Neuropsychological Correlates of Alogia and Affective Flattening in Schizophrenia

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We examined whether two negative symptoms, alogia and affective flattening, were more strongly associated with dysfunction of certain brain regions than with others in a sample of 27 schizophrenic subjects. Neuropsychological tests were used to measure the integrity of functioning of different brain regions. Functioning of left and right frontal regions was assessed by word fluency and design fluency, respectively, and functioning of the left temporohippocampal region was assessed by Hebb's digit-sequences recall test. Measures of alogia and affective flattening were obtained using the Scale for the Assessment of Negative Symptoms. The results indicated that more severe levels of these negative symptoms were associated with poorer performance on the fluency tests. These relationships were fairly specific as indicated in two ways: (1) only certain neuropsychological measures were associated with negative symptoms; and (2) poor performance on fluency tests was not associated with all psychiatric symptoms. In addition, affective flattening, but not alogia, appeared to be associated with lateralized dysfunction.

Key Words: Alogia, affective flattening, schizophrenia, temporolimbic system, neuropsychological approach

Introduction

Investigators have suggested that many different brain regions, including the frontal lobes (e.g., Weinberger et al 1986), the left hemisphere (e.g., Gur et al 1983), and the temporolimbic system (e.g., Saykin et al 1991), are disturbed in schizophrenics. As pointed out by Meehl (1990), it is unlikely that disruption of a single brain region can account for the significant heterogeneity of schizophrenic disturbances. In order to address the problems raised by this heterogeneity, several psychopathologists (e.g., Bannister 1968; Persons 1986; Costello 1992) have recommended examining schizophrenia from the perspective of individual symptoms instead of, or in addition to, the perspective of global diagnostic categories. The fruitfulness of this approach has been realized in furthering our understanding of the genetic contribution to schizophrenia (e.g, Berenbaum et al 1990) and the therapeutic efficacy of antipsychotic medication (e.g., Johnstone et al 1988). To date, this strategy of focusing on individual symptoms has not been utilized commonly in the examination of brain–behavior relationships in psychopathology.

Although it is unlikely that dysfunction of a single brain region can account for all of the symptoms of schizophrenia, it seems plausible that dysfunction of some brain

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regions may be more strongly associated with certain symptoms, whereas dysfunction of other brain regions may be more strongly associated with other symptoms. One advantage of a symptom-level approach is that it makes one's results more meaningful in terms of normal function. For example, instead of knowing that disruption of the left frontal cortex is more common among hebephrenic schizophrenics than among paranoid schizophrenics, it would be more useful to know that dysfunction of this brain region is associated with the disordered speech displayed by these patients. An additional advantage of this approach is that it allows investigators to ask whether the association between brain dysfunction and a given symptom occurs across psychiatric disorders. For example, one might ask if disturbances of the basal ganglia are associated with motor retardation regardless of the individual's psychiatric disorder (e.g., schizophrenia or major depression).

The goal of this study was to use a neuropsychological approach to examine which brain regions are associated with two symptoms of schizophrenia, alogia and affective flattening. Because such an approach allows specific brain regions to be linked to specific psychological functions, neuropsychological testing can give psychological meaning to the disturbances in brain structure and metabolism uncovered by approaches such as magnetic resonance imaging (MRI) and positron emission tomography (PET). We hypothesized that alogia and affective flattening would be associated with disturbances in frontal lobe functioning for two reasons: (1) both alogia and affective flattening have been found among neurological patients with frontal lobe damage (e.g., Stuss and Benson 1986); and (2) several researchers have found an association in schizophrenics between frontal lobe functioning and negative symptoms (e.g., Katsanis and Iacono 1991; Wolkin et al 1992). In addition to our interest in examining whether alogia and affective flattening are associated with frontal lobe functioning, we wished to examine whether these symptoms are related to lateralization of function, that is, whether they are more strongly associated with functioning of the right frontal or left frontal region.

