The temporal stability of electrodermal variables over a one-year period in patients with recent-onset schizophrenia and in normal subjects

ANNE M. SCHELL, a MICHAEL E. DAWSON, b KEITH H. NUECHTERLEIN, c KENNETH L. SUBOTNIK, d AND JOSEPH VENTURA d

a Department of Psychology, Occidental College, Los Angeles, California, USA
b Department of Psychology, University of Southern California, Los Angeles, California, USA
c Departments of Psychology and Psychiatry and Biobehavioral Sciences, University of California at Los Angeles, Los Angeles, California, USA
d Department of Psychiatry and Biobehavioral Sciences, University of California at Los Angeles, Los Angeles, California, USA

Abstract

Test–retest stability of electrodermal (EDA) variables indexing both general autonomic arousal (e.g., skin conductance level, number of nonspecific skin conductance responses) and attention to external stimuli (e.g., number of skin conductance orienting responses, electrodermal responder/nonresponder status) was assessed in 71 young, recent-onset schizophrenia patients and 36 demographically matched normal subjects. Significant stability over a 1-year period was found for both patients and normal subjects for most EDA variables and for responder/nonresponder status, with test–retest correlations generally being higher for normal subjects. The lower reliability for patients was not attributable to symptomatic fluctuations during the follow-up period and may reflect poorer arousal regulation among the patients. Among measures of responding to nontask stimuli, a simple count of the number of orienting responses occurring was more stable than was a traditional trials-to-habituation measure.

Descriptors: Schizophrenia, Electrodermal activity, Electrodermal lability, Reliability, Skin conductance orienting response, Electrodermal activity responders/nonresponders

In the psychophysiological study of schizophrenia, abnormalities in electrodermal activity (EDA) are among the most widely reported phenomena. In general, two consistent differences between schizophrenia patients and nonpatient subjects have been reported. The first is that schizophrenia patients show deficits in orienting as assessed with the skin conductance component of the orienting response, with approximately 50% of patients being “nonresponders” as opposed to approximately 10% of normal subjects. The majority of the remaining patients (“responders”) are reported to show a pattern of rapid habituation of the skin conductance orienting response (SCOR) (Bernstein et al., 1982; Dawson & Nuechterlein, 1984; Iacono, Ficken, & Beiser, 1993; Öhman, 1981). Orienting responses are thought to be related to attention to and processing of external stimuli (Öhman, 1992; Schell, Dawson, Hazlett, Filion, & Nuechterlein, 1993; Siddle & Spinks, 1992). Thus, the nonresponsiveness seen in schizophrenia may reflect reduced ability to allocate information processing resources to external stimuli, or perhaps reduced overall information processing capacity (Bernstein, 1987; Dawson, 1990; Dawson & Nuechterlein, 1984; Hazlett, Dawson, Filion, Schell, & Nuechterlein, 1997; Nuechterlein & Dawson, 1984).

The second electrodermal abnormality is that responder schizophrenia patients, who show at least some level of orienting activity to innocuous stimuli, exhibit abnormally high levels of tonic electrodermal activity as indexed by resting skin conductance level (SCL) and nonspecific skin conductance responses (NS-SCRs; Dawson & Nuechterlein, 1984; Dawson, Nuechterlein, & Schell, 1992; Öhman, 1981). This latter finding has been interpreted as reflecting higher general arousal levels in these patients.

Considerable evidence suggests that lowered SCOR responsiveness to stimuli may be an indicator of a vulnerability factor for...
Temporal stability of EDA variables in schizophrenia

Schizophrenia. SCOR nonresponding is more common among first-degree relatives of schizophrenia patients (Ficken & Iacono, 1990) and among remitted schizophrenia patients (Iacono, 1982) than among controls. Moreover, several studies that have followed the same patients in states of mild and severe symptoms and have found SCOR nonresponding in both states (Olbricht, 1991; Spohn, Coyne, Wilson, & Hayes, 1989; Zahn, Carpenter, & McGlashan, 1981).

On the other hand, at least in young, recent-onset schizophrenia patients, measures of heightened electrodermal arousal such as resting SCL, NS-SCR levels, and the rate of decline in SCL from the beginning to the end of a resting period appear to be episode indicators, since they were found to be elevated during a symptomatic state compared to normal subjects but not during remission (Dawson, Nuechterlein, Schell, Gitlin, & Ventura, 1994). Thus, phasic SCOR hyporesponsiveness may reflect information processing deficits related to the vulnerability to schizophrenia, whereas tonic EDA hyperactivity may reflect an arousal abnormality related primarily to the present state of the patient.

