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Cognitive Correlates of Schizophrenia Signs and Symptoms: II. Emotional Disturbances

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Abstract

We examined the cognitive and motor correlates of emotional disturbances in 47 schizophrenia spectrum individuals. Neither affective flattening nor anhedonia were significantly associated with tasks measuring working memory or attention/concentration, or with overall performance on tasks measuring fluency or episodic memory. In contrast, as expected, emotional disturbances were associated with patterns of hemispheric lateralization. Affective flattening and anhedonia were both associated with episodic memory laterality and there were similar trends with motor laterality. Anhedonia was also associated with medication motor side effects.

Keywords

affective flattening; anhedonia; hemispheric lateralization; medication motor side effects

1. Introduction

Although it is not uncommon for mental health professionals to think of schizophrenia as a “thinking disorder,” psychopathologists dating back to Bleuler (1911/1950) and Kraepelin (1919/1971) have considered emotional disturbances to be central to schizophrenia. Two of the most important and common emotional disturbances exhibited in schizophrenia are affective flattening and deficits in hedonic capacity (often referred to as anhedonia).

Affective flattening, sometimes called blunted affect, refers to diminished facial and vocal expressions of emotion. Affective flattening tends to be a stable sign of schizophrenia and also has prognostic importance – long-term follow-up studies have found that the presence of affective flattening is associated with poor long-term outcome among schizophrenia patients (Carpenter et al., 1978; Knight et al., 1979). An important clue to understanding affective flattening comes from research that has simultaneously examined facial expressions and reported subjective emotional experience – such research has demonstrated that schizophrenia patients with affective flattening are not more likely than patients without affective flattening to report diminished subjective experience of emotion¹ (Berenbaum and Oltmanns, 1992;

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¹As discussed below, it is common for schizophrenia patients to report a deficit in the ability to experience a particular type of emotion, pleasure, rather than a deficit in the ability to experience emotions in general.

Kring and Neale, 1996). Thus, among schizophrenia patients, deficits in the ability to experience emotion and deficits in the outward expression of emotion are independent.

That schizophrenia patients often report a deficit in the ability to experience pleasure was noted by both both Bleuler (1911/1950) and Kraepelin (1919/1971). Both Rado (1956, 1962), who referred to this phenomenon as anhedonia, and (Meehl 1962, 1975), who used the term hedonic capacity, posited that the diminished ability to experience pleasure plays a role in the development of schizophrenia. The results of several studies are largely consistent with this hypothesis, though such research has also found that most individuals with elevated levels of anhedonia do not develop schizophrenia spectrum disorders. Elevated levels of social anhedonia while in college have been found to predict the later development of psychotic symptoms and schizophrenia spectrum disorders (Chapman et al., 1994; Kwapil, 1998; Gooding et al., 2005). Similarly, Erlenmeyer-Kimling et al. (1993) found that physical anhedonia measured during adolescence predicted the development of psychosis in adulthood, though only among females.

We hypothesized that emotional disturbances in schizophrenia would be associated with signs of lateralized dysfunction and/or neuromotor problems rather than being associated with cognitive deficits. Our hypothesis was based in part on previous research conducted in our lab that had found that affective flattening² was associated with poorer performance on right relative to left hemisphere tasks (Stolar et al., 1994). A theoretical reason to expect these emotional features of schizophrenia to be associated with lateralized dysfunction is that several aspects of emotion appear to be lateralized. For example, the right hemisphere plays a much more prominent role than does the left hemisphere in the comprehension and expression of emotion (e.g., Tucker et al., 1977; Ross, 1981).

Two specific lateralization possibilities to explain anhedonia are suggested by the work of Davidson and colleagues (e.g., Davidson and Irwin, 1999) and Heller and colleagues (e.g., Heller et al., 1998). The work of Davidson and colleagues provides strong evidence that relative left vs. right frontal activation is associated with approach vs. withdrawal. Specifically, greater left relative to right frontal activation is associated with emotions associated with approach (most notably pleasure, but also anger, at least under some circumstances; Harmon-Jones et al., 2003), whereas greater right relative to left frontal activation is associated with emotions associated with withdrawal (those unpleasant emotions other than anger). Thus, based on Davidson's work, one might expect anhedonia to be associated with greater right relative to left hemisphere activation, particularly in frontal regions. In contrast, based on the work of Heller and colleagues, one might expect anhedonia to be associated with diminished right relative to left hemisphere activation. Heller et al. (1995) found that individuals who were depressed (and not anxious) tended to have lower levels of right posterior activation. Heller et al. (1995) had predicted these results on the basis of Heller's (1993) model positing that higher levels of right parietotemporal activation are associated with higher levels of emotional arousal.

