Flat Affect in Schizophrenia: A Test of Neuropsychological Models

by Jack J. Blanchard, Ann M. Kring, and John M. Neale

Abstract

This study evaluated the association between neuropsychological indices of frontal lobe and right hemisphere impairment and deficits in the expression of affect in schizophrenia. The advantages of the present study were that unmedicated schizophrenia patients were studied and relevant demographic characteristics were controlled. Furthermore, deficits in affective expression were assessed both in a clinical interview and in subjects’ response to affect-eliciting films. Despite the fact that schizophrenia subjects demonstrated significantly higher clinical ratings of affective flattening and less facial expression while viewing films than demographically matched control subjects, neuropsychological performance was not consistently related to these expressive deficits. The results thus failed to support for the proposed neuropsychological models of affective deficits in schizophrenia. The characterization of flat affect as a purely emotional deficit is questioned, and limitations of current neuropsychological theories of emotional expression and neuropsychological methods to test these theories in the study of schizophrenia are discussed as relevant concerns for future research.


Flat or blunted affect has been considered an important symptom in schizophrenia owing to its historical and clinical significance. Bleuler (1911/1950) viewed flat affect as a fundamental symptom of schizophrenia and noted its association with poor prognosis. In support of Bleuler’s early observation, contemporary research has provided converging evidence about the diagnostic and prognostic importance of flat affect. Although it is not pathognomonic of schizophrenia, flat affect has been shown to facilitate differential diagnosis (Carpenter et al. 1973), and it is correlated with the diagnosis of schizophrenia across a number of diagnostic systems (Endicott et al. 1986). Flat affect has also been shown to be a relatively enduring symptom that generally responds poorly to treatment (Roff and Knight 1978; Knight et al. 1979; World Health Organization 1979; Pfohl and Winokur 1982, 1983; Pogue-Geile and Harrow 1985). Furthermore, the presence of this symptom is related to poor clinical outcomes on several dimensions, including employment (Pogue-Geile and Harrow 1985; Breier et al. 1991), social functioning (Breier et al. 1991), severity of illness at discharge (Abrams and Taylor 1978), short-term outcome (Endicott et al. 1986), and general long-term outcome (Fenton and McGlashan 1991).

Explanatory models of flat affect have proposed various etiologies, invoking psychodynamic processes such as repression (Arieti 1955; Pao 1979), emphasizing the role of impaired social relations (Roff and Knight 1978) and social rejection (Strauss et al. 1974), and proposing that this symptom is the result of institutionalization (Wing and Brown 1970). A more recent hypothesis has suggested that flat af-
flect is the manifestation of dysfunction in the right hemisphere (Mayer et al. 1985).

The potential significance of right hemisphere dysfunction in flat affect derives from evidence obtained in neurologic populations with frank cortical lesions. Patients with right hemisphere lesions have been described as showing blunted affect or emotional indifference that is strikingly similar to the clinical picture of flat affect seen in schizophrenia (Denny-Brown et al. 1952; Gainotti 1972). Right hemisphere pathophysiology is associated with deficits in the ability to express emotions, both facially (Buck and Duffy 1980; Borod et al. 1986; Kent et al. 1988) and through vocal intonation (Tucker et al. 1977; Ross 1981; Ross et al. 1981; Weintraub et al. 1981). On the basis of such observations, Ross (1981) proposed that the right hemisphere is organized for affective, paralinguistic aspects of communication (e.g., facial expression, vocal intonation, gestures) in a manner homologous to the organization of the left hemisphere for propositional speech (see Borod 1992 for a recent review of the role of the right hemisphere in emotion).

Mayer et al. (1985) evaluated the hypothesis that right hemisphere dysfunction is related to affective flattening in schizophrenia. After controlling for performance on tasks of left hemisphere functioning, demographic variables, and a measure of pause duration in speech (presumably an index of depression), these authors found that right hemisphere functioning was significantly related to flat affect. Other predictors associated with flat affect were extrapyramidal symptoms, inpatient or outpatient status, and pause durations in speech. While these results provide initial support for the right hemisphere model of affective flattening in schizophrenia, interpretation of these results is constrained by several factors.

First, Mayer et al. (1985) evaluated medicated schizophrenia patients; as these authors acknowledged, such a procedure may confound ratings of affective flattening and neuropsychological performance (Blanchard and Neale 1992). Although Mayer et al. (1985) employed a rating scale for extrapyramidal symptoms in an attempt to evaluate the contribution of motor side effects to the findings, this procedure does not remove the possible error variance associated with motor side effects. In addition, demographic variables including handedness and education also pose interpretive difficulties: approximately 15 percent of the schizophrenia subjects in Mayer et al.'s study were left-handed or ambidextrous (Mayer 1983), and the effects of education were not accounted for in any analysis. These factors may cause problems, given the difficulty of establishing laterality in non-right-handed individuals (Filskov and Catanese 1986) and the relationship between education and neuropsychological performance (e.g., Heaton et al. 1986; Warner et al. 1987).

