Journal of Autism and Developmental Disorders, Vol. 14, No. 3, 1984

The Electrodermal Orienting Response to Auditive Stimuli in Autistic Children, Normal Children, Mentally Retarded Children, and Child Psychiatric Patients[1]

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Spontaneous electrodermal activity as well as electrodermal orienting responses to auditive stimuli of moderate intensity were recorded in a group of 35 autistic children and in three control groups. Mean number of spontaneous fluctuations in skin conductance did not differentiate the groups. Autistic children, as compared with the children from the control groups, were significantly more often nonresponsive to the first trial. When responding, autistic children showed electrodermal orienting responses characterized by large amplitudes and fast recovery. The functional significance of nonresponding is discussed, and it is concluded that the response pattern of the autistic child shows a striking resemblance to that of the electrodermal response of schizophrenic persons.

One of the outstanding disorders of autistic children is their inability to relate themselves in the ordinary way to people and situations. Their parents refer to them as having always been "as if in a shell," "acting as if people weren't there," "happiest when left alone," and "perfectly oblivious to everything about them."

From the very beginning of life there is an extreme autistic aloneness that, whenever possible, disregards, ignores, and shuts out anything that

[1]I am grateful to Prof. Dr. L. N. J. Kamp and Prof. Dr. J. L. Slangen for their constructive criticism on earlier drafts of this article, as well as to Dr. R. Verbaten for his help in analyzing the data.

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0162-3257/84/0900-0261$03.50/0

comes to the child from outside (Kanner, 1943). The autistic child seems to be closed to environmental stimuli. Unlike those of most normal children, the autistic child's verbalizations cannot be relied upon for obtaining data on responsiveness or on preference for certain types of stimuli. The contributions of psychological motives in the autistic child's unresponsiveness to sensory stimuli thus remains obscure.

The hypothesis that the peculiar manner in which autistic children handle people and situations is mainly due to physiological abnormalities in responsiveness and selective attention has been a prominent theoretical issue in the last decade. Various hypothetical deficits have been proposed. Hermelin and O'Connor (1970), on the basis of an extensive series of laboratory studies, concluded that children suffering from the autistic syndrome display impaired ability to integrate input and output data and to process information from various sources. A number of other researchers have suggested speculative physiological bases for this ability to process sensory information (Des Lauriers & Carlson, 1969; Hauser, DeLong, & Rosman, 1975; Simon, 1975). Ornitz (1970, 1971, 1974) postulated a failure of adequate vestibular modulation of sensory input and motor output. Rimland (1964, 1968) suggested that malfunction of the reticular activating system (RAS) resulted in underarousal to incoming stimuli, with consequent inability to relate new to remembered experience. In contrast, Hutt and Hutt (1968) postulated that a chronically high level of RAS activity produces blocking of senory pathways.

So far, empirical research in the neurophysiology of childhood autism has produced rather conflicting results. In a recent review, James and Barry (1980) conclude that no characteristic of CNS functioning can be unequivocally related to infantile autism; the single trend to emerge from the data is that children suffering from this syndrome show responses that are different from those of normal children. The nature and direction of these differences can only be surmised.

Part of the discrepant research findings can be ascribed to the fact that collection of reliable electrophysiological data from young psychotic children is time-consuming and fraught with difficulties. In view of the minimal behavioral compliance required, registration of electrodermal activity appears to be an appropriate method for collecting physiological data on a subject's responsiveness to sensory stimulation. Electrodermal activity has therefore been widely studied in schizophrenic populations (Bernstein, 1964, 1970; Gruzelier & Venables, 1972, 1974, 1975; Venables, 1963, 1966, 1977) as well as in children "at risk" for schizophrenia (Mednick & Schulsinger, Mednick et al., 1968; 1973; Venables, 1977).

A well-developed body of research has shown that the electrodermal orienting response (EOR) is integrally linked with the registration of

stimulus input. Subjects who are extremely open to environmental stimuli and prone to sensory overload will respond with EOR's characterized by large amplitude, rapid recovery, and slow habituation (Venables, 1973, 1975). Recovery rate appeared to be a particularly sensitive indicator of pathological openness to the environment (Venables & Fletcher, 1981).