For an abnormality to be considered a vulnerability indicator or trait marker for schizophrenia, it must have reasonable stability over time in schizophrenia patients. The demonstration that a group of patients differs from normal subjects with respect to a variable both when in a symptomatic state and in remission does not, in and of itself, establish that the variable has reasonable stability within individuals over time. It is possible that differences between group means could be created by one subgroup of patients during remission and by a different subgroup during relapse. EDA variables that normalize with remitted symptomatic state, such as SCL and NS-SCRs, may nevertheless show significant stability over time, as they may also reflect relatively stable tonic differences in activation or arousal between individuals onto which symptomatic changes are superimposed. Stability in measures of arousal over time may be particularly great if symptom levels are stable for some individuals. Arousal differences among individuals may, in fact, be causally related to symptom patterns if, as hypothesized, heightened arousal negatively impacts information processing and coping (Nuechterlein & Dawson, 1984; Turpin & Clements, 1992; Zabin & Spring, 1977). Thus, the stability over time of EDA measures in the general population and in schizophrenia patients in particular is a critical issue influencing how we conceptualize these EDA abnormalities.

The stability of EDA measures such as NS-SCR levels, resting SCL, and responsiveness to stimuli has been fairly well investigated in the general population (Bull & Gale, 1973; Crider & Lunn, 1971; Lacey & Lacey, 1958; Siddle & Heron, 1976; see Freixa i Baque, 1982 for a compilation of early studies). Early studies reported test–retest correlations (generally Pearson r values) over time periods from 1 day to 1 year or more for resting NS-SCR levels that ranged from approximately .40 to .75, with a median around .55; for basal SCL levels (including both resting SCL and SCL during periods of stimulation), correlations ranged from approximately .40 to .85, with a median around .60; and for number of SCORs elicited by a series of repeated stimuli, correlations ranged from approximately .30 to .80, with a median around .60. More recent studies have continued to report correlations in these ranges in the normal population (Iacono et al., 1984; Schell, Dawson, & Filion, 1988; Vossel & Zimmer, 1990). Thus, it is reasonable to conclude from the literature that the EDA variables of particular interest in the study of schizophrenia show moderate test–retest stability for periods up to a year or more in the normal population.

Stability of these measures among patients with schizophrenia has been much less studied and might be expected to differ from the general population, as in schizophrenia patients, tonic EDA measures such as SCL and NS-SCR levels may fluctuate with symptomatic state. Moreover, there is preliminary evidence that increases in arousal as indexed by EDA may occur prior to symptomatic exacerbation or relapse in remitted patients (Hazlett, Dawson, Schell, & Nuechterlein, 1997). These findings suggest that EDA may be less stable in patients with schizophrenia than in the general population, reflecting less successful arousal modulation in these patients, with increases in arousal setting the stage for the reemergence of symptoms. Thus, the degree to which EDA variables represent reliable individual difference variables in the population with schizophrenia must be separately examined.

Among the few studies that have examined reliability of EDA measures in schizophrenic patients, significant test–retest correlations have been reported for tonic SCL and NS-SCRs (Alm, Öst, & Öhman, 1987; Bernstein, 1965). In contrast, the few reports on phasic SCOR responsivity are more variable. In a group of young, principally recent-onset schizophrenic patients, Alm et al. failed to find significant test–retest correlations for either trials-to-habituation or the responder/nonresponder status measured from hospital admission to the end of treatment (a period of slightly greater than 1 year). Although interpretation of these data is somewhat complicated by the fact that the medication status of patients changed over time, symptomatic state changed, and no data are available from nonpatient controls, the poor reliability of these commonly used measures is of concern.

However, greater stability of responder/nonresponder status over a shorter period of time was reported by Spohn et al. (1989) in chronic hospitalized patients participating in a drug withdrawal study. Even though the medication status of most patients changed across sessions, 80% of patients had the same SCOR categorization initially and after 2 weeks, 74% had the same categorization initially and after 4 weeks, and 63% had the same categorization on all three test occasions.

The present study is an investigation of the 1-year test–retest stability of EDA variables reflecting both responsiveness to external stimuli and autonomic arousal over a 1-year period in both schizophrenic patients and demographically matched normal subjects. The patients were young, recent-onset schizophrenic patients whose medication status was fairly uniform both initially and at 1-year follow-up. A subset of patients whose symptomatic state was the same at both time points was separately examined to determine the effects of fluctuations in symptoms on EDA stability.