Finally, although it implicated the right hemisphere in flat affect, Mayer et al.'s (1985) study did not include measures of frontal lobe functioning. This is an important consideration, as frontal lobe dysfunction has been implicated in affective behavior (e.g., Blumer and Benson 1975; Weddell et al. 1990) as well as in schizophrenia (e.g., Berman et al. 1986; Weinberger et al. 1986; Weinberger 1987; Buchsbaum 1990). Because of their afferent and efferent connections with limbic structures (Nauta 1971, 1973) and with posterior sensory cortices (Nauta 1971; Powell 1973), it has been proposed that the frontal lobes play an important role in monitoring and modulating the internal milieu as well as in monitoring and responding to the external environment (Nauta 1971). Given such intimate associations with limbic and hypothalamic structures, "extensive lesions of the frontal cortex could be expected to have a marked effect upon the range and differentiation of the subject's viscer-endocrine, affective and motivational responses to his environment" (Nauta 1971, p. 182). Emotional disturbances in patients with frontal lobe damage are generally characterized as diverse (depending on the site and extent of the lesion); however, restricted affective behavior or lack of emotional reactivity has often been observed (Blumer and Benson 1975; Damasio and Van Hoesen 1983; Stuss et al. 1992). Decreased facial expression has been noted in patients with frontal lobe lesions (Kolb and Milner 1981; Weddell et al. 1990). In addition, lesions of the frontal lobe may also result in decreased verbal fluency (Benton 1968) and decreased spontaneous speech (Kolb and Taylor 1981), further contributing to a presentation of restricted affective expression.

Given the evidence suggesting the role of frontal lobe pathology in emotional and nonemotional expressive deficits, the possible role of such pathology in the symptom of flat affect in schizophrenia should be explored. Frontal lobe dysfunction has been hypothesized to be involved in schizophrenia historically (Kraepelin 1919/1971).
and more recently because of the behavioral similarity noted between neurologic patients suffering from frontal pathology and some schizophrenia patients, especially those evincing negative symptoms (e.g., Levin 1984a). Behavioral correspondence between the two groups has been noted in eye movement impairments (Levin 1984a; Katsanis and Iacono 1991) and in such symptoms as decreased motor behavior and blunted affect (Levin 1984a). Furthermore, there is some evidence suggesting physiological dysfunction of the frontal lobes in schizophrenia (Berman et al. 1986; Weinberger et al. 1986; Buchsbaum 1990). As yet, however, the association between frontal lobe functioning and affective flattening in schizophrenia has not been studied.

The present investigation represents one aspect of an ongoing research program concerning neuropsychological, perceptual, expressive, and psychophysiological aspects of emotion in schizophrenia. In earlier reports we have presented findings regarding deficits in neuropsychological functioning (Blanchard and Neale 1994) and in the perception (Kerr and Neale 1993) and expression (Kring et al. 1993) of affect in a population of unmedicated schizophrenia patients. In this report, we address the neuropsychological correlates of affective expression in schizophrenia. Specifically, in this study we evaluated the hypothesis that neuropsychological indices of right hemisphere and frontal lobe dysfunction are related to deficits in affective expression in schizophrenia. The advantages of the present study are that unmedicated schizophrenia patients were studied, relevant demographic characteristics were controlled, and the neuropsychological evaluation allowed for the assessment of performance thought to be related to the left and right hemispheres as well as the frontal lobes.

In testing hypotheses about neuropsychological substrates of emotional expression, it is important to recognize that the nature of the eliciting stimulus may affect the type of expression elicited (e.g., Ekman et al. 1981) as well as the differential involvement of various brain structures and the extent to which lateralized processes may be engaged (Rinn 1984). Specifically, expressions occurring in an interpersonal context (e.g., in an interview) may be more social than emotional in nature and may be influenced by certain display rules (e.g., Ekman et al. 1982). In contrast, a nonsocial affect-eliciting stimulus (such as a film) may result in expressions that are more likely to be emotional and spontaneous (Ekman et al. 1981). Thus, to evaluate whether the association between neuropsychological functioning and expressivity was influenced by the nature of the eliciting stimuli, we assessed expressivity in two different contexts—during an interview and while the subjects were viewing affect-eliciting films.

Method

Subjects. Twenty-eight male schizophrenia inpatients were recruited from the biological research wards of the Mount Sinai Medical Center, the Bronx Veterans Administration Hospital, and the Pilgrim State Psychiatric Center. Patients were tested no earlier than the third week of a medication washout period that was conducted as part of these patients' participation in pharmacologic studies.

All patients were diagnosed with schizophrenia according to DSM-III-R (American Psychiatric Association 1987). For 26 patients, diagnoses were obtained after two raters concurrently conducted a structured clinical interview with the Schedule for Affective Disorders and Schizophrenia (SADS; Endicott and Spitzer 1978). On the basis of information obtained in the interview and from chart review, independent diagnoses were assigned by each rater. These raters were trained by the Spitzer group and have achieved a high rate of agreement in previous studies (Keefe et al. 1987, 1988; Harvey et al. 1991). Final diagnoses were then determined in a consensus conference; only subjects for whom a consensus diagnosis of schizophrenia was assigned were included. For two patients for whom SADS interviews were not available, diagnoses were determined by chart review conducted by a senior investigator (J.M.N.); both patients had a history of multiple hospitalizations with clear schizophrenic symptomatology. The mean number of previous hospitalizations for schizophrenia subjects was 8.38, standard deviation (SD) = 6.86.

