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Deficits in Facial-Affect Recognition and Schizophrenia

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Abstract

It has been widely demonstrated that schizophrenic patients show a broad range of deficits in interpersonal skills. Recently, considerable attention has been focused on the ability of these patients to decode affective cues. This article reviews findings about facial-affect recognition in schizophrenia. While the literature on this topic is extensive, many investigations have suffered from significant methodological shortcomings. Strategies to resolve these shortcomings are presented. Possible relationships between problems of affect recognition and other symptoms that characterize schizophrenia are discussed. Neurological mechanisms of facial-affect recognition are reviewed and related to data on lateralized neurological impairment in schizophrenia. Suggestions for future research emphasize careful consideration of affect-recognition deficits in relation to other parameters of schizophrenia.

Recently, considerable attention has been focused on facial-affect recognition skills among psychiatric patients and, in particular, among schizophrenic patients. It has been suggested that disturbances of affect recognition may be one of the most consistent, and critical, components of the interpersonal difficulties of these patients (Feinberg et al. 1986). The importance of the ability to recognize facial cues of affect has been emphasized in numerous discussions. As early as 1873, Darwin proposed that facial expressions of emotion had evolved in the human species as a basic mechanism for social communication. He commented that facial expressions ". . .reveal the thoughts and intentions of others more truly than do words, which may be falsified" (cited in Anthony 1978). Izard (1971, 1982) and other theorists on emotions universally acknowledge that facial expression is the principal mechanism of emotional expression, serving both expressive and regulative (affecting the interpersonal behavior of others) functions (Charlesworth 1982). Because emotions are intimately connected with the process of social communication, the study of social skill is incomplete without an analysis of receptivity to emotional signals.

Data reported by Izard (1971) and others indicate that the ability to identify facial expressions of emotion is common to all sociocultural groups. Developmental data document that rudimentary affect-recognition skills are present in children as young as 3 years of age, and that by the age of 10, the performance of children is comparable to that of adults (Ekman and Oster 1979). More recent findings have addressed the neurological underpinnings of the ability to recognize affective states. There is some suggestion that facial-identity recognition, facial-affect recognition, and affective-voice-tone recognition are all mediated primarily by the right hemisphere of the brain (Benton 1980; Bradshaw and Nettleton 1981; Ross 1981; Ley and Bryden 1982). Lesions to the right hemisphere have been found to impair recognition of facial affect (e.g., DeKosky et al. 1980; Benowitz et al. 1983; Etcoff 1984*a*, 1984*b*).

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The relatively recent accumulation of data about deficits in facial-affect recognition in schizophrenic patients raises myriad issues relevant to psychosocial factors, treatment, biological abnormalities, and possible lateralization of brain dysfunction in this group. That affect-recognition deficits may result from specific lateralized brain lesions in nonschizophrenic patients makes these deficits in schizophrenic patients a particularly important area of investigation. In light of recent findings suggesting cognitive deficits and organic pathology in the brains of schizophrenic patients, consideration of the etiology of their affect-recognition deficits may shed further light on the complex interrelationships between social dysfunction and physiologically mediated cognitive and information-processing dysfunctions of schizophrenia. One etiological hypothesis is that affect-recognition deficits in schizophrenia (or in a subset of schizophrenic patients) are due to right-hemispheric lesions comparable to those that have been found to impair affect recognition in studies of neurological patients. However, other explanations for these deficits exist. Given the poor premorbid social history and limited social interaction of many schizophrenic patients, affect-recognition deficits in this group may result from poor social learning. Schizophrenic patients may have had only limited opportunity to learn the meaning of particular affective cues; problems in affect recognition among schizophrenic patients may be secondary to their limited attentional abilities; or, finally, schizophrenic patients may exhibit deficits in the perception of only *certain* affect states. In particular, it is likely that schizophrenic patients may have particular difficulty in the perception of negative affect.

Perceptual Organization of Facial Affect: Hemispheric Differences

The evaluation of facial-affect displays requires information-processing abilities, including attention to and decoding of visual (facial) stimuli. The neuropsychological aspects of recognizing facial affect have interested researchers for many years. A substantial number of recent investigations have addressed the nature of neurological mechanisms underlying the recognition of facial affect. How the ability to recognize facial affect relates to the ability to decode other affective cues and to recognize faces has been examined (albeit preliminarily) from both a functional and anatomical perspective. Also, functional deficits that may arise from damage to the neurological mechanisms underlying the recognition of facial affect have been considered.

The predominant hypothesis with regard to hemispheric lateralization of facial-affect recognition is that the nondominant hemisphere is primary for recognizing emotional aspects of stimuli. Findings from investigations of the functional asymmetry of the hemispheres have indicated that (at least in right-handed adults) the right hemisphere is more involved in facial-identity recognition, facial-affect recognition, and affective-voice-tone recognition than is the left hemisphere. As we shall see, even recent data suggesting that there may be some differential hemispheric specialization for perception of different facial-affect states have not altered the conclusion that overall control of affect recognition resides primarily in the right hemisphere.

Two primary experimental methodologies have been used in investigations of lateralization. Both have involved tachistoscopic presentation of affective stimuli (either photographs or drawings of faces) to individual hemifields. One series of investigations has focused on hemispheric differences in perceptions of facial emotions in neurologically normal adults. A second series has considered the performance of adult patients with unilateral damage to either the left or right hemisphere. The results of studies with nonpatients have typically shown a significant left-visual-field (right-hemisphere) advantage for the recognition of facial stimuli (e.g., Landis et al. 1979; Ley and Bryden 1979; Strauss and Moscovitch 1981). Similarly, studies involving neurological patients have revealed that patients with right-hemispheric lesions are typically more impaired than patients with left-hemispheric damage or controls on tasks involving identification and/or recognition of emotion (e.g., DeKosky et al. 1980; Benowitz et al. 1983; Etcoff 1984*a*, 1984*b*).