Method

Participants

Participants were 71 schizophrenia patients and 36 demographically matched normal individuals who were participating in Sample 1 of a longitudinal follow-through study of the early phases of schizophrenia, Developmental Processes in Schizophrenia Disorders (Nuechterlein et al., 1992). The patient and normal groups were dominantly male (82% and 72%, respectively), with an average age of 23.4 (SD = 4.2) and 24.4 (SD = 3.8) years and average years of education of 12.5 (SD = 1.8) and 13.4 (SD = 1.6). Both groups were predominantly Anglo in ethnicity, 89% for patients and 92% for normals. The time interval between initial and follow-up tests was 12.4 months (SD = 1.2) for patients and 13.1 (SD = 3.6) for normals. All patients received a diagnosis of schizophrenia or schizoaffective disorder, mainly schizophrenic,
using Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978), with their first psychotic episode within 2 years before entry into the longitudinal study. Other inclusionary and exclusionary criteria for patient and normal subject selection can be found in Nuechterlein et al. (1992). All patients and matched normal subjects from this project for whom electrodermal data were available at the outpatient baseline and 1-year points were included in these analyses.

**Overall Design**

Patients were treated at the Aftercare Research Program at the University of California–Los Angeles, using a common starting dosage of 12.5 mg of injectable fluphenazine decanoate (Prolixin) every 2 weeks as soon as their clinical state stabilized. Dosage was decreased if intolerable side effects occurred and increased as clinically indicated if significant symptoms recurred. Initial stabilization was followed by at least 1 year of neuroleptic treatment, which in turn followed by other voluntary protocols described elsewhere (Dawson et al., 1994; Nuechterlein et al., 1992). Subjects were generally seen in the Aftercare Program at 2-week intervals. A battery of neurocognitive performance, psychophysiological, and other measures was administered on prespecified occasions throughout the longitudinal procedure. The test occasions included the initial intake point, a point at which the patient had been clinically stabilized on medication, usually about 3 months after hospital discharge (initial outpatient stabilization), and a point approximately 1 year after the initial stabilization point (1-year follow-up). Other tests were triggered by clinical state; these could include a test at a point of symptomatic remission or what appeared to be the best clinical state for the patient, and tests associated with psychotic or nonpsychotic symptom exacerbation or relapse. Normal subjects were administered the same battery of tests at intervals comparable to those of patients. Written informed consent was obtained from each patient and normal subject prior to participation. Participants were also provided with additional information orally to further enhance their understanding of the research project.

The EDA data reported here were collected on two test occasions, the initial outpatient stabilization test (after the patient had been stabilized on injectable fluphenazine), and a follow-up test approximately 1 year later. At this initial test, all but 5 patients were receiving injectable fluphenazine (primarily 12.5 mg), and at the follow-up test, all but 6 were being treated with injectable fluphenazine. When clinically possible, antiparkinson medications with anticholinergic effects were discontinued for at least 24 hr prior to each electrodermal test. This was possible for all but 7 patients at the initial test and all but 5 patients at the follow-up test.

**Identification of Clinical States**

The Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962) was used to identify the clinical state of the patients on both test occasions. Remission was defined as being rated 3 or less (on a scale of 1 to 7) on all items on the standard 18-item BPRS (Overall & Klett, 1972) on the day of testing and for a period of at least 1 month preceding testing. Ratings of 3 or less comprise the nonpathological range of the BPRS (Lukoff, Nuechterlein, & Ventura, 1986; Ventura et al., 1993). A psychotic state was defined as having one or more of the three BPRS psychotic items (Unusual Thought Content, Hallucinations, and Conceptual Disorganization) rated 4 or higher on the day of testing. Patients were classified as being in Other Symptomatic State if they obtained a rating of 4 or higher on any nonpsychotic BPRS item.