Research to date on lateralization of affective flattening has yielded equivocal results. In neurological patients, links have been found between affective flattening and both right-frontal (Ross 1981) and left-frontal disturbance (Kolb and Milner 1981). In one of the first studies to examine the neuropsychological correlates of negative symptoms in schizophrenics, Mayer et al (1985) found that affective flattening, as measured by the Scale for the Assessment of Negative Symptoms (SANS), was associated with right hemisphere dysfunction. In contrast, Gruzelier et al (1988) found an association between social withdrawal and left-sided anterior functioning, and Liddle (1987) found an association between a psychomotor poverty syndrome (which included, but was not limited to, poverty of affect) and dysfunction of the left dorsolateral frontal region. Blanchard et al (1991) found that a left-frontal verbal fluency test was the only neuropsychological measure that was associated with scores on the Brief Psychiatric Rating Scale (BPRS) for anergia (which includes emotional withdrawal and blunted affect) after partialing out "disorientation" ratings. However, in the same sample of subjects, Blanchard et al (in press) did not find an association between affective flattening (measured independently using the SANS, as well as on the basis of facial expressiveness in response to filmclips) and frontal lobe functioning. Thus, the results of previous research have been quite inconsistent. One possible cause of these inconsistent results is that investigators have varied in the type of symptom measures they have used (e.g., whether they examined social withdrawal or affective flattening). A second possible factor that may have contributed to the inconsistent results is that investigators have not always attended to issues of laterality when examining the anterior–posterior dimension, or to the anterior–posterior dimension when examining the issue of laterality.

Most of the evidence suggests that alogia should be associated with disturbed left-frontal functioning. Not only has there been evidence that frontal-damaged patients with poor initiation of speech tend to have lesions to the left supplemental motor area, but there is an extensive literature (see Geschwind 1972, for a review) supporting the notion that the left hemisphere is responsible for speech production. Assuming that alogia represents a specific language disturbance, the expectation would be that alogia should be associated with left-frontal and not right-frontal functioning. However, it is possible that alogia in schizophrenic individuals represents a more general deficit in the initiation of behavior that extends into both the verbal and spatial domains. The effects of such a deficit would be most noticeable in everyday life in the verbal domain due to its deleterious impact on the spontaneous generation of speech. Although disruptions in the generation of material in the visuospatial domain would also result from this latent deficit, it would be less noticeable than alogia because our society depends primarily on verbal communication. If alogia represents the overt manifestation of a more general underlying disturbance in the initiation and generation of behavior, we would expect it to be associated with both right- and left-frontal disturbance.

Thus, the goal of this study was to examine whether alogia and affective flattening are more strongly associated with functioning of certain brain regions than with others. Based on past research, we predicted that both of these symptoms would be more strongly associated with frontal lobe function than they would be with the functioning of other brain regions. In particular, we contrasted the con-

tribution of frontal regions with that of temporolimbic ones. Furthermore, we wished to explore the degree to which the left frontal as compared to the right frontal region contributed to alogia and affective flattening. We also attempted to rule out the possibility that any associations we found between frontal functioning and alogia and affective flattening were artifacts of intervening variables such as age or sex. Finally, we wished to determine the degree to which frontal functioning specifically predicts alogia and affective flattening as compared to other symptoms of schizophrenia, such as hallucinations and delusions.

Methods

Subjects

Subjects were 27 individuals with DSM-III-R diagnoses of schizophrenia who were hospitalized at the time of their participation in the research project. Diagnoses were based on interviews conducted using the psychotic and mood disorders sections of the Structured Clinical Interview for DSM-III-R (Spitzer et al 1992), and a review of subjects' clinical records. The interviewers, who also made the clinical ratings described below, were a Ph.D.-level clinical psychologist (HB) and an advanced doctoral candidate in clinical psychology (DB), both of whom have had extensive experience using a variety of different structured clinical interviews and clinical rating scales. Each interview was conducted by only a single interviewer. Subjects ranged in age from 21 to 57 years (mean 36.0, SD 10.7). The majority of subjects (78%) were males. Developmental disability, alcohol or drug abuse, electroconvulsive therapy (ECT) within the previous 6 months, a history of seizures, head trauma, and/or neurological damage, English as a second language, or a score below 25 on the Mini Mental Status Exam (Folstein et al 1975) were causes for exclusion from this study.

