Electrodermal Activity as a Prodromal Sign in Schizophrenia

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Introduction

Schizophrenia is often characterized by successive periods of psychotic symptoms and remission. Phenomenological and behavioral changes sometimes occur within a few days or weeks prior to the appearance of psychotic symptoms (Herz and Melville 1980; Subotnik and Nuechterlein 1988). If further developed, these “prodromal signs” might serve as early warning signs of impending psychotic episodes and, with appropriate intervention, might be used to circumvent subsequent relapses; however, phenomenological and behavioral prodromal signs require accurate self-monitoring and reporting of subtle experiential and behavioral changes, and may have limited sensitivity and specificity (see review by Norman and Malla 1995).

Physiological measures that do not rely on self-report might be useful supplemental prodromal signs. A promising measure in this context is electrodermal activity (EDA), which is a measure of palmar sweat gland activity and a peripheral index of sympathetic nervous system arousal (Dawson et al 1990). EDA abnormalities in schizophrenia have been noted for many years (see reviews by Bernstein et al 1982; Ohman 1981). More relevant to the study of prodromal signs, however, is the finding that increases in EDA occur in schizophrenia patients during a psychotic episode compared to a symptomatically remitted period (Dawson et al 1994), and in the weeks prior to psychotic relapse or exacerbation in 2 patients tested on a weekly basis (Dawson et al 1992, pp 305–306).

These data suggest that increased EDA may serve as a prodromal sign in at least some schizophrenia patients. To test this hypothesis, we examined EDA measures in a symptom-free 3-week period prior to a psychotic relapse or exacerbation, a time period in which prodromal signs might be expected to appear. We then compared the EDA obtained from this “prodromal period” with the same EDA measures obtained in a “control period” that was not shortly followed by a relapse or exacerbation.

Methods and Materials

Subjects and Design

A within-subject design was possible because EDA measures were collected on repeated occasions from outpatients in the UCLA Aftercare Program participating in an ongoing longitudinal project entitled “Developmental Processes in Schizophrenic Disorders” (Nuechterlein et al 1992). All participants signed an informed-consent form and were provided additional information orally to enhance their understanding of the research project.

For the present analysis, we examined data from five male participants (average age of 25.2 years) in the longitudinal project who happened by chance to have EDA measured at two distinct times: 1) a “control period” in which the subject was in a state of remission and did not exacerbate or relapse for at least 7 months following; and 2) a “prodromal period” in which the subject was in a state of remission but exacerbated or relapsed...
within the following 3 weeks. Each patient received the same dosage of the same medication or received no medication during the two tests.

Symptomatic Measures

States of remission, psychotic relapse, and psychotic exacerbation were defined according to published criteria (for details, see Nuechterlein et al 1992, pp 395–396) in terms of scores on the brief psychiatric rating scale (BPRS) (Overall and Gorham 1962; Lukoff et al 1986). BPRS data were collected every 2 weeks by trained clinicians. The BPRS rates symptoms on a scale from 1 (not present) to 7 (extremely severe), with ratings of 4 and higher in the pathological range. Remission was defined as ratings of 3 or less on all BPRS scales for at least two consecutive biweekly evaluations. Psychotic exacerbation was defined in terms of increases of at least 2 points in the sum of the three BPRS psychotic items (unusual thought content, conceptual disorganization, and hallucinations) and a rating in the pathological range on at least one of these items. Two of the patients showed increases that qualified as minor “psychotic exacerbations” (ratings of 4 or 5 on at least one psychotic item), whereas the remaining 3 patients showed increases that qualified as “psychotic relapses” (ratings of 6 or 7 on at least one psychotic item).

Electrodermal Measures

Two tonic and three phasic EDA measures were recorded during both the prodromal and control periods. The two tonic measures were 1) log of skin conductance level (SCL) and 2) frequency of nonspecific skin conductance responses. The three phasic EDA measures were indexed by the number of repetitions of auditory stimuli before the skin conductance response habituated. The number of trials to habituation was measured to three types of auditory stimuli: 1) innocuous tones; 2) tones to which the subject was asked to attend; and 3) bursts of loud noise. Further details regarding the procedures of recording and quantifying the EDA measures are available elsewhere (Dawson et al 1992).

Results

As predicted, the mean of each electrodermal variable was higher during the “prodromal period” than during the corresponding “control period.” For each EDA measure a one-tailed nonparametric Wilcoxon signed-ranks test was performed to test the significance of the difference of prodromal and control values. A nonparametric test was used because with a small sample like that available here, the normality of the population distribution cannot be verified. The log SCL and the number of trials to habituation to the innocuous tones were found to be marginally higher in the prodromal period than in the control period (p = .062), whereas the remaining measures failed to reach statistical significance. The mean and standard deviation for the difference scores were 0.22 and 0.24 for the log SCL measure, and 3.60 and 4.83 for the number of trials to habituation measure.

We next examined the data for each individual patient. The mean SCL values for each patient during the control and prodromal periods are shown in Figure 1. As can be seen, SCL increased during the prodromal test compared to the control test in 4 of the 5 patients. (Although not shown in Figure 1, the number of trials to habituation to the innocuous tones also increased in the prodromal period compared to the control period in the same 4 patients.) The one exception was a patient diagnosed as having schizoaffective disorder, mainly schizophrenic, whereas the other patients were diagnosed as having schizophrenia according to research diagnostic criteria (Spitzer et al 1978). The 1 patient who failed to show EDA increases experienced a psychotic relapse associated with depression and suicidality, unlike the other 4 patients, and there is evidence that EDA decreases during psychotic depression (Sponheim et al 1995).

Discussion

The present results indicate that electrodermal activity is likely to increase prior to the exacerbation of psychotic symptoms in schizophrenia patients, particularly if the psychotic symptoms are uncomplicated by depression. This finding supports the hypothesis that many vulnerable individuals pass through a transient “intermediate state,” marked by sympathetic nervous system hyperarousal, before experiencing a schizophrenia episode (Dawson et al 1983; Nuechterlein and Dawson 1984). These results are consistent with findings from 2 patients in addition to those reported here who were tested on a week-by-week basis and exhibited increased SCL before exhibiting symptomatic relapse or exacerbation (see Figure 5 in Dawson et al 1992), with phenomenological changes during the prodromal period (tension, restlessness, trouble sleeping), and with reports that psychostimulants such as methylphenidate can trigger transient increases in positive symptoms in schizophrenia patients (Janowsky et al 1973). Thus, the present findings shed light on the nature of the early subclinical psychophysiological processes that underlie the emergence of psychotic symptoms.

Beyond these theoretical implications, the present findings also suggest that electrodermal measures may be useful adjuncts
to currently available prodromal signs; however, several critical issues need to be addressed satisfactorily before the practical implications can be realized (e.g., since EDA fluctuates even in the absence of imminent relapse, how large of an EDA increase over what sustained period of time qualifies as a likely prodromal sign?). Nevertheless, if the present findings are replicated in larger samples and adequate sensitivity and specificity are demonstrated, then clinicians and patients might monitor sympathetic activity as an aid in the detection of prodromal signs and attempt preventive intervention through increased medication, reduction of environmental stressors, or improved coping strategies.

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References


