Affective reactivity of speech and emotional experience in patients with schizophrenia

Alex S. Cohen*, Nancy M. Docherty

Department of Psychology, Kent State University, PO Box 119, Kent, OH 44240, USA

Received 17 September 2002; received in revised form 22 January 2003; accepted 7 February 2003

Available online 22 May 2003

Abstract

Communication disturbances are exacerbated by stress in patients with schizophrenia, a phenomenon known as affective reactivity of speech. However, a subset of these patients, those with the deficit syndrome, is characterized by a diminished capacity to experience emotion. We examined affective reactivity in the natural speech of schizophrenia patients with and without the deficit syndrome, with the expectation that deficit syndrome patients would evidence lower levels of affective reactivity. Two 10-min conversational speech samples were collected from each of 13 deficit syndrome patients, 22 nondeficit patients and 26 healthy control subjects. One speech sample from each participant was on the topic of emotionally negative, stressful memories, and the other was on emotionally positive, nonstressful memories. The audiotaped speech samples were analyzed blindly for frequencies of referential communication failure. All three groups showed significantly higher frequencies of communication disturbances in the emotionally negative speech sample than the positive speech sample. Nondeficit patients showed greater affective reactivity of speech than either deficit patients or controls. Conversely, deficit patients' speech was not more reactive to emotion than the speech of the control group. These results suggest that emotion-related variables mediate the relationship between stress and language symptom exacerbations in at least some patients with schizophrenia.

© 2003 Elsevier B.V. All rights reserved.

Keywords: Language; Exacerbations; Schizophrenia

1. Introduction

Schizophrenia is a heterogeneous disorder with substantial differences among patients in presenting symptoms, pathophysiology and clinical course. One approach used to create more homogeneous groups for empirical study involves subtyping patients based on differences in symptoms and behaviors that are believed to reflect differences in underlying pathological process (e.g., Crow, 1985; Liddle et al., 1989; Kirkpatrick et al., 2001). Some, but not all, patients with schizophrenia demonstrate marked symptom exacerbations in response to arousal of negative affect. This “affective reactivity” of symptoms may reflect a pathophysiological process present in a subgroup of patients (Docherty, 1996). Of particular note, some patients’ speech becomes significantly more disorganized during laboratory stress-induction paradigms (Docherty et al., 1994, 1998; Burbridge and Barch, 2002). There is evidence to suggest that
affective reactivity of speech reflects a distinct process that is present in some schizophrenia patients, and not others. It has been associated with other putative process discriminators, including reactivity of right ear advantage to emotionally laden stimuli in fused word dichotic listening tasks (Rhinewine and Docherty, 2002), higher than normal levels of startle reactivity to acoustic stimuli (Docherty and Grillon, 1995) and greater familiality of illness (Docherty et al., 1998). However, the processes underlying affective reactivity of speech are not well understood. The present study examined whether the presence or absence of the deficit syndrome of schizophrenia, which involves a diminished capacity to experience emotions, was related to patients’ level of affective reactivity of speech.

Nuechterlein and Dawson (1984) have hypothesized that symptom exacerbations in patients with schizophrenia are influenced by certain “mediating vulnerability factors”. In the context of a stress—diathesis model of schizophrenia pathology (for a review, see Fowles, 1992), mediating vulnerability factors may mediate the relationship between external stressors and subsequent exacerbations of psychotic symptoms. Accordingly, patients with higher levels of these mediating vulnerability factors are more likely to relapse under conditions of stress (e.g., Rosenfarb et al., 2000). There is reason to suspect that individual differences in the degree to which patients experience emotions, i.e., their level of emotionality, might be a mediating vulnerability factor. Thus, patients’ level of emotionality might influence the level of affective reactivity of symptoms that they exhibit.

Differences among patients in emotionality have been documented and have been used to help define a distinct syndrome within schizophrenia, denoted the deficit syndrome (Carpenter, 1988). The deficit syndrome is diagnosed based on the presence of two of the following six primary and enduring negative symptoms: flat affect, diminished capacity to experience emotions, poverty of speech, curbing of interests, diminished sense of purpose and diminished social drive (Kirkpatrick et al., 1989). Patients with the deficit syndrome have reported lower levels of stress than schizophrenia patients without the deficit syndrome following a laboratory stress-induction paradigm (Cohen et al., submitted for publication), and have evidenced lower levels of many negative emotions including anxiety, guilt, hostility (Kirkpatrick et al., 1993; Tek et al., 2001) and depression (Kirkpatrick et al., 1993, 1994, 1996). The deficit syndrome also has been associated with a lower severity of certain symptoms that are considered to be relatively “reactive” to external stressors, such as suspiciousness (Kirkpatrick et al., 1996), delusions and hallucinations (Kirkpatrick et al., 1993, 1996; Buchanan et al., 1994; Cohen and Docherty, submitted for publication; but see also Buchanan et al., 1997). In summary, because patients with the deficit syndrome experience lower levels of emotion, they may be less susceptible to symptom exacerbations in response to affectively negative circumstances.