The results of several recent studies of normal subjects have suggested that the advantage for the right hemisphere in emotional-recognition tasks may not be consistent across different *categories* of emotional stimuli (e.g., stimuli associated with anger versus those associated with happiness). These findings suggest that *both* hemispheres process emotion-related stimuli, but do so for different *types* of affect. Most commonly, the right hemisphere has been implicated in perception of negative affective stimuli, while

the left hemisphere has been associated with positive emotion (e.g., Reuter-Lorenz and Davidson 1981; Natale et al. 1983; Reuter-Lorenz et al. 1983). In contrast, the results of a study by Etcoff (1984*b*) revealed no emotion-specific differences between the affect-recognition performance of subjects damaged in the right hemisphere versus those damaged in the left hemisphere. Rather, subjects damaged in the right hemisphere were found to be more impaired at perceiving emotions, in general, than were subjects damaged in the left hemisphere or nonpatients.

Whether hemispheric specialization for facial emotion is more refined (i.e., shows greater differentiation between hemispheres) in normal than in brain-lesioned individuals remains unresolved, as the results of other investigations with brain-injured patients have been inconclusive (Cicone et al. 1980; Benowitz et al. 1983). Regardless, most recent models proposed to account for possible hemispheric specialization for perception of different affect states assign overall dominance for affect recognition to the right hemisphere. One important factor in this regard is that there are more distinct negative emotions than positive emotions (Ekman et al. 1972). Thus, right-hemispheric specialization for negative emotions may imply a more important role in affect recognition.

Perhaps the most widely accepted model for the recognition of positive versus negative affect proposes interactive inhibition between the hemispheres (Silberman and Weingartner 1986). According to this model, the right hemisphere is responsible for mediating recognition (and display) of negative emotions, and the left hemisphere is responsible for mediating positive emotions. However, ". . .the right hemisphere would retain dominance for controlling the balance between positive and negative affects, thereby controlling overall emotional tone" (Silberman and Weingartner 1986, p. 343). Thus, the right hemisphere continues to be regarded as playing a primary role in affect-recognition abilities and may be especially sensitive to stimuli associated with negative emotional displays.

The specific relationship of facial-affect recognition to facial recognition and auditory-affect recognition, and the relative role of localization within the right hemisphere, is unclear. Data suggest that the evaluation of facial expressions may be dissociated from the ability to recognize individual faces (Suberi and McKeever 1977; Ley and Bryden 1979; Cicone et al. 1980; DeKosky et al. 1980). Results obtained by Benowitz et al. (1983) even suggest that deficits in the ability to recognize facial expressions are separate from the ability to evaluate emotional situations through other nonverbal cues. Right-brain-damaged subjects exhibited a marked inability to evaluate facial expressions, but generally retained their abilities to evaluate emotions presented through other visual (body movements) and auditory channels. Thus, although all three abilities are assumed to be mediated by the right hemisphere, they may be localized to different right-hemispheric structures. Ross (1981) has coined the term "aprosodia" to refer to the disorders of affective language (including comprehension) that follow focal right-hemispheric damage. In a series of case discussions, Ross (1981) has depicted infarctions that were associated with different aprosodic classifications (e.g., global, transcortical, sensory). Depending on the specific location and size of the right-hemispheric lesion, facial-affect recognition, facial recognition, auditory-affect recognition, or some combination of these abilities could be affected.

Finally, Borod et al. (1986) examined the relationship between expression and perception of facial emotion in patients with unilateral cerebrovascular pathology (right-brain-damaged, left-brain-damaged, and right-handed normal males). Right-brain-damaged subjects were significantly impaired, relative to left-brain-damaged and control subjects, in expressing and perceiving emotion. The ability to pose a particular emotion was not related to the ability to identify the emotion for the total group or any of the individual subject groups.

Summary. Much has been learned about right-hemispheric lateralization of function related to emotion during the past decade. A considerable portion of this information has concerned the role of the right hemisphere in the recognition of emotion. Facial-affect recognition has been most thoroughly researched. It is possible to see a range of deficits in facial-affect recognition stemming from right-hemisphere infarctions. The ability to recognize *all* facial-affect displays may be destroyed, or more subtle distortions may result. The findings reported by Borod et al. (1986) and Ross (1981) suggest that affect-recognition and -display abilities may be independently affected depending on the specific lesion involved.

Problems in facial-affect recognition secondary to right-hemispheric impairment are distinct from more generalized impairments in visuospatial skills. Facial-identity recognition and recognition of nonfacial-affect cues are not highly correlated with facial-affect-recognition deficits in patients with right-hemispheric impairment.

Affect Recognition and Schizophrenia: Empirical Findings

Many studies have found that schizophrenic patients have deficits in the ability to recognize facial expressions of affect. The typical methodology has been to show subjects stimulus photographs depicting different emotional expressions and ask them to identify or compare the emotions being displayed. Spiegel et al. (1962) were among the first to use this procedure. They compared the performance of hospitalized schizophrenic patients to that of student volunteers on a measure of ability to categorize facial photographs depicting various affects. They found no differences between groups, but almost every subsequent study has found some type of deficit in facial-affect recognition among schizophrenic patients. Notwithstanding the frequency with which such deficits have been reported, there is no clear consensus about their precise nature or their functional significance. Two unresolved questions warrant special note: (1) Do schizophrenic patients uniformly have difficulties decoding affective states, or are their difficulties restricted to certain categories of affective display? (2) Are such deficits specific to schizophrenia, or do other psychiatric patients have similar problems in affect recognition? As we shall see, one reason for the lack of closure on these questions is the marked variability in methodologies that have been used in different studies.