Fifteen normal males were recruited from the civil service staff at Pilgrim State Psychiatric Center. Participants in the normal group were excluded if they or any of their first-degree relatives had ever been treated for psychopathology. T tests did not reveal any significant group differences in age (for schizophrenia subjects, mean = 39.04, SD = 9.68; for normal subjects, mean = 37.93, SD = 14.93) or education (for schizophrenia subjects, mean = 12.07, SD = 2.49, and for normal subjects, mean = 12.67, SD = 1.59). Handedness was
assessed with an abbreviated handedness questionnaire based on Oldfield's (1971) Edinburgh Inventory (Bryden 1977). Left-handed or ambidexterous subjects were excluded because of the difficulty of interpreting neuropsychological performance in these individuals (e.g., Filskov and Catanese 1986). Subjects with a history of head trauma or known neurological disease (including tardive dyskinesia) were excluded.

Procedure.

Neuropsychological testing. Details regarding the procedures of test administration, descriptions of tests, test reliability, and performance differences between schizophrenia and control subjects have been presented previously (Blanchard and Neale 1994). Briefly, neuropsychological tests were selected that are thought to reflect the functioning of the right and left hemispheres and the frontal lobes. Administration and scoring procedures followed published guidelines available for each test. Neuropsychological tests were administered and scored by persons who were blind to ratings of flattened affect and facial expressivity.

Functions evaluated that are thought to be subserved by the right hemisphere included perception of faces, visuospatial perception and orientation, nonverbal memory, and contralateral (left side) somesthetic perception and motor speed. The specific tests included the Facial Recognition test (Benton et al. 1983); Judgment of Line Orientation (Benton et al. 1983); Visual Reproduction subtest of Russell's (1975) revision of the Wechsler Memory Scale (WMS; Wechsler 1945); Tactile Form Perception (Benton et al. 1983), left hand; and the Purdue Pegboard (Lafayette Instrument Company, model 32020), left hand.

Evaluation of left hemisphere functioning included measures of verbal memory, auditory discrimination and comprehension, and contralateral (right side) somesthetic perception and motor speed. The battery included the Logical Memory subtest of Russell's (1975) revision of the WMS; an abbreviated (Golden and Anderson 1977) Speech Sounds Perception Test (Halstead 1947); the shorted Token Test (DeRenzi and Faglioni 1978); Tactile Form Perception, right hand; and the Purdue Pegboard, right hand.

Finally, frontal lobe functioning was assessed with the Wisconsin Card Sorting Test (WCST; Berg 1948; Grant and Berg 1948) and the Controlled Word Association Test (CWAT; Benton and Hamsher 1976). The WCST has been described as a measure of deficits in abstraction, concept formation, or ability to shift set that are often associated with frontal lesions. The CWAT evaluates verbal fluency, which has also been observed to be impaired in cases of frontal lobe dysfunction.

Flat affect ratings. Flat affect ratings were based on the last 5 minutes of a videotaped structured interview that centered on patients' treatment and hospitalization history or control subjects' employment history. The interview was intended to elicit naturalistic emotional responses in an interpersonal context for later coding by two raters using a modified version of the Affective Flattening or Blunting subscale of the Scale for the Assessment of Negative Symptoms (SANS; Andreasen 1984). Of the original eight items in this subscale, three were eliminated. The global rating of blunting is redundant with the remaining items: the subjective rating of the patient is not a measure of expressivity; and inappropriate affect is conceptually not part of the flat affect construct. Thus, the flat affect rating in this study was based on the following five items: unchanging facial expression, paucity of expressive gestures, poor eye contact, affective nonresponsivity, and lack of vocal inflections. Each item was rated on a Likert scale from 0 (not at all) to 5 (severe), resulting in a possible range of flat affect scores from 0 to 25.

Film viewing task. Full details regarding the procedure are reported elsewhere (Kring et al. 1993). Briefly, participants were tested individually in a session lasting approximately 1.5 to 2 hours, during which they were videotaped while viewing four different film clips (three emotional and one neutral) that ranged in length from 264 to 350 seconds. The emotional film clips were excerpts taken from contemporary movies (e.g., "Stripes") and represented three emotion domains: sad, fear/disgust, and happy. Pilot testing had previously determined that each film elicited more of its intended affect than any other affect. The neutral clip depicted different nature scenes. Because hospital regulations did not allow for completely unobtrusive videotaping, we were unable to conceal the camera. However, the videotaping procedure was made as unobtrusive as possible by covering external lights on the camera and by covertly beginning the recording with a remote control switch. Between film clips, participants completed different neuropsychological tests.

Coding facial expression. Par-
Participants’ videotapes were coded according to the Facial Expression Coding System (FACES; Kring and Sloan 1991). In FACES, an expression is defined as any change from a neutral facial display to a nonneutral display and back. When an expression is detected, coders rate its valence (positive or negative), intensity (using a 4-point Likert scale on which 1 = low and 4 = very high), and duration (in seconds). Thus, for each film, the number (frequency), mean intensity, and mean duration of both positive and negative expressions were recorded.