The present study examined whether patient’s capacity to experience emotions was related to the degree to which their speech becomes more disordered under conditions of stress. Three groups, including patients with the deficit syndrome, patients without the deficit syndrome and healthy controls, were compared on measures of communication disturbance in speech samples elicited during separate positive and negative affectively valenced narrative conditions. Affective reactivity of speech was determined by comparing levels of communication disturbance in the two affective conditions. We hypothesized that all three groups would exhibit some degree of affective reactivity of speech, but that patients with the deficit syndrome and control subjects would evidence significantly lower levels of affective reactivity of speech than patients with nondeficit schizophrenia.

2. Method

2.1. Participants

This study was part of a multi-faceted research project investigating language disorder in schizophrenia. Participants who presented histories suggestive of organic impairment were excluded from the study, as were participants with a primary language other than English. Furthermore, participants who met criteria from the Diagnostic and Statistical Manual of Mental Disorders—Fourth Edition (American Psychiatric Association, 1994) for substance abuse or dependence...
also were excluded. All participants received monetary compensation for participation in the present study. The participants’ demographic and descriptive information are presented in Table 1.

2.1.1. Patients

The patient group originally consisted of 51 stable outpatients with DSM-IV schizophrenia. However, 35 of these cases were included in the majority of the analyses used in the present study (see Section 2.2.2 for elaboration). Diagnoses were made by a clinical psychologist with diagnostic expertise (N.M. Docherty) based on information obtained using the Schedule for Affective Disorders and Schizophrenia—Lifetime Version (SADS-L; Endicott and Spitzer, 1978). In order to provide some assurance that participants could comply with the study protocol, participants who had Global Assessment of Functioning (GAF, DSM-III-R, American Psychiatric Association, 1987) scores below 35 were excluded from this study. Of the 35 patients, 8 were being prescribed typical antipsychotic medication, 19 were being prescribed atypical antipsychotics, 4 patients were prescribed both and 4 were not being prescribed any antipsychotics at the time of testing. Ten patients in the present study were being prescribed anticholinergic medications.

2.1.2. Controls

The control group consisted of 26 volunteers who were matched to the patients on age and parents’ socioeconomic status using the Socioeconomic Index (SEI; Hauser and Warren, 1997). Controls were recruited from university support staff and were excluded if they had any history of psychotic symptoms or if they met criteria for an axis I substance abuse or mood disorder based on information obtained from a SADS-L interview.

2.2. Procedure

2.2.1. Symptom rating scales

The Brief Psychiatric Rating Scale (BPRS; Lukoff et al., 1986) was used to measure patients’ symptomatology. Symptom ratings were made by graduate student level researchers who had attained acceptable levels of interrater reliability (all intraclass correlations >0.69, most >0.90).

2.2.2. Deficit syndrome score

Deficit syndrome scores were computed using the Proxy for Deficit Schedule (PDS; Kirkpatrick et al., 1993), a measure that derives deficit scores directly from BPRS ratings. Although PDS scores are derived from cross-sectional symptom ratings, they have been

Table 1

Demographics and clinical variables for deficit (Def), nondeficit (Nondef) and healthy control subjects (Con)