Subjects' education levels ranged from grammar school to graduate/professional school (median was completion of high school). The number of years since first psychiatric hospitalization ranged from <1 to 34 years (mean 14.4, SD 9.5). The length of the current hospitalization ranged from 4 to 1295 days (mean 267, SD 407). Antipsychotic medication levels (in chlorpromazine equivalents, with slow-release dosages adjusted to a daily equivalent) ranged from 0 to 4625 mg/day (mean 1230.7, SD 1226.0).

Procedure

We used the SANS (Andreasen 1983) to rate alogia and affective flattening. SANS items are rated using a six-point scale (0 = not at all/none; 5 = severe). The alogia score used in our analyses was computed by averaging scores on the "poverty of speech" and "latency of response" items. The item "poverty of content of speech" was omitted because previous research suggests that poverty of speech and poverty of content of speech may reflect different underlying disturbances (Berenbaum et al 1985). The final item on the SANS alogia scale, "blocking," was omitted because none of the subjects exhibited it. The affective flattening score used in our analyses was computed by averaging scores on the "unchanging facial expression," "poor eye contact," "affective nonresponsivity," and "lack of vocal inflections" items. The item "inappropriate affect" was omitted because we wished to distinguish between inappropriate and flat affect. Because of their association with depression and motor retardation, the items "decreased spontaneous movements" and "paucity of expressive gestures" were omitted as recommended by Knight and Roff (1985).[1] The internal consistencies of the alogia and affective flattening scales, measured using Cronbach's alpha, were 0.88 and 0.70, respectively. Hallucinations and delusions were rated using the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen 1984). For the purpose of the present study, we used the global hallucinations and delusions scores. The alogia and affective flattening scores were significantly correlated with each other ($r = .71$, $p < .001$), as were hallucinations and delusions ($r = .37$, $p < .05$). Therefore, we created negative symptom scores by averaging standardized alogia and affective flattening scores, and positive symptom scores by averaging standardized hallucinations and delusions scores.

The experimental portion of the study was conducted in two 1-hour sessions held within 50 hours of the clinical interview. In this second stage of the study, a researcher who was unaware of the results of the clinical assessments administered a series of neuropsychological tests, as well as tests of handedness, motor retardation, and general intelligence.

Handedness was measured using an eight-item questionnaire that queried the subject about hand use in eight common actions (e.g., throwing a ball). Only two subjects were left-handed based on the criterion of using the left hand for writing and/or use of the left hand for nonwriting activities an average of >20% of the time. Removal of the data for these individuals did not significantly alter the results presented below. Motor retardation was measured using a finger-tapping test

[1] The correlations between the alogia and affective flattening scale scores used in the analyses below and the global alogia and affective flattening scores were 0.98 ($p < .001$) and 0.87 ($p < .001$), respectively. Not surprisingly, therefore, the pattern of results did not differ when global alogia and affective flattening scores were used instead of the scale scores based on the sum of selected individual items.

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(Lezak 1983). To assess general intelligence, subjects completed the Vocabulary and Picture Completion subtests of the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler 1981). These subtests were chosen because they are the best measures of general mental ability within the Verbal and Performance Scales of the WAIS-R (Lezak 1983). The average test-retest reliabilities of the Vocabulary and Picture Completion tests are reported as 0.92 and 0.88, respectively (Wechsler 1981). Because the Vocabulary and Picture Completion test scores were significantly correlated ($r = .42$, $p < .05$), we computed average intelligence quotient (IQ) scores by averaging subjects' standardized Vocabulary and Picture Completion scores.