So far, three reports on studies of electrodermal activity in infantile autism have been published. Bernal and Miller (1971) measured skin conductance and compared 20 "autistic schizophrenic" children with a control group of 20 normal children of the same chronological age. No group difference in resting skin conductance level, spontaneous activity, differential responsivity to auditory and visual stimuli, or latency of evoked skin conductance responses were found. The autistic group was differentiated from the normal group on the basis of a lower mean response during the first block of three habituation trials; thereafter, both groups fell to a common response level and there were no further differences in habituation patterns. White (1974) reported a study comparing conditioning and generalization of skin conductance responses to tones in four groups of children: "nonorganic schizophrenic," "organic schizophrenic," "minimal brain dysfunction," and "normal." The "schizophrenic" children, regardless of their neurological status, gave progressively larger responses to a repeated tone, whereas responses progressively diminished in the control group. Latency measures indicated that the "schizophrenic" group displayed longer latencies during blocks 2 and 3 and shorter latency during the first block only. Palkovitz and Wiesenfeld (1980) found that a group of 10 autistic subjects displayed significant higher resting skin conductance levels and more spontaneous skin conductance responses than a group of 10 normal subjects of the same chronological age, whereas the overall magnitude of electrodermal rsponses to auditive stimuli failed to differentiate the groups. Due to differences in diagnostic terminology, differences in the way the subjects were stimulated, and insufficient matching on important variables such as mental age and use of medication, it is impossible to give a clear interpretation of the discrepant findings of the three studies just mentioned.

The first purpose of this study is to investigate electrodermal responsiveness to auditive stimuli in autistic children as compared with normal children, nonpsychotic child psychiatric inpatients, and mentally retarded children. Proceeding from the Venables concept of "openness to the environment," we assume autistic children to be in a pathological way "more closed" than other children. It is therefore predicted that autistic children will exhibit electrodermal responses with small mean amplitude, long mean recovery, and rapid habituation.

Several authors have suggested that, particularly in autistic children, responsiveness to environmental stimuli is ultimately influenced by cortical

and autonomic activation levels (Hutt & Hutt, 1970; Hutt, Forest, & Richer, 1975; Ornitz, 1974; Des Lauriers & Carlson, 1969). The number of spontaneous fluctuations in skin conductance level can be considered to be an activation index of the electrodermal system (Depue & Fowles, 1976), and recently several investigators (Bohlin, 1973; Thayer & Silber, 1971; Bundy & Fitzgerald, 1975; Edelberg & Muller, 1981; Venables & Fletcher, 1981) have found that in *normal* subjects amplitude as well as recovery rate of the skin conductance response covary with the number of spontaneous fluctuations in conductance. One can wonder whether this covariance between the activation level of the electrodermal system and the electrodermal responsiveness is the same for normal subjects as well as for subjects suffering from severe psychopathology. So the second aim of this study is to investigate and to compare this covariance phenomenon in autistic children, child psychiatric patients, mentally retarded children, and normal children, starting out from the prediction that the covariance between the number of spontaneous fluctuations and EOR variables will be similar for all groups.

METHOD

Subjects

The composition of the autistic group and the three control groups is shown in Table I. None of the children was hearing-disabled; none used psychotropic drugs. Tests were started after written parental permission had been obtained.

When examined, the autistic children were all inpatients at one of the child psychiatric centers in the Netherlands. Like Rutter (1978), we applied the following criteria in order to establish the diagnosis of infantile autism: (a) profound disturbance of emotional contact with people, (b) retardation in speech and language development, (c) abnormal behavioral rigidity such as "insistence on sameness" or preoccupations, and (d) age of onset less than 36 months.