<table>
<thead>
<tr>
<th></th>
<th>Deficit (N=13)</th>
<th>Nondeficit (N=22)</th>
<th>Controls (N=26)</th>
<th>Group comparisons (post hoc = Scheffe)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>34 ± 8</td>
<td>37 ± 11</td>
<td>39 ± 8</td>
<td>Def = Nondef = Con</td>
</tr>
<tr>
<td>Education</td>
<td>12.6 ± 1</td>
<td>12.1 ± 2</td>
<td>14.8 ± 2</td>
<td>Def = Nondef &lt; Con*</td>
</tr>
<tr>
<td>GAF</td>
<td>53 ± 9</td>
<td>49 ± 14</td>
<td>86 ± 5</td>
<td>Def = Nondef &lt; Con*</td>
</tr>
<tr>
<td>Parents SEI</td>
<td>72.7 ± 32.1</td>
<td>54.4 ± 16.6</td>
<td>64.6 ± 26.3</td>
<td>Def = Nondef &lt; Con</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>10</td>
<td>11</td>
<td>12</td>
<td>Def = Nondef = Con</td>
</tr>
<tr>
<td>Female</td>
<td>3</td>
<td>11</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cau.</td>
<td>11</td>
<td>16</td>
<td>21</td>
<td>Def = Nondef = Con</td>
</tr>
<tr>
<td>Afr. – Am.</td>
<td>2</td>
<td>6</td>
<td>4</td>
<td>Def = Nondef = Con</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>IQ</td>
<td>91 ± 15</td>
<td>85 ± 14</td>
<td>105 ± 9</td>
<td>Def = Nondef &lt; Con*</td>
</tr>
<tr>
<td>Psychosis</td>
<td>8.4 ± 5.2</td>
<td>13.7 ± 5.1</td>
<td></td>
<td>Def &lt; Nondef*</td>
</tr>
</tbody>
</table>


*p < 0.05.
found to correspond highly with ratings on the Schedule for Deficit Syndrome (SDS; Kirkpatrick et al., 1989), the ‘gold standard’ measure of the deficit syndrome. In essence, PDS scores are based on two deficit symptoms: blunted affect (measured by the BPRS blunted affect scale ratings) and diminished emotionality (measured by the AFFSCALE score, which is a summation of BPRS depression, anxiety, guilt and hostility scale ratings). The PDS score is computed by subtracting the AFFSCALE score from the blunted affect rating.

Deficit/nondeficit group assignment was determined following the method of Kirkpatrick et al. (1996) using the available cut-points in the present dataset that fell closest to the ideal recommended values. Patients in the highest 25% range of PDS scores (PDS score > \(-4\), \(n = 13\)) were included in the deficit group, while the nondeficit group was composed of all cases in the lowest 43% range of PDS scores (PDS score < \(-6\), \(n = 22\)). The remaining 32% (PDS score = \(-4\), \(-5\), \(n = 16\)) of cases were excluded from the between group analyses.

2.2.3. Positive syndrome scores

Severity of psychosis was measured using the positive syndrome score described by Ventura et al. (2000), which is a summation of five BPRS items: bizarre behavior, disorganization, hallucinatory behavior, suspiciousness and unusual thought content. A psychosis score could not be calculated for one of the nondeficit patients due to missing data. This case was excluded from the analyses examining psychotic symptomatology.

2.2.4. Ratings of affective reactivity

Participants were asked to produce three separate, 10-min-long narratives. During the first narrative, the participants talked about affectively neutral topics (i.e., hobbies, daily routine, etc.). This neutral condition was administered with the sole purpose of reducing the situational stress associated with speaking and being tape-recorded and to familiarize the participants with the procedures. The second and third narratives consisted of the participants talking about affectively valenced memories. In one of the two conditions, participants were asked to recount “nonstressful, good” memories from their lives (the affectively positive condition), and in the other condition, “stressful, bad” memories (the affectively negative condition). Patients were expected to do most of the talking; however, interviewers kept them on task as needed and elicited elaboration of condition-appropriate memories. The positive and negative narratives were collected on separate days to avoid carryover effects, and the order was counterbalanced. The narratives were transcribed and carefully proofread to ensure accuracy.

The transcribed narratives were then analyzed using the Communication Disturbance Index (CDI; Docherty et al., 1994). The CDI is designed to assess disturbances in communication of meaning, rather than thought disorder as such, or disorder of language structure. The CDI measures six conceptually distinct types of referential disturbances, including vague references, confused references, missing information references, ambiguous word meanings, wrong word references and structural unclarities. Instances of each type of disturbance in the speech samples are counted. In order to control for individual differences in verbosity, numbers of instances are then divided by the amount of speech (hundreds of words) in the narrative. Total CDI scores, which were used in the present study, are computed by summing the six referential disturbance scores. The CDI and its validation are described more fully in an earlier publication (Docherty et al., 1994). Affective reactivity of speech was assessed by comparing CDI ratings in the positive vs. negative speech samples.

2.2.5. IQ

Estimated IQ scores were derived from the Shipley Institute of Living scales (Shipley; Zachary et al., 1985). The Shipley-derived IQ score has been found to correlate highly with the Wechsler Adult Intelligent Scale—Revised full scale IQ score (Frisch and Jessop, 1989).