Coding of the patients’ videotapes was conducted by two undergraduates, and coding of the normal subjects’ videotapes was conducted by one of these undergraduates and a graduate student.1 Adherence checks were made periodically throughout the study to ensure that coders were remaining consistent. Coders were blind to the hypotheses of the study and to the nature and names of the films. In addition, they were unaware of whether the videotaped participants were patients or normal control subjects; however, they may have been able to discriminate between the two groups in some instances owing to variables that were difficult to control (e.g., some patients wore hospital gowns).

Results

Interrater Agreement.

Flat affect ratings. Where pairs of raters differed by more than one point on an item, they reached a consensus score by reviewing the videotape and discussing that item’s rating. Before consensus scores were assigned, flat affect ratings between the pairs of raters demonstrated adequate interrater agreement. The case 2 formula recommended by Shrout and Fleiss (1979) was used to compute agreement for pairs of raters separately for patients and normal subjects. For patients, the mean intraclass correlation coefficient (ICC) across pairs of raters was 0.81; for normal subjects, the mean ICC across pairs of raters was 0.89. Each participant’s total flat affect score is based on the mean of the two raters’ judgments.

FACES ratings. Intraclass correlations were computed for pairs of raters across all variables, following the recommendations of Shrout and Fleiss (1979) (ICC [2, 1]). For the patients, these correlations ranged from 0.65 to 0.99, with an average of 0.95 (based on r to z’ transformation). For the normal subjects, the correlations ranged from 0.55 to 0.98, with an average of 0.84. The low correlations reflect low variance, particularly for the neutral film clip. That is, very few participants exhibited facial expressions during the neutral clip.

Data Reduction.

FACES composite variables. As in previous studies using FACES (Kring et al. 1993, in press), composite variables comprising the frequency, intensity, and duration variables were computed. Z scores were computed because the FACES variables are measured in different units (i.e., duration is measured in seconds, intensity is measured by a Likert score, and frequency is a count of the number of expressions), and these standardized scores were added to form positive and negative expression composites for each emotion domain (sad, fear/disgust, happy). For example, the positive expression composite for the happy film was formed by summing the standardized positive frequency, positive intensity, and positive duration ratings for that film. We have previously reported the significant intercorrelations between the components that constitute the composite scores (see Kring et al. 1993). To further reduce the number of variables examined, a single facial expression score was chosen for each of the three emotional films.2 Each expression score was selected on the basis of the valence of the film content and previous findings indicating that these scores differentiated schizophrenia patients from normal subjects (Kring et al. 1993). Thus, the negative expression composite was used for the sad and fear/disgust films and the positive expression composite was used for the happy film.

Composite neuropsychological scores. Before composites could be created, it was necessary to address the issue of missing data for some of the tests that were to constitute the composite indices.

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1Because only one of the raters coded all participants’ videotapes, differences due to raters must be considered. Analyses including the FACES variables have been conducted with the single rater, and the results do not differ from those obtained using average scores of two raters (see Kring et al. 1993, for a fuller explanation). For the present study, average scores across both raters were used.

2Because the opportunity to express emotion is thought to be greatest during the emotional films, expressions during the neutral films were not included in these analyses. In addition, both patients and normal subjects displayed very few expressions during the neutral films (Kring et al. 1993).
Five neuropsychological test scores (three right hemisphere and two left hemisphere tests) were missing (1.2% of the data) owing to errors in administration or inability or unwillingness of a patient to participate in the task. So that these subjects could be retained in analyses, missing data were replaced with means from the group of schizophrenia subjects.

To address escalating experimentwise type I error rate when using multiple independent variables, we created composite scores of the right and left hemisphere test sets before conducting correlational analyses (after Cohen and Cohen 1983, pp. 169–170). Thus, the right hemisphere composite comprised six test scores (immediate and delayed recall scores of the WMS-Figural; Judgment of Line Orientation; Facial Recognition; Purdue Pegboard, left hand; and Tactile Form Perception, left hand) and the left hemisphere composite comprised six test scores (immediate and delayed recall scores of the WMS-Logical; Speech Sounds Perception; Token Test; Purdue Pegboard, right hand; and Tactile Form Perception, right hand). A composite score for the frontal lobe tests was not created, as there were only two of these tests (the WCST, perseverative errors, and the CWAT) and these tests correlated only moderately ($r = 0.31$, NS). Composite scores were formed by averaging the Z-scored test scores. Reliability analyses of the sets of standardized test scores for the schizophrenia subjects indicated adequate internal consistency for the use of composite scores. For the set of right hemisphere tests, coefficient alpha = 0.81; for the set of left hemisphere tests, alpha = 0.83.

Data analysis proceeded in two phases. First, differences between control and schizophrenia subjects on the measures of affective expressivity were evaluated to establish the extent of expressive deficits present in this sample of schizophrenia patients. Second, subsequent analyses focused on schizophrenia patients to evaluate the hypothesis that neuropsychological dysfunction is related to deficits in affective expression.