**Electrodermal Testing Procedures**

Electrodermal testing procedures have been fully described elsewhere (Dawson et al., 1994), and recording procedures followed standard methods (see Dawson, Schell, & Filion, 2000); they will only be summarized here. Each test session began with the attaching of recording electrodes, followed by a 5-min rest period and three successive series of tone presentations. The first series measured SCORs to mild nonsignal tones (78 dB, 1.0 s duration, 25 ms controlled rise and fall times) presented binaurally through headphones. The first 12 tones were 1000 Hz and the last three were 1200 Hz. These stimulus parameters were chosen to reproduce conditions under which SCOR orienting and habituation abnormalities have been found previously in patients with schizophrenia (Bernstein et al., 1982; Gruzelier & Venables, 1972). The second series measured SCORs to 12 task-significant tones. The tones were 500 Hz, 1.0 s in duration, and presented monaurally, with subjects being instructed to move their eyes toward the direction from which the tone was presented. The third auditory series, from which most of the phasic response measures reported here were taken, involved presentation of 12 loud (98 db, 1.0 s duration) white noise bursts.

**Dependent Variables**

Fifteen electrodermal summary variables were examined. The operational definitions of these variables are largely as found in Dawson et al. (2000). They included the following.

**Measures of tonic EDA arousal:**

1. NS-SCRs: the average number of nonspecific skin conductance responses (0.05 μS or greater) per minute across the resting phase and the three stimulus sequences.
2. SCL: the average skin conductance level during the experimental session, taken by averaging log SCL during the resting phase (the average of the minute-by-minute levels read during the resting phase) and the three log SCL values obtained during the three auditory series. For each series the average of the log SCL values at stimulus onset was obtained.
3. SCL decline: the linear regression coefficient for the regression of log SCL measured at stimulus onset on trial number, a measure of decline in SCL over time during the loud noise series.

**Measures of overall responsiveness to stimuli:**

4. TTH1 (Trials to Habituation): the number of trials during the nontask series that occurred prior to habituation of the SCOR. Habituation was defined as the occurrence of three consecutive trials on which no response occurred. If the subject failed to respond on the first three trials, TTH1 was equal to 0.
5. TTH2: the number of trials during the task series that occurred prior to habituation of the SCOR, as defined above.
6. TTH3: the number of trials during the loud noise series that occurred prior to habituation of the SCOR, as defined above.
7. NOR1: the total number of SCORs elicited by the tones during the nontask series.
8. NOR2: the total number of SCORs elicited by the tones during the task series.
9. NOR3: the total number of SCORs elicited by the tones during the loud noise series.

**Measures of individual response characteristics**: The following additional variables reflect the more fine-grained characteristics of individual elicited SCRs. They were taken from the data of the loud noise trial series, because the loud noises were much more likely to elicit SCRs than were the nonsignal or signal tones.

10. Amplitude: Average baseline to peak amplitude of SCRs; zero response trials are not included.

11. Magnitude: Average baseline to peak amplitude of SCRs with zero response trials included. The magnitude measure reflects the amplitude characteristics of individual responses, but also reflects overall responsiveness as does NOR, in that it reflects the number of trials on which a response did not occur.

12. Latency: Average time from stimulus onset to SCR onset for any response elicited by the loud noise and beginning in a window from 1.0 to 4.0 s after stimulus onset; zero response trials are not included.

13. Rise time: Average time from SCR onset to peak; zero response trials are not included.

14. Rise rate: Average value of amplitude divided by rise time of SCRs.

15. \( \frac{1}{2} \) recovery time: Time from SCR response peak to the point at which the response has fallen halfway back toward response onset baseline; zero trials are not included.

**Results**

**Clinical States Initially and at Follow-up**

The clinical states of the 71 patients at the initial outpatient test and at follow-up are shown in Table 1. As can be seen, on both the day of the initial outpatient test and at the 1-year follow-up test, only 18% of the patients were experiencing psychotic symptoms.

**EDA Correlations Across Time Among Patients and Normal Subjects**

For the patients and normal subjects separately, correlations were computed between initial outpatient and follow-up values for each EDA variable. These correlations are shown in Table 2. Degrees of freedom vary from one EDA variable to the next because a number of the variables (latency, rise time, rise rate, amplitude, recovery time) could not be calculated if a participant was completely nonresponsive to the loud noise. Equipment difficulties occasionally caused loss of data during one or the other of the trial series. Because test–retest correlations of these EDA variables would be predicted to be positive if they existed, one-tailed tests of significance were used. Measures of individual response characteristics (amplitude, latency, etc.) are taken from the loud noise stimulus series.

Test–retest correlations for the EDA variables across time were generally significant in both groups. They are particularly high for measures of tonic sympathetic activation (SCL, NS-SCR) and for variables that wholly or partly reflect overall responsiveness to stimuli, such as NOR and SCR magnitude, while being generally lower for measures of the properties of individual responses (amplitude, latency, rise time, etc.)