As noted, most studies have compared the affect-recognition abilities of schizophrenic patients to those of nonpatients. A common flaw has been the use of chronic, long-stay schizophrenic inpatients without inclusion of comparably ill psychiatric control groups. Dougherty et al. (1974) used Izard's (1971) procedure for assessing facial-affect recognition in a study with female State hospital schizophrenic patients and nonpatient controls. An emotion-labeling task and an emotion-recognition task were administered. Schizophrenic patients were significantly less accurate than controls on both tasks. On the recognition task, schizophrenic patients had the greatest difficulty with two categories of negative affect (disgust-contempt and shame-humiliation). However, because schizophrenic patients had been hospitalized for at least 5 years, the result may have been secondary to the effects of institutionalization and associated social isolation. Another shortcoming is that no information was provided about diagnostic procedures. Thus, the schizophrenic sample may not have been comparable to schizophrenic patients diagnosed on the basis of Research Diagnostic Criteria (Spitzer et al. 1975) or *DSM-III* (American Psychiatric Association 1980).

A similar study was conducted by Muzekari and Bates (1977). Subjects were shown a series of photographs depicting four emotions. College students were more accurate than schizophrenic patients on both open-ended and multiple-choice responses to the affect depicted in each photo. A series of χ^2 analyses indicated that nonpatients were more accurate on each of the negative scenes, but not on the happy scenes. Once again, however, the study is flawed by the fact that the patients had been hospitalized for extended periods. Also, the authors did not describe how patients were diagnosed. A subsequent study by Walker et al. (1980) is subject to the same criticism. The performance of schizophrenic patients in a State hospital on an affect-recognition task involving photographs from the Izard set was compared with that by nonpatients. Although no information was provided about length of hospitalization, it is likely to have been relatively long. The finding that nonpatients were significantly more accurate at identifying all of the emotions in the set therefore is not clearly the effect of schizophrenia, per se. Also, once again, no standardized diagnostic measure was administered.

In a better-controlled study, Cutting (1981) compared judgments of emotional expressions by chronic and acute schizophrenic inpatients, depressed inpatients, and outpatients with personality disorders. The task involved judging which of two faces in photographs was the friendlier. Acute schizophrenic patients differed significantly from all three other groups. In a second experiment, acute schizophrenic patients differed from remitted psychotic patients and psychotic depressives in judgments of both friendliness and meanness, but not in judgments of the age of

the faces. Although Cutting used Research Diagnostic Criteria (RDC) to diagnose patients, his criterion for "acute schizophrenia" did not correspond to the RDC. Rather, the term "acute" was defined as a continuous inpatient stay of less than 6 months.

Pilowsky and Bassett (1980) also used psychiatric control groups in a study of response to facial emotions in schizophrenia. Schizophrenic inpatients, hospitalized "neurotic" and alcoholic patients, and nonpatient controls were asked for spontaneous comments about the affect displayed in photographs from the Ekman et al. (1972) series. All patients had been assigned to groups on the basis of clinical diagnoses. No information was provided about duration of hospitalization or illness. Schizophrenic patients were less likely than any of the control groups to remark on the stimulus person's affect and tended to comment on his/her appearance. Also, schizophrenic patients displayed significantly greater verbosity, especially to photographs depicting fear or anger. The relatively uncontrolled affect-recognition methodology and the idiosyncratic outcome measures complicate interpretation of the data and preclude comparison with the results of other investigations.

Zuroff and Colussy (1986) compared schizophrenic patients in a State hospital to hospitalized affective disorder patients and nonpatients. Affective disorder patients were diagnosed as major depression, dysthymic disorder, or adjustment disorder with depressed mood. The subjects, all females, completed Izard's test of emotion recognition. Schizophrenic patients were less accurate on affect recognition than nonpatients, but did not differ from the depressed group. Schizophrenic patients were not differentially accurate in responding to negative, positive, or neutral emotional stimuli. Both patient groups were less accurate than nonpatients when they labeled an emotion as positive or neutral, but not when they labeled it as negative. The authors concluded that deficits in facial-affect recognition are similar in schizophrenic patients and at least some depressive patients:

> It appears unlikely, therefore, that an emotion recognition deficit is an intrinsic part of the psychobiology of schizophrenia; the deficit is interpreted more plausibly as either a general vulnerability factor or as a consequence of social withdrawal. [Zuroff and Colussy 1986, p. 415]

However, diagnoses were derived solely on the basis of chart review, and information about length of hospitalization was not provided. The effects of long-term institutionalization cannot be ruled out as possibly affecting the findings. Presumably, hospitalization for schizophrenic patients in a State hospital would be longer than that for patients with a diagnosis of adjustment disorder. Also, the range of severity of illness across the depressed group was apparently large. In fact, the authors comment that in some of the depressed subjects, the depressive condition was ". . .complicated by social withdrawal" (p. 416). Thus, these findings further highlight the need to include a carefully selected psychiatric control group in studies of the interpersonal skills of schizophrenic patients. Subject groups will show a range of interpersonal abilities, independent of social impairment which may occur as a direct consequence of the illness itself. The premorbid affect-recognition skill and social behavior of socially withdrawn depressed persons may be different from that of persons with depression who exhibit relatively less social withdrawal.

Data from the studies reviewed above are in conflict about whether schizophrenic patients have a specific impairment in the perception of negative affect. While earlier studies (Dougherty et al. 1974; Muzekari and Bates 1977; Pilowsky and Bassett 1980) suggested that at least some schizophrenic patients (i.e., those with a history of long periods of hospitalization) have differential deficits in comparison to nonpatients, Zuroff and Colussy (1986) failed to find such a deficit. Recently, it has been suggested that certain negative affect states may be more difficult to recognize than nonnegative states (Ekman et al. 1972; Zuckerman et al. 1975). Poor performance by schizophrenic patients could, therefore, reflect differential discriminatory power of the particular test stimuli used in different investigations. This is a potentially critical confound (Chapman and Chapman 1973). Also, the earlier studies failed to consider whether there was a differential deficit in facial-affect recognition relative to other abilities (Novic et al. 1984). In considering the results of these earlier studies, Walker et al. (1984) suggest that it is unclear whether the impairment "reflects a generalized deficit in extracting information from facial features, or. . .is specific to the process of visually decoding or labeling emotion" (p. 37). However, it should be emphasized that affect

recognition could be impaired by *any* attentional dysfunction and not just problems in deriving information from facial features.