**Affective Expression in Normal and Schizophrenia Subjects.** Ratings from the SANS and FACES are presented in table 1. Note that, owing to equipment failure or experimenter error, 8 of the 28 schizophrenia subjects were missing one of the measures of affective expression. (SANS flat affect ratings were not available for 4 schizophrenia subjects and 4 others were missing the FACES ratings.) As assessed by the modified SANS flat affect ratings, schizophrenia subjects had significantly greater affective flattening than normal control subjects ($t(37) = 4.35, p < 0.001$). To examine group differences in facial expressivity while watching the films, a multivariate analysis of variance (MANOVA) was conducted on the FACES ratings with group as the between-subjects factor and film as the within-subjects factor. The multivariate effect for group was significant ($F = 11.21, df = 1,37, p < 0.003$), but the effect for film and the group × film interaction were not ($F_s = 0.03$ and 0.58, respectively, $df = 2,36$, NS).

The relationship between ratings of flattened affect and ratings of expressivity during the films was evaluated with Pearson correlations. The SANS ratings of flattened affect were not significantly correlated with any of the FACES ratings for schizophrenia subjects (median $r = -0.15$, all $ps > 0.05$) or for control subjects (median $r = -0.20$, all $ps > 0.05$). The lack of association between these measures of expressivity probably reflects both the context in which these ratings were obtained and the domain of behavior sampled. The SANS ratings are based on an interpersonal interaction and can be expected to reflect both emotional and social expression, whereas the FACES ratings reflect spontaneous emotional expressions made during exposure to the affect-eliciting films. (See Ekman et al. 1981 for a

### Table 1. SANS flat affect and FACES ratings for schizophrenia and control subjects

<table>
<thead>
<tr>
<th>Rating</th>
<th>Schizophrenia subjects</th>
<th>Control subjects</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
</tr>
<tr>
<td>SANS flat affect</td>
<td>9.60 (4.37)</td>
<td>3.97 (3.09)</td>
</tr>
<tr>
<td>FACES, sad film</td>
<td>-0.45 (2.59)</td>
<td>0.73 (2.91)</td>
</tr>
<tr>
<td>FACES, fear/disgust film</td>
<td>-0.81 (1.61)</td>
<td>1.30 (3.44)</td>
</tr>
<tr>
<td>FACES, happy film</td>
<td>-0.90 (2.43)</td>
<td>1.44 (2.18)</td>
</tr>
</tbody>
</table>

*Note.—SANS = Scale for the Assessment of Negative Symptoms (Andreasen 1984); FACES = Facial Expression Coding System (King and Sloan 1991). (FACES scores for the sad and fear/disgust films are standardized negative expression composites; the FACES score for the happy film is the standardized positive expression composite.) SD = standard deviation.*
Finally, the SANS ratings are based on occurrences of facial expression, vocal intonation, gestures, and eye contact, whereas the FACES ratings are based exclusively on facial expression.

Demographics and Neuropsychological Performance in Schizophrenia Subjects. The intercorrelations of demographic and neuropsychological variables are presented in Table 2. Age was significantly negatively correlated with performance on the right and left hemisphere composites; older patients had poorer performance on both of these indices. Neuropsychological scores were moderately to highly intercorrelated. Most striking is the high correlation between the right and left hemisphere composite scores. Clearly, the attempt to create indices that were "predominantly" right or left hemispheric was compromised by the substantial shared variance (64%) between these two scores. Even after the effects of age were partialled out, the two residualized hemispheric composites remained highly correlated ($r = 0.82, p < 0.001$). The frontal lobe measures were also somewhat correlated with the two hemispheric composites. The CWAT was significantly correlated with the left hemisphere composite ($r = 0.46, p < 0.05$) and was moderately, though nonsignificantly, correlated with the right hemisphere composite ($r = 0.37$). Performance on the WCST also showed some association with the two hemispheric composites, although again these correlations did not attain significance ($rs = -0.35$).

Table 2. Intercorrelations between demographic and neuropsychological variables in schizophrenia subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Age</th>
<th>Education</th>
<th>Right composite</th>
<th>Left composite</th>
<th>CWAT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education</td>
<td>0.32</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right composite</td>
<td>-0.58&lt;sup&gt;1&lt;/sup&gt;</td>
<td>-0.13</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left composite</td>
<td>-0.58&lt;sup&gt;1&lt;/sup&gt;</td>
<td>-0.15</td>
<td>0.88&lt;sup&gt;2&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CWAT</td>
<td>-0.14</td>
<td>-0.21</td>
<td>0.37</td>
<td>0.46&lt;sup&gt;3&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>WCST</td>
<td>0.16</td>
<td>0.12</td>
<td>-0.35</td>
<td>-0.35</td>
<td>-0.13</td>
</tr>
</tbody>
</table>

Note.—Right composite = composite index of right hemisphere neuropsychological tests; left composite = composite index of left hemisphere neuropsychological tests; CWAT = Controlled Word Association Test (Benton and Hamsher 1978); WCST = Wisconsin Card Sorting Test (Heaton 1981), perseverative errors. Higher scores on the right composite, left composite, and CWAT indicate better performance, whereas higher scores on the WCST indicate poorer performance (more errors)

<sup>1</sup> $p < 0.005$ (two-tailed).
<sup>2</sup> $p < 0.001$ (two-tailed).
<sup>3</sup> $p < 0.05$ (two-tailed).
Table 3. Zero-order correlations between demographic and neuropsychological variables and measures of affective expressivity in schizophrenia subjects (n = 28)