In addition to lateralized dysfunction, there are also two reasons to suspect that anhedonia may be associated with neuromotor problems, including medication motor side effects. First, the basal ganglia, disturbances of which have been implicated in schizophrenia (e.g., Buchsbaum, 1990), are known to play an important role in movement disturbances and also play a role in positive emotional experiences (Lieberman, 2000). Second, dopaminergic disturbances have been implicated in at least some movement disorders (e.g., Parkinson's disease), antipsychotic medication targets dopamine neurotransmission, and dopamine has been linked to pleasure and reward (e.g., Bressan and Crippa, 2005).

²Stolar et al. (1994) did not examine anhedonia.

We had three reasons to suspect that neuromotor problems and/or lateralized dysfunction may be associated with affective flattening. First, motor problems are often found among individuals with schizophrenia (Manschreck, 1983), and neuromotor problems can precede the onset of schizophrenia (e.g., Fish et al., 1992). Further, Dworkin et al. (1993) found that among individuals at risk for schizophrenia (due to having a parent with schizophrenia), neuromotor dysfunction measured during childhood predicted affective flattening in adolescence. Second, the neuromotor problems that precede the onset of schizophrenia appear more often on the left side of the body (Walker et al., 1994), implicating a disturbance in the right hemisphere, and as noted above the right hemisphere plays a more prominent role than does the left hemisphere in the expression of emotion (e.g., Tucker et al., 1977; Ross, 1981). Third, the results of past research suggest that individual differences in emotional expression are associated with the ability to access and/or trigger motor programs necessary for emotional expression. For example, Berenbaum and Rotter (1992) found that individual differences in the accuracy and intensity of facial movements on a facial mimicry task were associated with intensity of facial expressions of emotion while participants viewed affect-eliciting filmclips; interestingly, performance on the facial mimicry task was not associated with self-reported emotional responses to the filmclips. Stolar et al. (1994) posited that affective flattening in schizophrenia reflects a right hemisphere disturbance that leads to difficulty accessing and/or triggering motor programs.

To summarize, the present study explored whether, among individuals with schizophrenia spectrum disorders, emotional disturbances (specifically anhedonia and affective flattening) are associated with: (a) cognitive deficits; and/or (b) signs of lateralized dysfunction (including, but not limited to, lateralized motor dysfunction); and/or (c) medication motor side effects.

2. Method

2.1. Participants

The participants were the same 47 individuals with schizophrenia spectrum disorders (39 schizophrenia patients and eight schizoaffective disorder patients) described in an accompanying report (see Berenbaum et al., 2006 for additional information). All participants were receiving outpatient services at the time of their participation in the study. Psychiatric diagnoses were made using DSM-IV criteria following the administration of the psychotic and mood disorders sections of the Structured Clinical Interview for the DSM-IV (First et al., 1998) and a review of clinical records.

2.2. Measurements/Instruments

Additional information concerning the cognitive measures described below can be found in the accompanying paper by Berenbaum et al. Affective flattening was measured using the unchanging facial expression, lack of vocal inflection, and affective nonresponsivity items from the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1983), which were then summed to form a single affective flattening score. We focused on those SANS ratings that most directly measure diminished emotional expression, omitting ratings that likely reflect social skill or other disturbances (i.e., poor eye contact, decreased spontaneous movements, paucity of expressive gestures, inappropriate affect, and subjective complaints of emotional emptiness). SANS ratings were made independently by the two experimenters who tested each participant. Interrater reliability³, measured using the intraclass correlation (Shrout

³Typically, interrater reliability is measured on the basis of two or more judges' degree of agreement concerning ratings based on the same observations or interview. In this case, the two judges observed the patients at different times, while engaging in slightly different activities. Thus, in this case, the intraclass correlation (ICC) is influenced by both interrater reliability and the degree to which the patients behaved differently at two different points in time, leading to a lower ICC than typically obtained.

and Fleiss, 1979), treating the raters as random effects and the mean of the raters as the unit of reliability, was .51. Disagreements were resolved by consensus following discussion with the first author.

Anhedonia was measured by asking participants to indicate how much pleasure they derived from 14 activities taken from the Urbana Pleasure Scale (Berenbaum, 2002). Participants were asked about three types of activities, social activities (e.g., “talking to a friend”), intellectual activities (e.g., “watching educational television”), and basic needs activities (e.g., “drinking some water when you are thirsty”). Participants indicated, using a four-point scale (ranging from “1=not at all” to “4=extremely”), how pleasurable each activity was. Because the amount of pleasure derived from the three types of activities were strongly correlated, and pleasure from all three types of activities were associated in similar ways with other variables, only results using a single total score, averaging across all activities, are reported here; higher scores indicate greater levels of anhedonia. The internal consistency of the 14-item scale, measured using Cronbach’s alpha, was .77.