Left- and right-frontal functioning were examined using the word and design fluency tests, respectively (Thurstone and Thurstone 1943; Jones-Gotman and Milner 1977). The word fluency test measures the number of different English words that begin with a specific letter that an individual can produce within a particular time frame. The design fluency test measures the number of different abstract designs than an individual can produce within a particular time frame. The "restricted" version of the design fluency test (create as many drawings as possible that can be constructed with four straight lines) was examined because it is more strongly associated with lesions of the right-frontal lobe than the "nonrestricted" version (Jones-Gotman and Milner 1977), and because it is easier to score than the nonrestricted version. The fluency tests were scored using the scoring procedures outlined by Jones-Gotman and Milner (1977) by one of the investigators (NS) who was blind to subjects' clinical scores. Because the word and design fluency test scores were significantly correlated ($r = .68$, $p < .001$), we computed average fluency scores by averaging subject's standardized word and design fluency scores. Left-temporohippocampal ability was tested with Hebb's digit-sequences recall test (Hebb 1961). In this test, 24 series of digits, whose length is one item greater than the subject's digit span, are recited by the experimenter. Unannounced to the subjects, one sequence is repeated every third trial, while intervening sequences are never repeated. Only the repeated sequences are used to score performance on this test. The test-retest reliabilities of the word fluency, design fluency, and digit sequences recall tests, examined in a separate nonclinical sample (Stolar 1992), were 0.91, 0.90, and 0.78, respectively.[2]

Table 1. Descriptive Statistics

Measure	Mean	SD	Range
Alogia	1.2	1.6	0–5
Affective flattening	0.8	0.9	0–3.4
Hallucinations-delusions	2.7	1.2	0–5
Word fluency (left frontal)	30.1	15.3	3–56
Design fluency (right frontal)	9.9	7.6	1–30
Digit sequences recall (left temporohippocampal)	2.4	2.7	0–8
WAIS-R Vocabulary[a]	7.7	3.3	2–13
WAIS-R Picture Completion[a]	7.7	2.5	4–12

[a]Scaled scores

Results

We began by examining the distributions of the symptom, neuropsychological, and intelligence test scores. As can be seen in Table 1, there was a reasonable range of scores on all of these measures.

The correlations between the positive and negative symptom scores and the neuropsychological and intelligence test scores are reported in Table 2. As expected, increased negative symptoms were significantly negatively correlated with performance on the fluency tests. In contrast, poorer performance on the fluency tests was not associated with more severe positive symptoms. Furthermore, the correlation between fluency scores and negative symptoms differed significantly from the correlation between fluency scores and positive symptoms ($z = 2.27$, $p < .05$). The severity of negative symptoms was not significantly correlated with performance on the digit se-

Table 2. Correlations between Symptoms and Neuropsychological and Intelligence Test Scores

	Negative symptoms	Positive symptoms
Word/design fluency (frontal)	−0.44[a]	0.31
Digit sequences recall (left temporohippocampal)	−0.03	−0.05
IQ	−0.36[a]	0.48[b]

[a]$p < .05$ (one-tailed)

[b]Because our a priori hypothesis was that more severe symptoms would be associated with poorer performance on neuropsychological and intelligence tests, we could not reject our one-tailed hypothesis when we found that more severe positive symptoms were associated with better performance.

[2]The Corsi block-tapping test (which assesses right-temporohippocampal functioning) and the recency/recognition tests for words and symbols were also administered to subjects; however, they were not utilized in the analyses because of floor effects among the schizophrenics on the block-tapping test and extremely poor test-retest reliabilities of the recency/recognition tests in a sample of college students. Scores on these tests were not significantly correlated with each other or with any of the other variables measured in this study.

quences recall test, indicating that negative symptoms are not significantly associated with all neuropsychological measures. Furthermore, the correlation between negative symptoms and fluency scores differed significantly from the correlation between negative symptoms and digit sequences recall performance ($z = 1.77, p < .05$). Negative symptoms were also significantly negatively correlated with IQ. Although negative symptoms were more strongly associated with fluency scores than with IQ, the difference was not statistically significant.

In order to examine whether the significant correlations reported in Table 2 were inflated due to outliers, we also computed Spearman rank-order correlations. The pattern of Spearman rank-order correlations was similar to that obtained using Pearson product-moment correlations. For example, the Spearman rank-order correlations between negative symptoms and fluency and IQ scores were -0.44 and -0.32, respectively.

