that are generally considered more sensitive to the effects of acute brain insult. It is possible, therefore, that the "diffuse"

global deficits associated with factor 1 scores in this sample represent longstanding, static dysfunction—a pattern consistent with developmental compromise. Valid inferences about neuropsychological process cannot be drawn from the limited data presented here, but we think it is important for neuropsychological studies to distinguish between deficits associated with failure in the normal development of cognitive abilities (perhaps due to genetic and/or perinatal traumatic factors) and deficits that occur later in life after relatively adequate cerebral development.

Because of the small sample, missing data, and the preliminary stage of our investigations, we would caution against making definitive inferential statements at this point. We do not mean to propose expansion of the type I/type II or positive/negative symptom dichotomies to a "trichotomous" construct. Moreover, we do not report here any attempts to subtype patients. When clusters of symptoms are detected, it does not imply that any patient will be found to manifest one cluster of symptoms and not others.

Our findings are consistent with the hypothesis that a process independent of the positive/negative distinction may be associated with widespread neuropsychological impairment. We suggest that investigators of the "defect state" in chronic schizophrenia consider the possibility that it is not a monothetic construct. The results of this study could be seen as evidence that the defect state may include distinct processes involving the blunting of affect and volition on the one hand, and a more purely cognitive disturbance on the other. This distinction is slightly different from that made by Lewine, Fogg, and Meltzer (1983, p. 368), who interpret their results to support the view that "cognitive-affective negative symptoms, and social withdrawal" are independent. Our findings are consistent with theirs, however, in suggesting that separate processes may exist within the broadly defined domain of type II symptoms.

It appeared plausible from our results that the symptoms of cognitive disorganization could be associated with failure in the development of a normal repertoire of cognitive abilities. The specificity of these dysfunctions to the schizophrenic syndrome is unknown. Future research on such defects in psychiatric disorders and in illnesses of known etiopathology are needed to resolve this and related issues.

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Appendix: Composition of Neuropsychological Scales

Each of the neuropsychological scales was calculated as the sum of standardized (to mean = 0; SD = 1) raw scores on the individual variables that comprise that scale (with equal weights assigned to each variable) divided by the number of variables comprising the scale. The scales are formed so that higher scores indicate better functioning. A list of references for each of the tests has been published elsewhere (Bilder 1984, pp. 177–179) and is available on request.

Motor = Hand Grip Strength (total of three trials with each hand)
- \+ Finger-Tapping Test (total of best three 10-second trials within five taps, with each hand)
  Motor Performance Battery (total time to complete 12 tasks of repetitive and alternating movements of the fingers, forearms, and feet)

Somatosensory = Graphesthesia (finger tip number writing perception, four trials/finger on each hand)
- \+ Finger Gnosis (40 trials/hand of finger identification, number of fingers touched, and number of fingers between fingers touched)
- \+ Right-Left Discrimination Test (identification of right and left in intrapersonal and extrapersonal space, 32 trials)

Perceptual = Spatial Block Span (maximum sequence performed correctly on at least one of two trials)
- \+ Facial Recognition Test (total correct, maximum of 54 items)
- \+ Perceptual Closure (identification of fragmented figures, total correct of 26 items)
- \+ Poppelreuter Figure Analysis (identification of overlapping line drawings of objects, total correct of 14 items)

Language = Verbal Fluency (controlled word association; total of different words produced/60 seconds in each of two phonemic and two semantic categories)
- − Auditory Discrimination Test (error score, discrimination between words differing by a single phoneme)
- \+ Sentence Repetition (total number of sentences repeated correctly, maximum = 22)
- \+ Picture Naming (total correct, pictures of 22 objects and animals)
- \+ Responsive Naming (total correct responses to 10 descriptions)
- \+ Complex Ideational Material (comprehension, total correct answers to 12 questions)
- \+ Similarities (subtest of WAIS-R, total raw score)

Memory = Logical Memory (subtest of Wechsler Memory Scale, total number of ideas recalled from two paragraphs)
- \+ Visual Reproductions (subtest of Wechsler Memory Scale, total raw score)
- \+ Associate Learning (subtest of Wechsler Memory Scale, total raw score)
- \+ Paired Face Recognition (recognition of faces previously paired, total correct of 20 trials)
- \+ Recurring Figures Test (continuous recognition of previously presented geometric and nonsense forms, total score over 140 trials corrected for false alarms)
- \+ Selective Reminding Test (total words recalled over 15 trials, 10-word semantically homogeneous list)

Attentional = Digit Symbol Substitution (subtest of WAIS-R, total number/90 seconds)

+ Digit Span (subtest of WAIS-R, total raw score forward)
− Trail-Making Test (Part B, total time)
− Trail-Making Test (total errors)
− Target-Detection Tests (total time to complete five different tasks requiring "cancellation" of numbers, letters, and geometric forms)
+ Target-Detection Tests (total hits)

Executive = Competing Programs Tests (total correct in two tasks requiring responses to conflicting instructions, 20 trials/task)
+ Wisconsin Card-Sorting Test (maximum categories achieved)
− Wisconsin Card-Sorting Test (total number of perseverative errors)
+ Picture Arrangement (subtest of WAIS-R, total raw score)

Global = WAIS-R (sum of all subtest raw scores)
+ Raven Standard Progressive Matrices (total correct)

Family Therapy in Schizophrenia

Family Therapy in Schizophrenia, edited by William R. McFarlane, has been recently published by The Guilford Press (200 Park Avenue South, New York, NY 10003). Although family therapy originated nearly three decades ago through early efforts to understand the etiology of schizophrenia, it was all but abandoned in later years, as biological or constitutional factors were shown to contribute more to the occurrence of schizophrenia than family psychopathology. Though the etiology of the disorder is still understood in constitutional terms, recent findings suggest that the family—the key social unit in the patient's life—may have a substantial impact on treatment.

Family Therapy in Schizophrenia focuses on approaches developed since 1975. These approaches, brought together here for the first time, differ from earlier ones, according to McFarlane, in two important ways: "They seem to have major therapeutic effects on the schizophrenic process, beyond those achievable with drug therapy; and they all—with the exception of the systemic variety—start from a major expansion of family systems theory that include extrafamily factors."

This volume presents practical strategies—developed by leading family therapists and researchers—for involving families of schizophrenics in the therapeutic process. The book is addressed to family clinicians, psychiatrists, rehabilitation counselors, psychiatric nurses and social workers, hospital and clinic administrators, and students in training for years to come.