Of particular interest is the greater stability of the NOR1 measure compared to that of the TTH1 measure in both patients and normal subjects, because both have frequently been used as measures of responsiveness to stimuli in schizophrenia research. Both measures here are computed from the data of the innocuous tone trial series. However, NOR1 is a simple count of the number of tones out of the series of 12 that elicited an SCOR, whereas TTH1 takes into account information about the sequence of trials on which responses do and do not occur. A subject who responds on three of the innocuous tone trials will have an NOR1 value of 3, regardless of the trials on which those three responses occurred. However, depending on the sequence of response and nonresponse trials, that subject could have TTH1 values ranging from 0 (if the first response occurred on the fourth trial) to 10. The responsiveness measure that is uninfluenced by sequencing (NOR1) is more stable in both groups. We tested the significance of the difference between the test–retest correlations of NOR and TTH1 in both normal subjects and patients using a procedure described by Olkin.
(1967) and found the difference for normal subjects between the values of .64 and .19 to be significant \( (p < .05) \); the smaller difference for patients was not significant. Similarly, NOR2 and NOR3 show greater stability than do TTH2 and TTH3, although these differences are not as pronounced as for NOR1 and TTH1 and are not significant.

Also of interest is the tendency for SCR magnitude to be more stable than SCR amplitude in both groups. The magnitude measure averages response amplitudes across all trials, with a zero being entered for trials on which no response occurs, whereas the amplitude measure is based only on trials on which a nonzero response occurs. The measure which integrates information about the frequency as well as the amplitude of responses is more reliable.

**Differences in Stability Between Schizophrenia Patients and Normal Subjects**

Three EDA variables were of a priori interest to us because of their hypothesized status as episode indicators in schizophrenia (NS-SCRs, SCL, and SCL decline). It can be seen in Table 2 that for NS-SCRs and SCL, the stability is somewhat higher among the normal subjects than among the patients. Greater stability would be predicted for the normal subjects with respect to variables that are episode indicators, because these will be influenced by level of symptomatology, which could vary from the initial test to the follow-up test for these patients. A priori tests for these variables of the significance of the difference between independent correlations revealed that for the NS-SCR measure, the difference is significant, \( p < .04 \) (one-tailed).

For the remaining EDA variables, directional hypotheses were not made, so two-tailed tests for the significance of the difference between the correlations for the patients and normal subjects were carried out, and a Rom procedure (a sequentially rejective test for carrying out post hoc comparisons; Rom, 1990) was used to control for Type I error. The only difference between these correlations that reached significance was for \( \frac{1}{2} \) recovery time, which showed greater reliability among normal subjects.

To determine if the lower level of reliability seen in the patients for variables thought to be symptomatically related was, in fact, due to symptom changes within patients, we identified a subgroup of 19 patients who were the most symptomatically stable between the initial outpatient and follow-up tests. These patients were those who were the most symptomatically stable between the initial and 1-year tests; stable patients had standard deviation averages response amplitudes across trials, with a zero being entered for trials on which no response occurs, whereas the amplitude measure is based only on trials on which a nonzero response occurs. The measure which integrates information about the frequency as well as the amplitude of responses is more reliable.

**Stability of Responder Versus Nonresponder Status**

As discussed above, a distinction has often been made between SCOR responder and nonresponder schizophrenia patients, usually with reference to responses to innocuous, nontask stimuli. To examine the stability over time of this classification, we classified both patients and normal subjects as responders or nonresponders on the basis of their TTH1 values during the innocuous tone series, both initially and at follow-up. Following the definition used by Bernstein et al. (1982), a responder was a subject who responded at least once during the first three presentations of the nonsignal tone, whereas a nonresponder was a subject who failed to respond to any of the first three tones. Consistency of classification over time and kappa values for patients and normal subjects are shown in Table 3.

For normal subjects, initial status was significantly related to status at follow-up as measured by kappa, with 68% being stable fluctuations occur over time in the patient group. To verify this hypothesis, we calculated for each subject the absolute magnitude of the initial–follow-up difference in NS-SCRs and carried out a \( t \) test comparing the groups. The group difference was significant, \( t = 3.15, df = 104, p < .01 \), with the patients showing the larger changes. Figure 1 shows the changes between the initial and follow-up tests for NS-SCRs for patients and normal subjects. As can be seen, changes among the patients are larger and more variable than among the normal subjects.