<table>
<thead>
<tr>
<th>Predictor</th>
<th>SANS flat affect (interview; n = 24)</th>
<th>FACES (film; n = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Sad</td>
</tr>
<tr>
<td>Age</td>
<td>0.17</td>
<td>0.06</td>
</tr>
<tr>
<td>Education</td>
<td>0.08</td>
<td>-0.64²</td>
</tr>
<tr>
<td>Right composite</td>
<td>0.06</td>
<td>-0.12</td>
</tr>
<tr>
<td>Left composite</td>
<td>-0.02</td>
<td>-0.13</td>
</tr>
<tr>
<td>CWAT</td>
<td>-0.01</td>
<td>0.00</td>
</tr>
<tr>
<td>WCST</td>
<td>-0.02</td>
<td>-0.03</td>
</tr>
</tbody>
</table>

Note.—SANS = Scale for the Assessment of Negative Symptoms (Andreasen 1984); FACES = Facial Expression Coding System (Kring and Sloan 1991). (FACES scores for the sad and fear/disgust films are the standardized negative expression composites; the FACES score for the happy film is the standardized positive expression composite.) Right composite = composite index of right hemisphere neuropsychological tests; left composite = composite index of left hemisphere neuropsychological tests; CWAT = Controlled Word Association Test (Benton and Hamsher 1976); WCST = Wisconsin Card Sorting Test (Heaton 1981), perseverative errors. Higher scores on the right composite, left composite, and CWAT indicate better performance, whereas higher scores on the WCST indicate poorer performance (more errors).

¹ p < 0.05 (two-tailed).
² p < 0.01 (two-tailed).

less expressive during the sad film. Neuropsychological performance was uncorrelated with the SANS ratings of flat affect and expression during the sad film. The CWAT was negatively correlated with expression during the fear/disgust film, indicating that better performance on the CWAT was associated with less expression during this film. Finally, better performance on the left hemisphere composite was significantly correlated with greater expression during the happy film. However, the correlation between the left hemisphere composite and expression during the happy film was only slightly larger than that for the right hemisphere composite (rs = 0.41 and 0.38, respectively). A comparison of the two correlations (Steiger 1980, equation 7) indicated that they were not significantly different (t = 0.31, NS). This similarity suggests that the left and right hemisphere composite scores were comparably associated with expression during the happy film.

To determine whether chronicity was related to the neuropsychological or expressivity measures (i.e., the modified SANS and FACES ratings), we computed Pearson correlations between these variables and the number of prior hospitalizations. The number of prior hospitalizations was not significantly correlated with either neuropsychological performance (median r = -0.22, all ps > 0.05) or measures of expressivity (median r = 0.02, all ps > 0.05).

Although the results of the zero-order correlations suggest a failure to find an association between right hemisphere or frontal functioning and expressivity, it may be informative to determine whether such a relationship emerges after possible confounding variables, such as demographic characteristics, are controlled. Age was significantly negatively correlated with both hemispheric composites, and age and education were significantly correlated with expressivity during the happy and sad films, respectively. Thus, partial correlations were computed between the neuropsychological variables and the measures of expressivity, with age and education controlled. The results of these partial correlations generally indicated a diminution in the magnitude of the relationships between the neuropsychological and expressive measures; the only significant correlation obtained was that between the CWAT and expressivity during the happy film (r = -0.45, p < 0.05). Consistent with the zero-order correlations, these results fail to support the hypothesized relationship between right hemisphere or frontal lobe impairment and deficits in affective expression.

Discussion

In this study we sought to evaluate the relationship between neuropsychological functioning, particularly that of the right hemisphere and frontal lobes, and deficits in affective expression in schizophrenia. The results were not consistent with the hypotheses that right hemisphere or frontal lobe dysfunction would be related to greater affective flattening on the SANS or less expressivity during viewing of affect-eliciting film clips.

In no analysis was an association found between ratings of af-
ffective flattening on the modified SANS and any index of neuropsychological functioning. For ratings of facial expressivity during the films, zero-order correlations indicated that the hemispheric composites were related to expression only during the happy film clip, for which greater facial expressivity was related to better performance on both the right and left hemispheric composites. Zero-order correlations also indicated that the measures of frontal lobe functioning were significantly correlated with expression only during the fear/disgust film: better performance on these tasks was actually related to less expressivity. Finally, partial correlations with age and education controlled also failed to support the neuropsychological hypotheses.

This study fails to replicate the findings of Mayer et al. (1985), which suggested an association between right hemisphere dysfunction and flat affect. It is unlikely that the null findings of the present study are attributable to a lack of neuropsychological impairment in these schizophrenia patients, since there is marked neuropsychological dysfunction in these patients compared with control subjects (Blanchard and Neale 1994). Furthermore, the expressivity measures were reliable, had reasonable variability, and successfully discriminated schizophrenia patients from control subjects, indicating that patients had markedly diminished affective expression. Thus, despite the presence of neuropsychological dysfunction and deficits in emotional expression, there was no evidence that impaired performance on right hemisphere or frontal lobe tasks was related to measures of decreased emotional expression.