A total fluency score was computed by averaging across scores on verbal and design fluency tasks, which were first standardized (i.e., converted into z-scores). A total memory score was computed by averaging across scores on verbal and face memory tasks, which were first standardized. A total working memory score was computed by averaging across scores on reading span and A-X CPT tasks, which were first standardized. The Digits Forward subtest of the WAIS-R (Wechsler, 1981) was administered as a measure of attention/concentration.

Lateralized motor performance was measured using a task described by Blyler et al. (1997). On this task, participants are asked to draw four diagonal lines, as straight as they can; two lines are drawn using the left hand and two lines are drawn using the right. The lines are digitized into x and y coordinates, and a linear regression analysis is then conducted. The root mean residual (RMS) associated with each line provides an index of the degree to which the line was straight – the straighter the line, the lower the RMS. The laterality score was then computed as follows: $(\text{RMS-left} - \text{RMS-right}) / (\text{RMS-left} + \text{RMS-right})$. Thus, higher (more positive) scores are indicative of better right hand relative to left hand performance. Lateralized motor performance was only examined among individuals who were right handed, as indicated by participants’ reports that when writing they used their right hand at least 90 percent of the time; these individuals also reported using their right hands the majority of the time when performing a variety of tasks (such as throwing a ball and using a spoon).

Dyskinetic motor side effects (i.e., signs of tardive dyskinesia) were measured using the Dyskinesia Identification System Condensed User Scale (DISCUS; Kalachnik and Sprague, 1993). Pseudoparkinsonian side effects were measured using the gait, salivation, glabella tap, and tremor items from the Modified Simpson/Angus scale (Simpson and Angus, 1970). Because the dyskinetic and pseudoparkinsonian motor side effect scores were highly correlated ($r=0.60$, $P<0.01$), and because the patterns of results were similar for the two scores, we computed a single extrapyramidal motor side effect score by summing the standardized dyskinetic and pseudoparkinsonian motor side effect scores.

2.3. Procedure

All participants were tested on the same day by the same two experimenters. One experimenter always administered the SCID, the assessment of medication motor side effects, the line drawing task, the Urbana Pleasure Scale, and the arrangement tasks, whereas the other experimenter always administered the remaining cognitive measures as well as the interview used to measure disturbed discourse coherence, verbosity, and syntactic complexity (see accompanying paper).

3. Results

Affective flattening scores ranged from zero to seven ($M=1.5$; $SD=1.8$). Anhedonia scores ranged from 1.9 to 3.9 ($M=3.0$; $SD=0.5$). The correlation between affective flattening and anhedonia was .21 (*NS*). Information concerning other symptoms can be found in the accompanying reports.

Associations (measured using Spearman rank order correlations, since as expected many of the score distributions were skewed) between the emotional disturbance measures and the cognitive and motor measures are presented in Table 1. As can be seen in Table 1, affective flattening and anhedonia were not significantly associated with average performance on the fluency, episodic memory, working memory, or digit span tasks. As predicted, however, affective flattening and anhedonia were associated with lateralized task performance. Both affective flattening and anhedonia were significantly associated with the memory laterality score; higher levels of affective flattening and anhedonia were associated with worse non-verbal relative to verbal memory performance, suggesting greater right than left hemisphere impairment. There was also a trend for anhedonia to be associated with the motor laterality score, with higher levels of anhedonia being associated with worse left hand relative to right hand performance, suggesting that anhedonia was associated with poorer right relative to left hemisphere performance. Affective flattening and anhedonia were not equally strongly associated with all of the laterality scores; there were trends for both affective flattening and anhedonia to be more strongly associated with the memory laterality score than with fluency laterality score, $z=1.60$, $P<0.06$, and $z=1.49$, $P<0.07$, respectively. In addition to being associated with memory and motor laterality, anhedonia was also associated with greater medication motor side effects. Further, there was a trend for anhedonia to be more strongly associated with medication motor side effects than was affective flattening, $z=1.60$, $P<0.06$.

4. Discussion

Neither affective flattening nor anhedonia were significantly associated with tasks measuring working memory or attention/concentration, or with overall performance on fluency and memory tasks. The absence of significant correlations with these cognitive measures cannot be explained by psychometric shortcomings of these tasks, since in the same sample of individuals, these same tasks were significantly associated with verbal communication disturbances (see accompanying paper by Berenbaum et al.). In contrast, both of these forms of emotional disturbance were associated with episodic memory laterality scores. In addition, motor laterality scores were also associated with affective flattening and anhedonia, albeit more weakly. In both cases, the results indicated that higher levels of emotional disturbance were associated with poorer right relative to left hemisphere performance.