**Table 3. Responder versus Nonresponder Classification (Based on TTH1) Initially and at Follow-up**

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<th>Controls Follow-up test</th>
<th>Patients Follow-up test</th>
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<td></td>
<td>Responder</td>
<td>Nonresponder</td>
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<tr>
<td>Initial test</td>
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<tr>
<td>Responder</td>
<td>12 (34%)</td>
<td>6 (17%)</td>
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<td>Nonresponder</td>
<td>5 (14%)</td>
<td>12 (34%)</td>
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<td>Kappa (33)</td>
<td>( .37, p &lt; .03 )</td>
<td>Kappa (69) = .12</td>
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in their classification, equally divided among stable responders and stable nonresponders. For the patients, 56% are stable in classification, with stable nonresponders being more common. Initial status was not significantly related to follow-up status. Although controls but not the patients show significant within-group stability in classification over time, the proportion of subjects who are stable in their classification does not differ significantly between the two groups. These results suggest that there may be slightly greater stability in responder/nonresponder classification for normal subjects than for patients, particularly in the responder category.

Because test–retest reliability was somewhat greater for the NOR variable than it was for the TTH1 measure, we reclassified subjects as responders or nonresponders based on the NOR1 values initially and at follow-up and examined that stability of classification over time, which is shown in Table 4. For the normal subjects, there is no change in stability across the two methods of classification, with 68–69% being stable in classification with the NOR measure. For the patients, the improvement in stability with the NOR-based classification is more marked, with 64% being either stable responders or nonresponders. As Tables 3 and 4 show, considerably more patients were classified as responders both initially and at follow-up (12) using the NOR measure. This indicates that a pattern of responding to the nonsignal tones in which there are no responses given during the first three presentations of the tones (making a person a TTH1 nonresponder) but responses are given to subsequent tones (making a person an NOR responder) is more common among patients than among normal subjects. Similarly, NOR2 produced greater stability in responder/nonresponder classification than did TTH2, and NOR3 greater stability than TTH3.

Differences Between Patient and Normal Groups Over Time
To examine group differences in EDA over these time points, we carried out a two-way (Groups × Time Point) ANOVA of electrodermal variables that we and other investigators have previously reported to discriminate between schizophrenia patient and normal groups, either in symptomatic or remitted states or both. These were overall NS-SCR and SCL measures and SCL decline over time, which appear to be psychotic state indicators, and the measures of responsivity TTH1, TTH2, and TTH3, which have been suggested to be vulnerability indicators.

The ANOVA of the NS-SCR levels indicated a significant overall effect of group, $F = 6.91, df = 1.104, p < .01$, with the patients having higher levels than the normal subjects at both time points. Neither the effect of time nor the interaction were significant. Both initially and at follow-up, the NS-SCR rate of the patients was approximately twice that of the normal subjects [1.62 ($SD = 1.94$) vs. 0.84 ($SD = 0.97$) initially and 2.01 ($SD = 2.59$) vs. 0.92 ($SD = 1.11$) at follow-up].

We have previously reported (Dawson, Nuechterlein, & Schell, 1992) that in young, recent-onset patients, NS-SCR levels are greater among patients than among normal subjects only among subjects who are electrodermal responders. To investigate this question in the present sample, we divided both patients and normal subjects into responders and nonresponders based on the NOR1 variable, with a nonresponder having an NOR score of 0. The NOR1 variable was selected because of its greater time stability than the TTH1 variable. Separate analyses were necessary for the initial test and the follow-up test, because responder/nonresponder classification was not entirely consistent over time in either group. Nonresponder patients and normals did not differ in NS-SCR levels at either time point (.33 vs. .20 initially and .56 vs. .27 at follow-up). However, responder patients had significantly higher NS-SCR levels than responder normals at both time points (2.52 vs. 1.41 initially; $t = 2.24, df = 60, p < .03$; 3.19 vs. 1.47 at follow-up; $t = 2.46, df = 56, p < .02$).

Analysis of the average SCL yielded no effects that approached significance, nor did analysis of the SCL decline over time measure.

For all of the measures of responsivity (TTH1, TTH2, TTH3, NOR1, NOR2, and NOR3), neither the effect of group, time, or the interaction were significant. None of these measures differed significantly between the patient and normal groups at either test occasion. For the TTH1 measure obtained from the nontask tone series, which has been reported most often in the literature, the percentages of nonresponders in the patient and control groups at the initial outpatient test respectively were 52% and 48%, and at the follow-up test were 62% and 51%.