In accounting for the null findings of this study versus those obtained by Mayer et al. (1985), it may be relevant to consider subject characteristics. Although both studies were based on samples of chronic schizophrenia patients, the gender composition of these samples did differ: Mayer et al.'s sample consisted of 50 percent females, whereas in the present study the sample was exclusively male. However, no gender differences in ratings of flat affect or neuropsychological performance were found by Mayer (see Mayer 1983). Other factors discussed previously may also be relevant, since the medicated status of patients and demographic variables such as age, education, and handedness introduce difficulties in interpreting the findings obtained by Mayer et al. (1985).

The present findings indicating a lack of association between the SANS affective flattening rating and the ratings of facial expresivity during the films raise questions about the extent to which the SANS is actually measuring an affective phenomenon. Given the interpersonal context within which the SANS ratings were made, expression during this interview may have had more to do with an individual's adoption of social norms of communication that regulate emotional displays than with actual emotional experience (Halberstadt 1991). Dworkin (1992) recently presented an alternative view of the SANS affective flattening rating not as a measure of an affective deficit, but as an index of social skill or interpersonal competence: "[I]t is just as plausible that a patient has, for example, poor eye contact or a lack of vocal inflections because the patient suffers from a social deficit and lacks the ability to engage in the socially appropriate behaviors that constitute competent interpersonal interaction" (p. 60). The present results lend some preliminary support for viewing the SANS rating of affective flattening as a rating of expressiveness that has multiple determinants (Mayer et al. 1985) and may include variables that are more related to social-interpersonal factors than to affect. (See Gallaher 1992 for a related discussion of the relationship between individual differences in expressiveness and social vs. emotional traits.)

A review of this study's findings also raises broader issues relating to the methods of testing hypotheses involving lateralized dysfunction in schizophrenia and the status of neuropsychological models of emotional expression. First, neuropsychological methods may be less than ideal for testing hemispheric models of psychopathology. A number of studies have found that the most replicable pattern of neuropsychological performance in schizophrenia is that of generalized dysfunction (e.g., Chelune et al. 1979; Taylor and Abrams 1984; Goldberg et al. 1990; Braff et al. 1991; Hoff et al. 1992; Blanchard and Neale 1994). Used in such a context of generalized cognitive impairment, neuropsychological measures may not be capable of discriminating performance dimensions other than general ability. Furthermore, the high intercorrelation of some tasks, as evidenced in the correlation between composite indices of left and right hemisphere functioning in the present study, is problematic. This substantial shared variance between measures raises questions about the discriminant validity of tasks and contributes to colinearity in regression analyses (as
conducted by Mayer et al. 1985) that can result in spurious findings or obscure genuine relationships (Pedhazur 1982; Cohen and Cohen 1983).

With regard to the application of neuropsychological models to understanding deficits of emotional expression in schizophrenia, it is important to acknowledge the limitations of these models. Although evidence reviewed previously suggests the role of the right hemisphere in emotional facial expression, there has also been an accumulation of contradictory findings. For example, coding of facial muscle movements in normal subjects indicated that stronger asymmetries on the left side of the face, which would suggest greater right hemisphere involvement, did not occur consistently in spontaneous emotional expressions but were restricted to deliberate, non-emotional expressions (Hager and Ekman 1985). Furthermore, in studies of neurologic patients, investigators have sometimes failed to find differences in facial expression between subjects damaged in the left and right hemispheres. This has been true in both posed (Caltagirone et al. 1989) and spontaneous expression conditions (Mammucari et al. 1988). The lack of consistency in results is in part attributable to the variability of lesion location, lesion etiology, and posed versus spontaneous expression and to the variety of eliciting stimuli and systems used to rate expressivity in these studies. (See Zoccolotti et al. 1990 for a fuller discussion of some of these methodologic issues.) As Borod (1992) has concluded, additional research using larger samples of neurologic patients, using patients with more focal lesions, and assessing a larger range of emotions is required to address the inconsistent findings in the literature. Thus, it may be premature to adopt extant hemispheric models of emotional expression to explain the affective deficits of schizophrenia.

In addition to the concerns about the experimental evidence for the right hemispheric model of expression, it should be noted that neuropsychological models of emotional experience have evolved in complexity beyond the simple right-versus-left dichotomy. There is some evidence suggesting that the experience of positive (approach) and negative (avoidance) affects may be differentially lateralized. (See Davidson 1992 for a review.) In this model the experience of positive affect is related to activation of the left anterior region, while the experience of negative affect is associated with activation of the right anterior region (Davidson et al. 1990; Ekman et al. 1990; Tomarken et al. 1990, 1992). To the extent that the expressions under study reflect emotional experience, evaluations of neuropsychological hypotheses of schizophrenia patients' expressive deficits will need to consider anterior cerebral asymmetry as well as valence of the emotion studied. Unfortunately, neuropsychological tasks probably lack the sensitivity to discriminate right from left frontal dysfunction, particularly in patients without frank cortical lesions (as in the study of psychopathology). For example, the most widely used putative frontal task, the WCST, has been shown to be sensitive to both left and right anterior lesions as well as diffuse lesions (Milner 1963; Robinson et al. 1980; Heaton 1981; Anderson et al. 1991). Perhaps more direct measures of cortical functioning (e.g., electroencephalographic asymmetry; Davidson 1988) should be considered in future studies of emotional deficits in schizophrenia.