Next, we examined whether the significant correlations between negative symptoms and fluency performance and IQ were artifacts of individual differences in demographic variables, medication, or motor retardation. As can be seen in Table 3, the correlations between symptoms, neuropsychological test scores and IQ and the remaining variables tended to be rather small. We also computed partial correlations in which we removed shared variance with these potential nuisance variables. All zero-order correlations that had been statistically significant remained significant when partialing out shared variance with the demographic, medication, and motor retardation variables, with one small exception. The partial correlation between negative symptoms and IQ when removing shared variance with finger tapping was no longer statistically significant, though there was still a trend in the same direction ($r = -.30, p < .08$).

After having found that more severe levels of negative symptoms were associated with worse performance on the fluency tests, we explored the possibility that alogia and affective flattening might be related to lateralization of function. As can be seen in Table 4, alogia was significantly negatively correlated with both design and word fluency. In contrast, affective flattening was significantly associated with design fluency but not with word fluency. In fact, there was a strong trend for affective flattening to be more highly associated with design fluency than with word fluency ($z = 1.58, p < .06$). There was a similar trend for affective flattening to be more highly associated with the nonverbal IQ test than with the verbal IQ test ($z = 1.36, p < .09$).

To further explore the lateralization issue, we computed difference scores in which we subtracted standardized design fluency scores from standardized word fluency scores, and subtracted standardized Picture Completion scores from standardized Vocabulary scores. These difference scores were computed to index lateralized differences in functioning. This was done because raw scores index not only lateralized functioning, but some general level of functioning as well. By computing a difference score, the general level of functioning portion of these scores is eliminated, leaving a measure of lateralization of function. As can be seen in Table 4, there was a trend for affective flattening, but not alogia, to be associated with these difference scores. This suggests that affective flattening is associated with lateralized dysfunction whereas alogia is not. This impression is reinforced when we examined alogia residual scores from which shared variance with affective flattening had been removed, and affective flattening residual scores from which shared variance with alogia had been removed. The correlation between the fluency difference score and the affective flattening residual score was 0.21, compared to a correlation of 0.003 between the fluency difference score and the alogia residual score. The correlation between the IQ difference score and the affective flattening residual score was 0.33 ($p < .05$), compared to a correlation of -0.20 between the IQ difference score and the alogia residual score.

In order to examine whether the correlations reported in Table 4 were inflated due to outliers, we also computed

Table 3. Correlations between Symptom, Neuropsychological, and Intelligence Test Scores, and Sociodemographic, Treatment, and Motor Retardation Variables

	Negative symptoms	Positive symptoms	Word/design fluency	Digit sequences recall	IQ
Age	0.08	0.02	0.07	0.25	0.002
Sex[a]	−0.10	0.28	0.16	0.08	0.13
Education	0.12	0.15	0.31	−0.19	0.33
Length of current hospitalization	0.24	0.01	0.03	−0.12	−0.11
Chlorpromazine unit equivalents	−0.12	0.10	−0.11	−0.09	−0.17
Finger tapping	−0.21	0.23	0.31	−0.35[b]	0.43[b]

[a]Male coded as 1, female coded as 2.
[b]$p < .05$

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Table 4. Correlations between Alogia and Affective Flattening and Neuropsychological and Intelligence Test Scores

	Alogia	Affective flattening
Design fluency (right frontal)	−0.49[c]	−0.45[c]
Word fluency (left frontal)	−0.32[c]	−0.21
Picture Completion (nonverbal)	−0.28[b]	−0.44[c]
Vocabulary (verbal)	−0.24	−0.16
Word minus design	0.21	0.30[b]
Vocabulary minus Picture Completion	0.34	0.26[a]

[c] $p < 0.01$; [b] $p < 0.1$; [a] $p < 0.05$

Spearman rank-order correlations. The pattern of Spearman rank-order correlations was similar to that obtained using Pearson product-moment correlations. For example, the Spearman rank-order correlations between affective flattening and word and design fluency scores were −0.22 and −0.53, respectively.