To evaluate the issue of a differential deficit in affect recognition in schizophrenic patients, long-stay chronic patients (chart diagnosis) were compared to a matched group of nonpatient controls by Novic et al. (1984) on a test of facial-affect recognition using the Izard photographs and a facial-recognition task (Benton and Van Allen 1973). The authors first ensured that the two tests were of comparable discriminatory power and reliability. Schizophrenic patients tended to perform more poorly than controls on the affect-recognition measure. However, this difference was eliminated when facial-identity recognition was included in the analyses as a covariate. It is noteworthy that after the authors had selected photos that were reliable and had good discriminatory power during construction of their affect-recognition measure, they were left with only those photos dealing with negative affect. Novic et al. suggest that "previous findings. . .that chronic schizophrenics perform more poorly than normals on items with negative but not positive affect may merely reflect the more robust discriminatory power of those items" (pp. 535–536).

In a similar study, Walker et al. (1984) compared hospitalized schizophrenic patients (RDC diagnoses) with affective disorder patients (RDC schizoaffective, depressed type and major depressive disorder) and nonpatient controls. No information about duration of illness was provided. Subjects completed a facial-identity discrimination task based on the Benton, an emotion-discrimination task (in which subjects determine whether the emotions depicted in pairs of photos are the same or different), an emotion-labeling task, and a multiple-choice emotion task. Stimuli for the emotion-recognition tasks were taken from the Izard photos. The tasks were pretested to ensure that they were comparable and of adequate discriminative power. There were significant group differences on all three emotion-recognition tasks, but not on the facial-discrimination task. On the emotion-recognition tasks, schizophrenic patients consistently differed from nonpatients. There was also a significant difference between schizophrenic patients and the affective patients on the emotion-discrimination task. Thus, these data indicate a specific deficit in facial-affect cue processing among schizophrenic patients which is not affected by processing of facial identity.

Finally, Feinberg et al. (1986) compared the facial-affect-recognition abilities of hospitalized schizophrenic patients to those of hospitalized patients with major depressive disorder, and nonpatients. Patients were diagnosed using RDC. Four tasks were constructed from the facial-affect photographs of Ekman et al. (1972). Two tasks were designed to investigate facial-identity matching, independent of the emotion expressed (one task presented inverted faces, while in the other task faces were presented right side up). The other two tasks were designed to assess emotion recognition (matching) and emotion labeling, respectively. While depressed patients differed from controls only on the emotion-labeling task, schizophrenic patients showed deficits on all four tasks when compared with controls. Also, schizophrenic patients performed more poorly than depressed patients on the emotion tasks. Feinberg et al. conclude that while schizophrenic patients are impaired on a broader range of facial-perception skills than depressed patients, it is in the area of emotion discrimination and recognition that they show the greatest deficit. This study is unique in the use of carefully controlled stimulus-exposure intervals. For the facial-identity task using inverted faces, each slide was shown for 2 seconds. For the other tasks, an exposure of 500 ms was used. This procedure more closely approximates the brief duration of spontaneous facial expressions.

These recent studies offered significant methodological advances over earlier investigations in their use of psychiatric control groups, attention to the discriminative power of the stimulus materials, and control of the duration of stimulus presentation. However, the number of subjects included in the samples has been small, and there have been shortcomings in subject descriptions, particularly with regard to duration of illness/hospitalization. Given the variability in designs across studies, there has been virtually no replication of findings. No consistent diagnostic specificity has been found across studies that included patient control groups (e.g., alcoholic vs. "neurotic" vs. depressed patients have been recruited as control subjects). Even the stimuli used have varied, and only Feinberg et al. (1986) controlled for duration of stimulus presentation. Another shortcoming is that virtually no attention has been given to the possible impact of medication on facial-affect recognition. Only

those studies by Novic et al. (1984), Walker et al. (1984), and Feinberg et al. (1986) provided information about the medications that patient groups were receiving when tested.

Therefore, it is not surprising that the results are equivocal. While the studies consistently implicate *some* deficits in facial-affect recognition among schizophrenic patients, the precise nature of these deficits remains unclear. Further study and greater methodological control are needed before a cogent picture of the abilities of schizophrenic patients in facial-affect recognition can be derived. Much greater attention must be paid to issues of diagnosis and subcategorization of schizophrenic and psychiatric control samples. As we noted, only one study in this area has addressed the issue of differential facial-affect recognition abilities among subtypes of schizophrenia (Cutting 1981), and this study did not adhere to current empirically derived criteria for subclassification. Other methodological shortcomings must similarly be addressed.

Basic Pathology of Schizophrenia: Relationship to Affect Recognition

An increasing amount of evidence is indicative of brain pathology in at least some schizophrenic patients. However, findings about the localization and extent of the pathology are inconsistent. While some data suggest lateralized dysfunction (e.g., Nasrallah 1986), other findings indicate pathology in lower brain structures serving the cerebrum (e.g., Mirsky 1969).

The nature of brain disease in schizophrenia obviously has important implications for the etiology of affect-recognition problems in these patients. Impairments could stem directly from dysfunction of the right hemisphere, or could be secondary to more generalized disruptions in the ability to sustain attention and concentration. Attentional impairments may stem from pathology in the lower brain or any other point in the perceptual system (Worden 1966). Alternatively, deficits in affect recognition may stem from faulty learning histories and/or the impoverished social lives that typically characterize the lifestyles of these patients (Morrison and Bellack 1981).

Different schizophrenic patients may exhibit similar deficits in facial-affect recognition (topographically) which are mediated by different factors. Different schizophrenic subtypes may be more or less likely to exhibit problems in facial-affect recognition.