2.2.6. Analyses

The analyses were conducted in two parts. First, a two-way (group \(\times\) condition) analysis of variance (ANOVA) compared the deficit, nondeficit and control groups on CDI ratings in the affectively negative vs. positive conditions. These analyses determined whether (1) participants as a group demonstrated affective reactivity of speech, (2) groups differed on levels of communication disturbances across the two conditions.
conditions and (3) there was a group by condition interaction, with the three groups differing on degree of reactivity of speech. Second, residualized reactivity scores were computed by regressing the CDI ratings in the affectively positive condition from the CDI ratings in the affectively negative condition. A one-way ANOVA was employed to compare the three participant groups on these reactivity scores. This analysis provided a second comparison of the three groups on level of affective reactivity of speech, a comparison that took into account baseline differences in communication disturbances. All results reported are two tailed.

3. Results

Table 1 contains descriptive information for the deficit, nondeficit and control groups. These three groups did not differ in age, male/female ratio or ethnic composition. Patient groups had significantly lower levels of education, IQ and GAF scores than the control group. The deficit and nondeficit groups did not differ on level of education, IQ or GAF, but the deficit syndrome group showed significantly less severe psychotic symptoms $t(32) = 2.94, p < 0.01$ compared to the nondeficit group.

3.1. CDI ratings compared in the deficit, nondeficit and control groups

Table 2 contains the means and standard deviations of the CDI ratings in the affectively positive and negative conditions for the deficit, nondeficit and control groups. These ratings were compared by means of a two-way (group $\times$ condition) repeated measures ANOVA. There was a significant main effect for group, $F(2,57) = 27.62, p < 0.001$, indicating that the three groups differed on total CDI ratings across conditions. There was a significant main effect for condition, $F(1,56) = 58.91, p < 0.001$, indicating that subjects across groups exhibited higher CDI ratings in the affectively negative condition compared to the affectively positive condition. Most importantly, the interaction effect was significant, $F(2,57) = 9.618, p < 0.001$, indicating that there were significant differences among the groups with respect to degree of affective reactivity of speech.

Three paired $t$-tests were then used to compare CDI ratings in the affectively positive and negative conditions for each group separately. The deficit, nondeficit and control groups each evidenced higher CDI ratings in the affectively negative condition ($t(12) = 4.497, p = 0.001$; $t(20) = 5.365, p < 0.001$ and $t(25) = 4.102, p < 0.001$, respectively), indicating that all three groups demonstrated significant levels of affective reactivity of speech. These results are summarized in Fig. 1.

3.2. Affective reactivity of speech compared between the deficit, nondeficit and control groups

Residualized affective reactivity scores were computed by regressing the CDI ratings in the affectively
positive condition from the CDI ratings in the affectively negative condition. A one-way ANOVA was conducted to compare the deficit, nondeficit and control groups on these scores. The ANOVA yielded a significant effect \( F[2, 58] = 7.74, p = 0.001 \), indicating that the three groups differed on level of affective reactivity of speech. A Scheffe post hoc analysis indicated that (1) the nondeficit group demonstrated significantly higher levels of affective reactivity of speech than either the deficit syndrome group \( (p < 0.01) \) or the control group \( (p < 0.01) \), and (2) the deficit syndrome and control groups did not differ significantly from each other on level of affective reactivity. This indicates that the nondeficit patients exhibited significantly elevated levels of affective reactivity of speech compared to both deficit syndrome patients and controls, even when differences between groups in baseline levels of communication disturbances were controlled statistically.

Bivariate correlations also were calculated between patients’ deficit syndrome severity scores (using the PDS as a continuous measure) and affective reactivity scores to determine whether severity of deficit symptomatology was related to affective reactivity of speech. All 51 patients were included in this analyses, including the 16 patients who were ambiguous with respect to deficit/nondeficit group assignment. Deficit syndrome scores were inversely correlated with affective reactivity scores \( (r[51] = -0.321, p < 0.05) \), indicating that higher levels of deficit syndrome symptomatology were associated with lower levels of affective reactivity.

### 3.3. Controlling for level of psychosis, medication and gender in the deficit and nondeficit patient groups

In order to control for differences in level of psychotic symptomatology between the deficit and nondeficit groups, a univariate ANOVA was conducted comparing the deficit and nondeficit patients on level of affective reactivity of speech, covarying out psychosis scores. The deficit and nondeficit groups remained significantly different, \( F[1,31] = 4.79, p < 0.05 \), indicating that the difference in level of affective reactivity of speech between the deficit and nondeficit patients was not due solely to differences in psychosis severity.