In addition to these variables of a priori interest, the remaining six EDA variables were also subjected to Group × Time ANOVAs. No significant effects were observed.

Discussion
The test–retest correlations obtained here for the normal subjects are consistent with previous reports in the literature, being particularly high for NS-SCR levels (Bull & Gale, 1973; Crider & Lunn, 1971; Freixa i Baque, 1982; Iacono et al., 1984; Lacey & Lacey, 1958; Schell et al., 1988; Siddle & Heron, 1976; Vossel & Zimmer, 1990). The 1-year follow-up correlations for rates of nonspecific responses and skin conductance levels clearly indicate that these measures of resting arousal are relatively stable traits of individuals, and the test–retest reliability for number of orienting responses elicited by the three series of stimuli indicates the same for these measures of allocation of processing resources to environmental stimuli. The significant reliability seen in these EDA measures is consistent with the considerable evidence for genetic input into these variables derived from both twin studies and from the increased incidence of abnormalities among relatives of schizophrenia patients (for twin studies of EDA responsiveness, see Lykken, Iacono, Haroian, McGue, & Bouchard, 1988).

There is no inconsistency in observing that EDA variables such as NS-SCRs and SCL may show significant stability among schizophrenia patients and still reflect (or even precede) symptomatic changes, just as it is not inconsistent to observe that these EDA variables show significant stability among normal subjects while still reflecting the action of environmental variables such as activating tasks or stressful stimuli (Boucsein, 1992, pp. 217–254, 261–292; Dawson et al., 2000). Nor is it inconsistent to observe that variables that reflect EDA responsivity to stimuli, which are

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arameters and part by transient states. For variables such as NS-SCRs and SCL, it may be the state aspects that are of greatest interest for schizophrenia, because (at least in this young, recent-onset population) they may predict short-term outcome in an already symptomatic group (Dawson, Nuechterlein, Schell, & Mintz, 1992) and possibly impending relapse or exacerbation in remitted patients (Hazlett et al., 1997).

One aspect of the present data of particular interest is the relative stability of different measures of EDA responsiveness to stimuli. When considering measures of responsiveness in the normal subjects, we observed a much higher test–retest correlation of NOR1 ($r = .64$), which is a simple count of the number of orienting responses to the nonsignal tones, than of TTH1 ($r = .19$), the trials-to-habituation measure that has frequently been used (Bernstein et al., 1982; Dawson & Nuechterlein, 1984; Dawson, Nuechterlein, & Schell, 1992; Iacono et al., 1993; Öhman, 1981). This was true in spite of the fact that the two measures intercorrelated highly both initially and at follow-up ($r = .70$ and .85, respectively). The probable reason for this, as discussed above, is the marked effect that the particular location of a single SCOR in a trial series can have on a trials-to-habituation measure such as TTH1.

The greater stability of the NOR1 measure is also manifested in the responder/nonresponder classification, which was more stable in the patient group when classification was based on NOR1 than when based on TTH1, owing partially to the smaller number of patients classified as nonresponders at both points. For the normal subjects, the method of responder/nonresponder classification did not strongly influence stability of classification or the number classified as nonresponders. The data indicate that erratic responding (failing to respond during the first three trials but then responding on a later trial or trials) is more common among the patients, which leads to patients being classified as nonresponders using the TTH measure but as responders using the NOR measure. This phenomenon of erratic “stop-and-go” responding among schizophrenia patients has also been noted by Zahn et al. (1981). These results suggest that NOR may be a more useful and reliable measure of responsiveness to innocuous, nonsignal stimuli than is a trials-to-habituation measure, and its use in future research is to be recommended. It should be noted, however, that this classification does not have as high a reliability (kappa = .36 for the normal subjects and .28 for patients) as do the tonic arousal measures and NOR1 taken as a complete count of number of SCORs to nontask stimuli.

Also important in these data are the tendencies toward lower correlations for patients than for normal subjects for the tonic arousal measures SCL and NS-SCRs, even among patients who are low and stable in symptomatology. Amount of change over time was significantly greater for NS-SCRs in the patients than the normal subjects. The significantly greater instability in NS-SCRs suggests poorer arousal modulation in the patients than in the normal subjects. The greater instability cannot be attributed to fluctuations in symptomatic state that may have occurred between the initial and follow-up measures, because the symptomatically stable subgroup of patients also showed reduced test–retest correlations relative to the normal subjects that were virtually identical to those shown by the entire patient group. Rather, the instability may reflect a more universal decreased ability on the part of the patients to regulate arousal. Consistent with poorer arousal regulation is our previous observation among these patients that when in a symptomatic state, not only is their SCL at rest higher than among normal subjects, indicating hyperarousal, but SCL does not decline over time during a resting period as it does among normal subjects, indicating poor arousal regulation (Dawson et al., 1994).