Lateralized Brain Impairment in Schizophrenia. There has been considerable discussion of lateralized brain dysfunction in schizophrenia. However, much of this discussion has focused on *left*-hemispheric dysfunction, whereas, as we have noted, affect-recognition ability has been found to be primarily mediated by the right hemisphere.

The relevance of hemispheric asymmetry for psychiatry was first emphasized by Flor-Henry (1969), who reviewed the records of 50 patients with both psychosis and temporal lobe epilepsy. He found that 43 percent of patients with schizophreniform psychoses had left-sided foci, and only 22 percent had right-sided foci. Conversely, 44 percent of patients with manic-depressive psychoses had right-sided foci, and only 22 percent had left-sided foci. These findings prompted numerous subsequent attempts to relate schizophrenia to a left-hemispheric dysfunction and affective disorders to a right-hemispheric dysfunction.

The results of several later studies of psychosis occurring with temporal lobe epilepsy confirmed Flor-Henry's data (e.g., Sherwin 1981), although some reports did not (e.g., Stevens and Hermann 1981). Other findings suggesting left-hemispheric dysfunction in schizophrenia have involved handedness (Nasrallah et al. 1981), skin conductance (Gruzelier and Venables 1974), dichotic listening (Kugler and Caudry 1983), electroencephalography (EEG) (Abrams and Taylor 1979), visual evoked potentials (Connolly et al. 1983), neuropsychological test performance (Silverstein and Meltzer 1983), neuroanatomical asymmetries (Coffman et al. 1984), cerebral blood flow (Gur et al. 1982), positron emission tomography (Widen et al. 1983), and neurochemical asymmetry (Reynolds 1983) (see Nasrallah [1986] for a review of these findings). However, findings in several of these areas (e.g., handedness: Taylor et al. 1977; neuropsychological testing: Flor-Henry and Yeudall 1979; dichotic listening: Hatta et al. 1984; Johnson and Crockett 1982) have also been inconsistent. Also, both right- and left-sided EEG abnormalities have been reported with schizophrenic patients (e.g., Abrams and Taylor 1979). Etevenon et al. (1983) found different patterns of EEG abnormalities among paranoid and residual schizophrenic patients. EEG differences between paranoid schizophrenic and control subjects occurred only on the left, whereas

differences between residual schizophrenic patients and control subjects were primarily on the right. The results of additional investigations have confirmed this difference in schizophrenic subtypes on EEG measures (e.g., Stevens and Livermore 1982).

There is evidence that schizophrenia may be associated with a defect in interhemispheric integration, which in turn may relate to a dysfunction of the right, left, or both hemispheres. Post-mortem studies have revealed conflicting anatomical evidence (e.g., Rosenthal and Bigelow 1972; Nasrallah et al. 1979). One possible explanation is that anatomical abnormalities may vary across subtypes of the disorder and, especially, paranoid versus nonparanoid patients. Whereas differences (thickening) in the corpus callosum have been found in nonparanoid schizophrenic patients in comparison to nonpatients, no such thickening was observed in the brains of paranoid schizophrenic patients (Nasrallah et al. 1979). However, subsequent histological data reported by Nasrallah et al. (1983) have revealed abnormalities in the corpus callosum of paranoid, but not nonparanoid schizophrenic patients.

Several findings suggest that right-hemispheric dysfunction may be associated with schizophrenia. Schweitzer (1982) observed a right-hemispheric deficit for spatial identification among schizophrenic patients in comparison to normal controls. He suggested that this deficit may result in compensatory overactivation of the left hemisphere, in accordance with earlier reported findings (Gur 1978; Schweitzer et al. 1978). Mathew et al. (1981) reported reduced cerebral blood flow for the right hemisphere of schizophrenic patients. However, a subsequent study revealed reduced blood flow to both hemispheres (Mathew et al. 1982). Hartlage and Garber (1976) reported that schizophrenic patients had deficits in spatial reasoning, but not in nonspatial reasoning. No other neuropsychological tests were administered, however, and subsequent investigations using a more comprehensive battery have reported test results suggesting bilateral deficits (e.g., Kolb and Whishaw 1983).

Thus, the data regarding hemispheric dysfunction in schizophrenia are inconsistent. The findings are inadequate to evaluate whether a specific right-hemispheric lesion underlies the affect-recognition deficits of schizophrenic patients in general, or in a subset of these patients. The precise nature of the brain pathologies associated wih schizophrenia, or specific subtypes of schizophrenia, has yet to be specified. Further research is needed to evaluate the role that brain lesions may play in facial-affect-recognition deficits. This research should take into account differences among subtypes of the disorder.

An alternative hypothesis about the potentially deviant lateralization of psychological processes in the brains of schizophrenic patients pertains to hemispheric preference. As opposed to specific identifiable lesions underlying disordered information processing in schizophrenia, different subgroups may exhibit differential preferred information-processing strategies. One of the most promising distinctions in this regard may be that between paranoid and nonparanoid schizophrenia. Magaro (1980; Magaro and Chamrad 1983*a*) has conducted a series of investigations comparing the hemispheric preference of paranoid and nonparanoid schizophrenic patients. In the most recent of these studies, Magaro and Chamrad (1983*b*) observed that paranoid patients exhibited a deficit in processing faces when presented to the left visual field (right hemisphere). Paranoid patients were not deficient in recognizing faces presented to the right visual field. In bilateral presentations, paranoid patients preferred left-hemisphere processing over right-hemisphere processing, and did worse than nonparanoid patients in recognizing faces presented to the left visual field.