Discussion

The results of this study indicate that negative symptoms are associated with poor performance on fluency tests that are linked to frontal lobe functioning. Moreover, there is a fair degree of specificity in this relationship. This specificity is demonstrated in two different ways. First, poor performance on the fluency tests was associated with more severe levels of negative symptoms, but was not associated with more severe levels of positive symptoms. Second, negative symptoms were not significantly associated with scores on all neuropsychological tests, but rather were only associated with those tests linked to frontal function. It is important to consider that the differences in the associations of negative symptoms with different neuropsychological tests are probably not attributable to differences in the reliabilities of the tests, all of which were quite reliable in a sample of nonpsychiatric patients. In addition, the association between negative symptoms and fluency test performance does not appear to be an artifact of their being associated with other confounding variables since the associations were not eliminated after partialling out shared variance with medication level, motor retardation, or any of the demographic variables.

What might our findings indicate about the neural disturbance underlying schizophrenia? Our finding that alogia is associated with fluency tests that are presumed to reflect both right- and left-frontal functioning suggests that alogia does not represent a specific verbal disorder. Instead, our results suggest that alogia is a symptom of a more generalized disturbance in the initiation and generation of behavior. Because individuals with alogia are capable of talking, alogia cannot be explained as a simple deficit in motor competence, as one might see in some cases of paralysis. Similarly, because alogia was not significantly correlated with performance on the WAIS-R Vocabulary test, alogia cannot be attributed to a disruption in verbal representations in long-term memory, as one might see in certain forms of amnesia. The association between alogia and poor performance on the two fluency tasks lead us to propose a cognitive mechanism that may underlie poverty of speech. We hypothesize that alogia is the manifestation of an underlying deficit in the ability to access or utilize abstract representations in the absence of external cues. This underlying deficit can reveal itself in a number of different ways, including poor performance on both word and design fluency tasks, and difficulty generating speech. Our hypothesis follows from Goldman-Rakic's (1987) argument that the basic mechanism disrupted in frontal damage that causes poor performance on the word fluency test is "the mechanism(s) by which symbolic representations are accessed and held on line to guide behavior in the absence of (or in spite of) discriminative stimuli in the outside world" (p. 380). Thus, the diminished ability to recruit or utilize representations "on line" for the purpose of achieving a goal leads to difficulty producing words and designs. An example of such a deficit utilizing representations "on line" might be the process of arranging lines in order to produce designs or arranging letters in order to produce words.

Regardless of the veridicality of the hypothesis we have described above, it appears that the deficit that underlies alogia is not limited to the domain of language production and the functioning of the left frontal region. It is not surprising that psychopathologists have focused their attention on alogia rather than nonverbal manifestations of fluency. In normal living, a deficit in word fluency manifested as poverty of speech would be noticed and would be detrimental, whereas a deficit in design fluency would not be as noticed or as detrimental in a society dependent much more on verbal than spatial communication.

In contrast to alogia, affective flattening appeared to be associated with lateralization of function. Affective flattening was quite strongly associated with performance on the design fluency and WAIS-R Picture Completion tests, but was not significantly associated with performance on the word fluency and WAIS-R Vocabulary tests. Our finding that affective flattening was associated with right hemisphere dysfunction is consistent with the findings of Mayer et al (1985), but is inconsistent with the findings of Blanchard et al (in press). There are several possible explanations for the discrepant results. First, we and Mayer et al (1985) studied medicated schizophrenics, whereas Blanchard et al (in press) studied unmedicated schizophrenics. Second, all three studies used different sets of right hemisphere tasks. For example, we studied right frontal lobe functioning, whereas

neither Blanchard et al nor Mayer et al included measures of right frontal functioning. Finally, there may have been important differences in the characteristics of the three samples of schizophrenic patients. For example, potential subjects with scores <25 on the Mini Mental Status Exam were excluded from our study. Our anecdotal observation is that such patients tend to appear quite disorganized, and often do not appear to have flattened affect. Thus, it is possible that including these patients, who would have undoubtedly performed quite poorly on all of the neuropsychological tests, would have obscured the relationship we found between affective flattening and right hemisphere dysfunction. Perhaps Blanchard et al (in press) included such patients in their study, and that is why they did not find a relationship between affective flattening and right hemisphere dysfunction.