The finding that affective flattening was associated with poorer right relative to left hemisphere performance is consistent with the findings of Stolar et al. (1994) and with our theoretical expectations given that the right hemisphere plays a more prominent role than does the left hemisphere in the expression of emotion. It is worth noting that affective flattening was not associated with fluency laterality; in fact, there was a strong trend for affective flattening to be more strongly associated with episodic memory laterality than with fluency laterality. It will be important for future research to determine which specific portions of the right hemisphere are associated with affective flattening, and why. The results of the present study are consistent with, but by no means prove, our hypothesis that affective flattening is associated with a right hemisphere disturbance that affects the ability to access and/or trigger motor programs associated with emotional expression.

Like affective flattening, anhedonia was associated with poorer right relative to left hemisphere performance, and was associated with episodic memory and motor laterality rather than with fluency laterality. This pattern of findings is inconsistent with a hypothesized lateralization hypothesis to explain anhedonia based on the work of Davidson and colleagues (namely that anhedonia is associated with greater right relative to left hemisphere activation, particularly in frontal regions). In contrast, this pattern of findings is consistent with a hypothesized lateralization hypothesis to explain anhedonia based on the work of Heller and colleagues (namely that anhedonia is associated with diminished right relative to left hemisphere activation, particularly in parietotemporal regions, that leads to diminished emotional arousal).

Interestingly, anhedonia but not affective flattening was associated with medication motor side effects. One possible explanation for this finding is that the medication side effects themselves interfere with patients' abilities to experience pleasure. A second possibility is that anhedonia is influenced by disturbances involving dopamine and/or the basal ganglia, both of which play roles in positive emotional experiences (Lieberman, 2000; Bressan and Crippa, 2005). A final, rather speculative possibility is that some neurobiological factor that increases vulnerability to schizophrenia (which may involve dopamine and/or the basal ganglia) makes individuals vulnerable to both anhedonia and to the side effects of antipsychotic medication. Such a possibility is consistent with all of the following: (a) differential patterns of cortical activation precede the onset of tardive dyskinesia (Szymanski et al., 1996); (b) neuromotor problems can precede the onset of schizophrenia (e.g., Fish et al., 1992); (c) tardive dyskinesia is associated with obstetric complications and neurological hard signs (Ismail et al., 2001); (d) signs of dyskinesia are found among approximately one-quarter of medication-naïve, well siblings of schizophrenia patients (Ismail et al., 2001); and (e) at least some pleasure deficits appear to be associated with vulnerability to psychosis and schizophrenia spectrum disorders (e.g., Erlenmeyer-Kimling et al., 1993; Chapman et al., 1994; Kwapil, 1998; Gooding et al., 2005).

The results of the present study raise the possibility that cognitive disturbances may not be associated with affective flattening or anhedonia. That attention was not found to be associated with emotional disturbances in schizophrenia is consistent with the results of Berenbaum and Oltmanns (1992), who found that emotional responses of persons with schizophrenia did not vary as a function of the cognitive demand of the affect-eliciting stimuli. That working memory was not found to be associated with emotional disturbances is particularly interesting given that past research has consistently found working memory deficits in schizophrenia patients (Lee and Park, 2005), and in the same sample of patients described in the present paper, working memory was significantly associated with disturbances in discourse coherence (see accompanying paper by Berenbaum et al., 2006). Thus, while working memory disturbances may be central to schizophrenia as a whole, and also important for understanding differences among schizophrenia patients in terms of their levels of formal thought disorder, they were not found to be associated with individual differences in emotional disturbances among schizophrenia patients in the present sample. Although it is premature to conclude that emotional disturbances in schizophrenia are not associated with cognitive deficits, the results of the present study suggest that affective flattening and anhedonia are likely to be associated with some form of lateralized dysfunction, and that anhedonia is also likely to be associated with the same neural substrates involved in medication motor side effects, such as dopamine and/or the basal ganglia. Such a pattern is consistent with the assumption that underlies our research, namely that the different signs and symptoms of schizophrenia are differentially associated with the different etiological factors that give rise to them.

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Table 1

Associations Between Emotional Disturbances and Cognitive and Motor Variables

<u>Cognitive and Motor Variables</u>	<u>Affective Flattening</u>	<u>Anhedonia</u>
Fluency	0.01	−0.06
Episodic Memory	0.01	−0.19
Working Memory	−0.001	−0.12
Digit Span	0.13	−0.06
Fluency Laterality	0.04	0.10
Memory Laterality	0.39**	0.42**
Motor Laterality	0.23	0.34 [†]
Medication Motor Side Effects	0.03	0.33*

[†]
 $P < 0.06$ *
 $P < 0.05$ **
 $P < 0.01$ (2-tailed)