> The results over all stimuli or hemispheres strongly demonstrated the operation of a right hemisphere deficit for paranoid schizophrenics that was mainly related to the processing of facial stimuli, and a left hemisphere deficit for nonparanoid schizophrenics that was mainly related to the processing of alphabetical stimuli. These results are in agreement with our hypothesis that paranoids rely more upon left hemisphere processing while nonparanoids rely more upon right hemisphere processing at the expense of left hemisphere processing. [Magaro and Chamrad 1983*b*, p. 1283]

No mention has been made by Magaro and colleagues of the organic pathology that might underlie these differences in hemispheric preference. It has also not yet been determined whether patients with paranoid schizophrenia have deficits in facial-affect recognition, in addition to their problems in facial-identity recognition. While affect-recognition deficits would be theoretically consistent with left-hemisphere

preference, empirical demonstration is needed. Also, several studies using a dichotic listening procedure have shown left-hemispheric overactivation (exaggerated right-ear preference and fewer shifts of attention away from the right ear) in paranoid as compared with nonparanoid patients (Lerner et al. 1977; Gruzelier and Hammond 1978; Nachson 1980).

Thus, lateralized organic pathology, differential hemispheric preference, or some combination of both factors conceivably could be involved in disturbed affect recognition of subgroups of schizophrenic patients. Further research must evaluate these possibilities. The paranoid-nonparanoid distinction may be particularly important in this regard.

Deficit Symptoms, Attentional Impairments, and Affect Recognition. Considerable attention has recently been focused on deficit or "negative" symptoms of schizophrenia. Negative symptoms typically have been indexed by behavioral signs such as flat affect, alogia, avolition, anhedonia-asociality, and attentional impairment (Andreasen 1982; Andreasen and Olsen 1982). More recent reports indicate that proposals of a clear-cut dichotomy on which patients could be sorted by positive or negative symptomatology may be oversimplified; rather, patients may exhibit mixed (concurrent positive and negative) symptomatology (Bilder et al. 1985; Green and Walker 1986).

Nevertheless, findings have implicated a relationship between negative symptoms and neuropsychological deficits on measures of attention and information processing (e.g., Cornblatt et al. 1985). Using a backward-masking task, Green and Walker (1986) demonstrated that negative symptoms predicted the interstimulus interval at which the masked target could be identified among schizophrenic patients. Greater negative symptoms were associated with longer intervals. The authors conclude that "research on perception in schizophrenia may obtain more consistent results by examining perceptual processes as they relate to symptoms" (Green and Walker 1986, p. 185). These findings may have important implications for research on facial-affect recognition in schizophrenia. As suggested by Green and Walker's comments, there may be a relationship between facial-affect recognition, a perceptual process, and negative symptomatology. It is noteworthy that facial-affect recognition during ongoing interpersonal interactions may involve similar demands to those inherent in backward-masking tasks. During an interaction, a stimulus complex of facial-affective cues is displayed by the interpersonal partner. This display may be very brief, may change rapidly, and may be followed by discordant affect displays. Prior displays may be "masked" by subsequent changes in the partner's affect.

While it is clear that further research should consider possible relationships between facial-affect recognition and particular deficit symptoms of schizophrenia (Neale et al. 1985), it should also be noted that negative symptomatology may be heterogeneous. Green and Walker (1985) used the Scale for the Assessment of Negative Symptoms (Andreasen 1982; Andreasen and Olsen 1982) to derive a single negative symptom score for all patients in their study. However, a number of investigators have argued that negative symptoms are not a unitary phenomenon, and that separate summary scores should be derived for different types of deficit symptoms. Pogue-Geile and Harrow (1985) have proposed that deficits in interpersonal behavior such as flat affect and poverty of speech be considered as "Type A" negative symptoms. "Type B" negative symptoms would include deficits in intellect, cognitive functioning, and psychomotor performance in timed tests. They suggest that Type A and B symptoms may differ on numerous dimensions, including prognostic significance. Lewine et al. (1983) similarly distinguish between cognitive-affective negative symptoms and social withdrawal. Such distinctions may have important implications, both for understanding the relationship of negative symptoms to perceptual processes and for treatment. Mayer et al. (1985) reported right-hemispheric dysfunction in a sample of schizophrenic patients with flat affect. A relation between affect-recognition deficits and Type A symptoms could relate to right-hemispheric impairment. Alternatively, a relationship between affect-recognition deficits and Type B symptoms might suggest that impaired attentional processes underlie social-perceptual problems of certain schizophrenic patients. Remediative efforts for patients would differ accordingly.

Clearly, further research is needed to develop a better understanding of deficit symptoms and their relationship to problems in affect recognition. As the assessment methodologies relating to each of these areas continue to be refined, it is likely that this research will generate important findings about the relationship of

affect-recognition deficits to defect states in schizophrenia.

Future Directions

Despite considerable effort, there are many unanswered questions about facial-affect recognition among schizophrenic patients. Issues in particular need of evaluation in future research include: (1) differential abilities in facial-affect recognition among subtypes of schizophrenia; (2) neuroleptic effects on affect-recognition abilities; (3) the validity, functional significance, and stability of facial-affect recognition as typically assessed; (4) methodological factors that may affect findings; and (5) treatment implications.

Differential Abilities in Facial-Affect Recognition Among Subtypes of Schizophrenia. Schizophrenia is a heterogeneous disorder, and different subtypes may exhibit different deficits in the recognition of facial affect. The spectrum of such deficits includes global impairments in the ability to decode facial cues of affect, as well as more focused problems in differentiating between several different positive or negative emotions (e.g., distinguishing anger from contempt). An even more specific deficit might involve the inability to identify one particular affect (e.g., consistently mislabeling anger). Factors that might be associated with deficits in facial-affect recognition include right-hemispheric impairment or atypical patterns of hemispheric preference, attentional impairments, premorbid history, degree of social isolation, or the presence of thought disorder.

Research should be designed to consider variability in schizophrenic subtypes and differential deficits in ability to recognize facial affect. Carefully diagnosed subgroups of schizophrenic patients should be compared on tasks assessing facial-affect recognition, and analyses should consider performance on positive versus negative affect states separately. As we noted, paranoid schizophrenic patients may be a particularly important subgroup to assess in light of Magaro's findings regarding visuospatial deficits in the processing of facial stimuli among paranoid patients. It might also be worthwhile to examine facial-affect recognition in primary relatives of patients with schizophrenia, as well as in the schizophrenia-spectrum disorders.