We hypothesize that affective flattening, in at least some schizophrenics, is the result of a disturbance in right hemisphere functioning that leads to a deficit in either the ability to access mental representations of actions and expressions, or the ability to utilize the representations (e.g., trigger the action/expression programs) after they have been accessed. This hypothesis bears some resemblance to the proposal by Frith and Done (1988) that negative symptoms are caused by a disturbance in the pathways between the prefrontal lobes and the striatum, resulting in difficulty initiating self-generated action. That affective flattening is the result of a deficit accessing or triggering action/expression programs is not a commonly held view. However, there are several divergent pieces of evidence from research with both nonpsychiatric and psychiatric subjects consistent with this hypothesis. Berenbaum and Rotter (1992) found that nonpsychiatric subjects who exhibited less spontaneous facial expression of emotion performed less accurately on a facial motor control task. In addition, there is abundant evidence that schizophrenics exhibit a wide variety of motor disturbances (Manschreck 1983). Finally, a deficit accessing and/or triggering action/expression programs might also explain why schizophrenics have been noted to report feeling as emotional as normal controls even though they exhibit less facial expression of emotion (Berenbaum and Oltmanns 1992; Kring et al, 1993).

Our finding that affective flattening was not associated with left-frontal functioning is consistent with the findings of Blanchard et al (in press). Although several studies have found associations between left-frontal activity and clusters of symptoms that included affective disturbances (Liddle 1987; Gruzelier et al 1988; Blanchard et al 1991), none of the reported associations were based on affective flattening by itself. In fact, Liddle (1987) found that of the different components of "psychomotor poverty syndrome," affective flattening was least strongly associated with neuropsychological dysfunction. The symptom clusters that have been found to be associated with left-frontal lobe dysfunction have all included either social/emotional withdrawal or motor retardation. Thus, it is possible that social withdrawal and motor retardation, but not affective flattening, are associated with a left-frontal lobe disturbance. This hypothesis is consistent with the model of Davidson and Tomarken (1989) linking approach behavior with relative left-anterior activation.

There are several reasons why the conclusions drawn from the results of the present study should be considered quite tentative. First, the sample was not large, and it was very heterogeneous. Second, although the fluency tasks we used are sensitive to frontal lobe dysfunction, it is quite plausible that poor performance on these tasks could be caused by disturbances in other brain regions. Third, the two negative symptoms were strongly correlated, as were the two fluency tasks and the two intelligence tests. As a result, it would probably be prudent to view the analyses examining individual negative symptoms and lateralization of function as sources of hypotheses worthy of future examination. Finally, although none of the symptom, neuropsychological, or intelligence variables were significantly correlated with chlorpromazine unit equivalents, such analyses are not capable of ruling out potential medication effects (Blanchard and Neale, 1992).

The present study suggests several avenues for future investigation. First, because this study had a reliable measure of functioning of right frontal regions, but not of right posterior regions, it will be important for future research to examine the relationship between alogia and affective flattening and right-temporohippocampal functioning. This will be especially important in order to specify which regions of the right hemisphere are associated with these two symptoms. Second, although the neuropsychological and intelligence tests reported in this paper were all reasonably matched in terms of reliability, they were not all matched in terms of difficulty. The use of tasks matched on difficulty would improve our ability to determine the degree to which there are differential deficits in the functioning of specific brain regions that are associated with specific symptoms (Chapman and Chapman 1989). Finally, it will be important to use imaging techniques such as positron emission tomography (SPECT) and single-photon emission computed tomography in order to verify that the poor performance of schizophrenics with alogia and affective

function. Nonetheless, the present study illustrates the usefulness of employing a symptom level approach in allowing brain–behavior relationships to be revealed. The results suggest that frontal regions may play an important role in the occurrence of negative symptoms. They also suggest that alogia may be the manifestation of a generalized disturbance in the initiation and generation of behavior, rather than being a specific language disturbance, whereas affective flattening may be more specifically linked to lateralized dysfunction.

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