Deficit and nondeficit patients did not differ significantly on the type of medications being prescribed. In order to examine medication effects on level of affective reactivity of speech, a one-way ANOVA was conducted comparing four groups of patients: those who were receiving typical, atypical, both typical and atypical or no antipsychotic medications. There were no significant group differences on CDI ratings from the stressful or nonstressful conditions, or residualized affective reactivity scores. Patients who were receiving anticholinergic medications then were compared with those who were not. Again, there were no group differences on the CDI ratings or affective reactivity scores. Finally, there were no significant differences between males and females on CDI ratings or levels of affective reactivity in any of the three groups.

### 4. Discussion

The deficit, nondeficit and control groups each exhibited increased levels of disorder in their speech in the affectively negative condition as compared to the affectively positive condition. However, nondeficit patients demonstrated markedly and significantly higher levels of affective reactivity of speech than either patients with the deficit syndrome or healthy controls. Although patients with the deficit syndrome evidenced higher levels of baseline communication disturbances than controls, they resembled control participants in degree of affective reactivity of speech. Given that the patient groups did not differ with respect to level of education, IQ or psychosocial functioning, and that controlling for psychotic symptoms did not significantly change the results, it is unlikely that the present findings are due to generalized deficit effects. In summary, the results of this study support the idea that the relationship between negative affect/stress and speech disturbances is mediated, at least in part, by patients’ capacity to experience emotions. Hence, differences among patients in their capacity for emotional experience may help to explain previous findings that some patients exhibit marked affective reactivity of speech while others do not.

Alternatively, it is possible that both a diminished capacity for emotional experience and diminished levels of affective reactivity of speech are phenomena that are specific to the deficit syndrome, and that nondeficit patients tend to exhibit high levels of affective reactivity of speech regardless of their level
of emotional experience. In support of this formulation, when the nondeficit patient group was examined alone, deficit syndrome scores were not at all related to affective reactivity scores ($r[22] = -0.074, \text{ns}$). This finding adds some support to the idea that the deficit syndrome reflects a categorically defined subgroup of patients with a distinct underlying pathological process (see Kirkpatrick et al., 2001). However, further research will be required to determine whether the deficit syndrome truly reflects a categorical or continuous construct, and whether lower levels of affective reactivity occur exclusively in patients with deficit symptomatology.

Whether or not affective reactivity of speech is related to emotional capacity only in patients with the deficit syndrome, the capacity for emotional experience appears to be a promising construct for discriminating subtypes or distinct processes within schizophrenia. The present findings add further support to the idea that the diminished emotionality seen in the deficit syndrome may have beneficial or protective value (see Cohen et al., submitted for publication). Generally speaking, patients with the deficit syndrome are considered to be more severely ill than nondeficit patients in terms of overall psychopathology. However, deficit syndrome patients were more nearly normal with respect to affective reactivity of speech and consistent with some prior research had less severe psychotic symptomatology than nondeficit patients (Kirkpatrick et al., 1993; Buchanan et al., 1994, but see Buchanan et al., 1997). While conclusions are premature, these results suggest that in some cases, positive and negative/deficit symptom severity may be inversely related. Further research will be required to determine whether diminished emotionality in patients is associated with lower affective reactivity of other “reactive” positive symptoms such as disorganized behavior and clarify how positive symptoms such as hallucinations and delusions differ between deficit and nondeficit patients.

Several limitations of the present project warrant mention here. First, although there were no obvious medication effects, the methodology used in the present study did not permit a full analysis of the potential impact of medications on symptomatology or levels of affective reactivity of speech. Second, the use of a cross sectional case identification tool for deficit syndrome group assignment is not ideal because it does not assess the degree of stability of the putative deficit symptoms. Nonetheless, the PDS has been found to correspond highly to the SDS, a longitudinal measure of deficit syndrome. Furthermore, because PDS scores are based almost exclusively on affective symptoms from the BPRS, the PDS may actually be a better measure of diminished emotionality than the SDS. One drawback to using the PDS for group assignment is that it requires removal of the patients with mid range scores. This procedure reduced the sample size considerably in the patient group. However, the between-group findings were significant despite the relatively small sample size.

4.1. Conclusions

The present study found that patients with the deficit syndrome evidenced significantly lower levels of affective reactivity of speech than patients with nondeficit schizophrenia. These results suggest that emotion-related variables mediate the relationship between stress and language symptom exacerbations in at least some patients. Further research is required to determine whether other symptoms besides communication disturbances are similarly related to level of emotionality, and whether patients’ level of emotionality is a mediating vulnerability factor specific to the deficit syndrome, or whether it is a factor in schizophrenia more generally.

Acknowledgements

This research was supported by NIMH grants MH58783 and MH57151 to Dr. Docherty.

References