The relationship of deficits in facial-affect recognition to other aspects of cognitive/information-processing performance should be evaluated. As discussed, facial-affect recognition is assessed by perceptual tasks that require subjects to attend selectively to, and sustain attention to, socially relevant stimuli. These stimuli involve a visual array, which must be processed contextually, and which may involve varying degrees of stimulus clarity or ambiguity. Neuropsychological- and cognitive-testing procedures that provide comparable task demands in selective attention, sustained attention, and perceptual load (but in relationship to nonsocially relevant stimuli) should be included in protocols as control tasks. These additional testing procedures are necessary to evaluate alternative explanations for the affect-recognition deficit. In particular, their use will permit examination of whether problems in affect recognition reflect more generalized deficits in attention. Specific measures or procedures that would be helpful include the Continuous Performance Test (Rosvold et al. 1956), the Rey-Osterrieth Complex Figure Test (Osterrieth 1944), the Bender-Gestalt Test (Bender 1938), visual search and visual attention tasks such as the "oddball" task (Monk 1984), and tests of facial recognition such as those used by Novic et al. (1984) and Walker et al. (1984)—for example, the Test of Facial Recognition (Benton and Van Allen 1973). To date, no study with schizophrenic patients has even used screening procedures to control for or rule out impaired vision.

In addition, the issue of whether problems in recognition of facial affect reflect right-hemispheric dysfunction should be addressed. The presentation of stimuli separately to the right- and left-visual field is one way to address this issue. Also, attempts should be made to correlate deficits in facial-affect recognition to findings from EEG or other (e.g., positron emission tomography, computed tomography, and magnetic resonance imaging) assessments of hemispheric functioning. Referring back to Ross's (1981) discussion of the aprosodias, one might nominate the category of anomic aprosodia (poor comprehension of nonverbal emotional cues but intact prosody and prosodic comprehension) as most resembling deficits in recognizing facial affect. Ross postulates that anomic aprosodia should result from a lesion of the right-angular gyrus. The possibility of this or another right-hemispheric lesion should be carefully evaluated in schizophrenic patients with affect-recognition deficits.

Measures of premorbid functioning may help to consider the role of limited social learning opportunities and social isolation as po-

tential contributing factors. The etiology of affect-recognition deficits in the patient with good premorbid adjustment who exhibits right-hemispheric pathology is likely to be different from that for patients with poor premorbid history and no apparent right-hemispheric lesion. The identification of factors contributing to the development of problems in affect recognition could have important implications for rehabilitative efforts, as we discuss below.

Medication Effects on Facial-Affect Recognition. Few studies have considered the effects of neuroleptics or anticholinergic medications on the ability to perceive facial cues of affect. In fact, only those studies by Novic et al. (1984), Walker et al. (1984), and Feinberg et al. (1986) provided information about the medications that patient groups were receiving at the time of testing. Braff and Saccuzzo (1982) have demonstrated that neuroleptics improve visuospatial processing in schizophrenic subjects. However, Ross (personal communication, 1986) contends that neuroleptics may impair prosody. Impairment of cognitive performance by muscarinic anticholinergic drugs has been demonstrated with a variety of agents using both acute administration in nonpatient volunteers and chronic treatment in patients (cf. Tune et al. 1982; Katz et al. 1985). Perlick et al. (1986) observed that recent memory correlated inversely with serum anticholinergic levels for verbal recall (but not for recognition memory) in chronic schizophrenic inpatients taking neuroleptic medications. The fact that recognition memory did not correlate with anticholinergic blood levels, the authors comment, suggests that the memory deficit may not be a primary memory disorder. Instead, it may be related to other factors that influence recall, such as motivational state and fluctuations in attention. That is, anticholinergic activity may exacerbate deficits in attention and motivation, which may in turn impair memory functions. Regardless of the mechanisms involved, the effect of antipsychotic and anticholinergic medications on social performance abilities, in general, is in need of further study. Analyses of the variance in performance on measures of interpersonal response skills that is attributable to neuroleptic dose should be conducted. Where possible, assessments of unmedicated patients should be made. The stability of social skills deficits over time, as medication is titrated, should be evaluated (see below).

Validity, Functional Significance, and Stability of Deficits in Facial-Affect Recognition. Studies of facial-affect recognition abilities of schizophrenic patients have seemingly been conducted in a vacuum. None of the studies that we reviewed considered the relationship of performance on measures of facial-affect recognition to performance on other measures of interpersonal skill. The ecological validity of the basic methodology for assessing facial-affect recognition in schizophrenia is undermined. The demands that are presented in this methodology obviously differ from those inherent during in vivo interpersonal interactions. The single-channel presentation of cues, along with the uncontrolled duration of cue presentation that has characterized most investigations, is quite different from the rapidly changing, multichannel presentation of cues that occurs beyond the laboratory.

Several possible validational strategies exist. One strategy would be to compare responding on the traditional procedure for assessing affect recognition, in which still photographs are used as stimuli, to performance on measures that present facial-affect cues in a different manner. For example, videotaped scenarios that display facial-affective cues (with or without audio cues) and computer-generated facial images (displayed either statically or dynamically) could be used to estimate the construct validity of the methodology used to assess facial-affect recognition.

More important is criterion validity. The issues here are whether deficits in the ability to recognize facial affect on laboratory measures correlate with problems in the recognition of facial affect during interpersonal interactions and/or with more general interpersonal dysfunction. According to the behavioral model of social skills, social-perception skills are critical for effective interpersonal responding (Morrison and Bellack 1981). Without the ability to "read" relevant interpersonal cues accurately, an individual would be unable to respond in a manner that was consistent with the interpersonal context. Unfortunately, naturalistic assessment strategies are difficult to conduct. Also, the ability to recognize facial affect is not easy to assess in a naturalistic manner, in that the assessment process requires that some sort of probe be administered to the subject following a particular, discrete affective display. Therefore, the assessment procedures would disrupt the natural flow of interpersonal interactions.