The present data further strengthen the case that electrodermal variables, both those that reflect general arousal and those that reflect responsivity to and processing of environmental stimuli, are moderately stable characteristics of individuals, albeit characteristics that also respond to transient environmental variables or (in the case of patients) degree of schizophrenic symptomology. The lower degree of stability seen among patients, even symptomatically stable patients, compared to normal subjects, particularly on the NS-SCRs variable, suggests that arousal regulation may be poorer among patients. Such diminished regulation may lead to broader fluctuations in arousal, as seen in Figure 1, which may be related to symptomatic exacerbation or relapse. Thus, EDA anomalies might play roles as vulnerability factors in schizophrenia and also as indicators of periods of increased exacerbation risk.

These recent-onset schizophrenia patients demonstrated higher levels of sympathetic arousal as reflected in their NS-SCR rate both initially and at follow-up. However, this difference occurs only for electrodermal responder subjects, consistent with previous findings (Dawson, Nuechterlein, & Schell, 1992). We have previously interpreted the elevated NS-SCR level in these patients as being a episode indicator for schizophrenia (Dawson et al., 1994), because it normalizes when patients are in remission. Presumably, the heightened NS-SCR levels seen in the present data reflect the degree of continuing symptomology seen both initially and at follow-up. This interpretation of the NS-SCR differences between patients and normal subjects is strengthened by the fact that at initial outpatient assessment and at follow-up NS-SCR levels were significant correlate with BPRS total scores, $r = .27$ and .26, respectively, $df = 68, p < .03$ one-tailed, whereas SCL measures were not. This suggests that NS-SCR levels may be more affected than is SCL by the low levels of symptomology seen in these patients, many of whom were free of symptoms in the clinical range on the days of testing.

Unlike many previous investigators (see reviews by Bernstein et al., 1982; Dawson & Nuechterlein, 1984; Iacono et al., 1993; Öhman, 1981), we did not observe differences between the groups in SCOR responsivity. None of the trials-to-habituation or number of SCOR measures differed significantly between the groups at either test point. As can be seen in Table 3, nonresponder as measured by TTH1 was near or above 50% in both groups at both test occasions. We have suggested previously (Dawson, Nuechterlein, & Schell, 1992) that the lack of a group difference with respect to the TTH1 measure is due to an unexpectedly high rate of nonresponsiveness in the normal group, which might have been due to the normal group being matched to the patient group with respect to gender, age, and education. This resulted in a predominantly young male group with only a high school education, which is not typical of the control groups usually reported in the electrodermal literature. Iacono et al. (1993) have argued that the nature of the control group may strongly affect the outcome of studies of relative responsiveness of schizophrenia patients and normals. Studies using hospital staff as controls have reported a lower rate of control nonresponsiveness than have studies such as the present one using a community-based demographically matched control group. This is clearly an issue that warrants further investigation.
The failure of the normals in the present study to be more responsive than the patients during the task-significant tone series and the loud noise series, as they were during an inpatient test (Dawson, Nuechterlein, & Schell, 1992), is due to greater habituation from the inpatient test to the two outpatients tests reported here among the normals. Patients show little or no decline in any measure of responsiveness across the inpatient and two outpatients tests, whereas the normal group shows substantial decline in responsiveness to the task-significant tones and loud noises.

Even though responsivity measures may not consistently distinguish schizophrenia patients from matched normal groups, and the responder/nonresponder classification may be low in reliability, responsivity measures may nonetheless be of use in predicting the course of schizophrenia. Brekke, Raine, Ansel, Lencz, and Bird (1997), Katsanis and Iacono (1994), Öhman et al. (1989), and Wieselgren, Öh Lund, Lindström, and Öhman (1994) have reported that the responder/nonresponder distinction has implications for functional outcome in schizophrenia, and we have also found this to be true in this group of patients with respect to the NOR1 measure taken at the first outpatient test (Rissling, Schell, Dawson, & Nuechterlein, 2000). Therefore, SCOR responsivity measures, even when they fail to differ from a control group, may still be of use in identifying important subgroups of schizophrenic patients (e.g., in predicting outcome).

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