In summary, in this neuropsychological study of unmedicated schizophrenia patients, we failed to find a differential relationship between impairment on tasks purported to be sensitive to right hemisphere and frontal lobe functioning and deficits in emotional expression. Research on affective deficits in schizophrenia should take care to understand the eliciting context of expressivity and should take into consideration both the affective and the social-interpersonal aspects that may contribute to observed expressivity. Future studies should weigh the limitations of adopting neuropsychological methods, such as problems inherent in the study of patients with generalized impairment and the sometimes substantial shared variance of tasks. Finally, the evidence for some neuropsychological models of emotional expression, such as that in which the right hemisphere has a predominant role in all emotional expression, remains preliminary, and other data indicate the need to understand the differential involvement of other cerebral regions in various emotions.

References


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In Memoriam

Dr. Charles Shagass, internationally respected for his research on brain function and mental illness, died October 27, 1993. As a psychiatrist and scientist, he was one of the pioneers in the study of biological causes of mental disorders. As an educator, he was known for his honesty, integrity, and wit. His major contributions are the establishment of a scientific field, the electrophysiology of mental disorder, and his abiding influence in support of a biological approach to psychiatry—an approach that now increasingly dominates the field.

In a career spanning five decades, Dr. Shagass published over 250 scientific papers, and wrote or edited 7 books on biological aspects of psychiatric disorders. As a national and international leader, he held presidencies in the Society of Biological Psychiatry (1974–75), the American Psychopathological Association (1974–75), the National Association of State Mental Health Research Institutes (1979–81), and the World Federation of Societies of Biological Psychiatry (1981–85). He was a Fellow of the American Psychiatric Association, the Collegium Internationale Neuro-Psychopharmacologicum, and the American College of Neuropsychopharmacology. In recognition of his contributions, he was awarded the Samuel Hamilton Award of the American Psychopathological Association (1975) and the Society of Biological Psychiatry Gold Medal (1977).

Dr. Shagass was born in Montreal on May 19, 1920. He attended public secondary and high schools, graduated with first class honors from McGill University in 1940, and went on to earn an M.S. in physiological psychology from the University of Rochester.

His first paper, which was also his first major scientific contribution, reported successful conditioning of the alpha rhythm in humans. It was published in 1941 with Dr. Herbert Jasper, and is a classic still cited by investigators studying neurophysiological processes in learning.

After discharge from the Royal Canadian Air Force, Dr. Shagass returned to McGill, earning an M.D. in 1949. During his sophomore year at McGill Medical School, he met Dr. Robert Malmo who was directing the Psychology Laboratory at the Allan Memorial Institute. Together they carried out a number of psychophysiological studies on psychiatric patients.

Dr. Shagass was impressed with the need for an objective criterion of anxiety that went beyond clinical observations, and this led to the second of his major contributions to biological psychiatry. He decided to titrate brain activity with sodium amytal in order to measure the level of a person's
anxiety in an objective way. This resulted in the development of his sedation threshold technique, which was the first biological test to differentiate neurosis (now called anxiety disorders) from psychosis. When Dr. Shagass was placed in charge of the electro-physiology laboratory at the Allan Memorial Institute, he continued his work on the sedation threshold and began investigating psychiatric correlates of EEG responses to photic stimulation.

By 1954, it became clear that new methods for measuring brain excitability more directly were needed, and Dr. Shagass began to use evoked potentials (EPs) to study cortical excitability cycles in psychiatric disorders. This was the third of Dr. Shagass’s major contributions to biological psychiatry, and one that continues today in laboratories around the world. Central to this electrophysiological research was the assumption that abnormal behavior reflects an altered balance between excitatory and inhibitory brain processes which would also produce altered EPs.

In 1958, Dr. Shagass left McGill and moved to the University of Iowa. At this time, he was awarded a grant from the National Institute of Mental Health, possibly the first for human EP research in psychiatry. At Iowa, he developed an EP averaging system, began measuring cortical excitability cycles in patients, and soon demonstrated differences between normals and psychiatric patients with respect to recovery functions.

Dr. Shagass moved to Philadelphia in 1966 to become Professor of Psychiatry at Temple University and establish the Temple University Psychiatric Electrophysiology Laboratory at Eastern Pennsylvania Psychiatric Institute. He continued his work on electrophysiological predictors of mental illness by conducting large studies which evaluated EPs in psychiatric patients. These studies documented and confirmed systematic differences in the electrical brain activity of psychiatric patients. The program is still ongoing today in several laboratories. During this time, his major collaborators were Drs. Marco Amadeo, Richard Josiassen, Donald Overton, Richard Roemer, and John Straumanis.

Dr. Shagass earned a well-deserved reputation as an excellent clinician and teacher. He always devoted at least half of his time to these activities. His clinical orientation can best be described as a comprehensive and problem-solving approach, adhering to a broadly conceived medical model. This approach attempted to evaluate fully the intrapsychic, biological, interpersonal, social and situational aspects of the patient's difficulties. Dr. Shagass thought, since psychiatry is a medical specialty, the biological approach should regain a dominant position in the discipline because it was concordant with medicine’s emphasis on pragmatic relevance.

Dr. Shagass always believed that his primary public service functions were performed through his work. In addition to his research, teaching, and clinical services, he served for 18 years on review committees for the National Institute of Mental Health.

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