Still, a number of alternatives could be implemented to address the validity of deficits in facial-

affect recognition. The relationship between deficits on measures of facial-affect recognition and more general measures of social dysfunction should be considered (even if the more general measures are also laboratory assessment procedures). For example, accuracy in recognizing facial affect could be compared with role-play measures of overall social skill and/or interview ratings of overall social adjustment on the Social Adjustment Scale (Weissman and Bothwell 1976). Also, procedures to assess affect-recognition abilities could be built into the basic protocol for role-play assessment, and performance could be compared to social-skill ratings and the "traditional" measure of facial-affect recognition. Wallace and others have piloted procedures in which patients are questioned during a role-play procedure about salient cues that were given by the role-play confederate (Wallace et al. 1980). The relationship of deficits in the ability to recognize facial affect should also be examined in relationship to other aspects of affect recognition (e.g., auditory or voice-tone recognition), as well as to affective communication skills. Ross (1981) has suggested that deficits in the ability to comprehend emotional prosody often occur in conjunction with impairments in emotional prosody. No studies have carefully considered the relationship between the ability to decode facial affect and the ability to encode facial affect in schizophrenic patients.

Finally, no study has yet assessed the reliability of deficits in the ability to decode facial affect among schizophrenic patients. The stability of deficits in facial-affect recognition is a particularly important issue, as it pertains to the relationship of social disabilities to other deficit symptoms of schizophrenia. Studies should evaluate the stability of facial-affect recognition during acute episodes of the disorder, as well as across periods of relative symptomatic remission. Premorbid abilities in facial-affect recognition should also be evaluated in subjects at risk for schizophrenia.

Methodological Factors

As mentioned, the inconclusiveness of the findings regarding facial-affect recognition deficits among schizophrenic patients may reflect variability in methodologies across studies. Critical factors include (1) stimulus materials, (2) length of stimulus presentation, and (3) specific task demands.

Stimulus Materials. A number of different sets of facial-affect display materials have been developed, but the most commonly used materials are those of Izard (1971) and Ekman et al. (1972). Each of these sets consist of high-quality black-and-white photographs of carefully posed actors and actresses. However, the comparability of the two sets has not been established. Other investigators have used their own stimulus display materials, which range from stick drawings of faces to carefully posed color photographs. For future research, we suggest that investigators use either of the validated sets of stimulus materials. Direct comparisons of the use of these two sets of stimulus photographs with schizophrenic subjects should be conducted to evaluate their differential efficacy in identifying affect-recognition deficits among these patients.

Length of Stimulus Presentation. During in vivo social interactions, affective cues are displayed quite briefly, and different cues may occur in close temporal proximity to one another as the interaction proceeds. Relatively few investigations of facial-affect recognition among schizophrenic patients have attempted to reproduce this brevity of cue display. Most studies have permitted subjects to examine photographs of faces without time constraints. In view of the attentional impairments of many schizophrenic patients, the duration of the display is an especially important variable to be controlled. Any attempt to duplicate naturally occurring short displays of facial-affect cues must be careful not to make the display so short that it confounds the assessment of schizophrenic patients' affect-recognition abilities with their attentional impairments. The duration of stimulus presentation should be carefully selected on the basis of pilot studies with nonaffective stimulus presentations of equivalent complexity.

Affect Matching Versus Affect Labeling. Studies have varied in the specific demands imposed by their methodologies. Some procedures have required the subject to *label* a particular affect being displayed, while others have required the subject to indicate whether two emotions are the "same" or "different." Still others have required the subject to select which of several labels best suits the emotion being displayed. While schizophrenic patients have been found to do poorly in studies using each of these methodologies, it is important to use procedures that maximize resemblance to real-life task demands. As a result, we recommend that investigators *always* include an emotion-labeling condition.

Finally, other more general methodological issues should also be addressed. Perhaps foremost among these is the careful diagnosis and description of subjects involved in both the target and control groups recruited for investigation. Standardized diagnostic interviews should be used to identify subjects, and the reliability of diagnoses should be reported. Investigators should also report such critical descriptive factors as duration of illness and hospitalization, medication and dosage, and sociodemographic factors.

Treatment Implications

Although greater specification of the origins and parameters of deficits in facial-affect recognition among schizophrenic patients remains a top priority, the presence of this deficit among at least some schizophrenic patients should have implications for treatment. As we noted, the failure to "read" nonverbal cues of emotion presumably could contribute to inappropriate social responses. Social skills training programs for schizophrenic patients might focus more on emotion perception. Such training could include practice with the photographs typically used in studies of facial-affect recognition, and progress to participation in modeled social interactions in which emotional cues are salient (Walker et al. 1984). The presence of cognitive deficits that may adversely affect a patient's ability to learn should be carefully evaluated. For those patients with significantly impaired cognitive functioning, affect-recognition training could be modeled after procedures that have been used for the re-training of a variety of skills in the rehabilitation of brain-injured patients (cf. Goldstein and Ruthven 1983).

Conclusion

Affect-recognition deficits have been one of the most consistently noted deficits in the literature regarding the social impairment of schizophrenic patients. Yet, there is still much to be learned about the precise nature of these deficits, and their overall impact on and relationship to other aspects of schizophrenic symptomatology. In this article we have reviewed studies of affect recognition among schizophrenic patients, and have considered the relationship of affect-recognition deficits to the growing literature on lateralized impairments and deficit symptoms of schizophrenic patients. Suggestions for future research emphasize a careful consideration of affect-recognition deficits in relation to other behavioral parameters of schizophrenia. Future investigations in this area will have important implications for the treatment of social impairment in schizophrenia. As more is learned about the relationships among the diverse behavioral dysfunctions that characterize schizophrenic patients, more comprehensive re-training programs can be developed.

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