Table I. The Composition of the Autistic Group and the Three Control Groups

				Chronological age		Performance IQ	
Groups	*N*	♀	♂	*Mean*	*SD*	*Mean*	*SD*
Autistic	35	9	26	8.9	3.2	71	19.3
Normal	45	9	36	8.8	2.0		
Child psychiatric patients	38	6	32	9.9	1.5	96	9.4
Mentally retarded	20	2	18	18.2	4.4	48	11.2

The case records showed that all the autistic children had been observed in detail during daily life; all had been submitted to neurological examination; detailed development histories were available and, although a wide variety of intelligence tests proved to have been used, a performance IQ had recently been established for all children. The records of the autistic children were analyzed by an independent judge, an experienced child psychiatrist. The mean age at onset was 28 months; 12 autistic children were mentally retarded (mean performance IQ = 47, *SD* = 6.2), 11 autistic children were mildly retarded (mean performance IQ = 69, *SD* = 5.3), and 12 autistic children were functioning at or above a low normal intellectual level (mean performance IQ = 91, *SD* = 6.5). Five children in the autistic group were suffering from epilepsy, two showed marked abnormalities at neurological examination, and six showed soft neurological signs. None suffered from a progressive neurological condition. The autistic children averaged 6.6 (*SD* = .9) positive scores out of the nine criteria of Creak's working party (1961). The normal children were sampled from a primary school in Utrecht. The nonpsychotic psychiatric patients were inpatients at the University of Utrecht Department of Child Psychiatry. Some 20 of them were considered to suffer from "emotional disorders," 15 were labeled with "conduct disorders," and 3 were diagnosed as "mixed disorders" (Rutter, 1978). All had a performance IQ above 85. The mentally retarded children were all suffering from Down's syndrome and were inmates of a home for mental defectives at the time of the study.

Apparatus and Physiological Recording

Skin conductance was recorded at the institute where the child was staying, in a room that was familiar to him/her, and in the presence of an adult the child knew (mother, group leader). The recording apparatus was set up in an adjacent room, and the test subject could neither see nor hear it. During recording, silence was observed in the examining room as well as in adjacent rooms and corridors. Skin conductance was measured with the aid of the constant voltage method described by Lykken and Venables (1971). Siemens electrodes for children AG-AgCl (type 4399556) were applied to the palmar side of the middle phalanx of the index finger and ring finger of the left hand; .5% potassium chloride electrolyte was used. Via a skin conductance meter (Lykken & Venables, 1971), the electrodes were connected with a W + W − 314 recorder. Through bilateral headphones (type Kenwood K-H-32) the child was offered 24 acoustic stimuli of 85 dB, 1,000 cps, 1 sec duration, at random intervals ranging from a minimum of 10 seconds to a maximum of 35 seconds.

Scoring of Physiological Activity and Statistical Analysis

Changes in skin conductance were scored by hand, applying the following criteria:

1. *Spontaneous* fluctuations are changes in skin conductance that exceed .05 micro-mho (μmho), so far as they do not occur within 1-5 seconds of the start of the stimulus. The number of spontaneous fluctuations were scored in the 5-minute period prior to auditive stimulation.
2. The term *response* applies when a change in skin conductance that exceeds .05 μmho occurs within 1-5 seconds after the start of the stimulus.
3. *Amplitude* is the difference between the skin conductance level prior to stimulation and the skin conductance at the peak of the response, expressed in μmho.
4. *Latency* is the time lapse between the start of acoustic stimulation and the moment at which conductance is .05 μmho above the initial level.
5. *Recovery time* is the time (in seconds) required to reduce the skin conductance level of the responsive peak to half its amplitude.
6. *Recovery rate* is half the amplitude divided by the recovery time.
7. The *rate of habituation* is expressed in terms of the number of stimuli required until the test subject fails to respond to three consecutive stimuli.

During the trials the temperature and relative humidity in the examining room were recorded. The mean temperature was 20.6° C (SD = 1.75), and the relative humidity was 40.8% (SD = 2.9).

In this study each child received several stimuli in succession. The EOR's induced by these stimuli are not independent of each other, and this implies that the study can be characterized as a repeated-measures design. Consequently, the EOR parameters amplitude, latency, recovery time, and recovery rate were submitted to analysis of multivariance (Finn, 1976). The mean rate of habituation per group was compared by means of a t-test; the responsiveness to S_1 was analyzed with aid of the chi-square test for independent samples. For all statistical analysis, α = .05.

Procedure

To begin with, the child was given 15 minutes during which he could play with the toys in the examining room, talk with the test leader, and reconnoiter the room. After this he/she was invited to sit in a chair behind a table and was connected to the recording apparatus. Before placing the ear-

phones on the child's head, the test leader gave the following instructions:

> I would like you to sit as still as you can, and especially not move your left hand. You may look at your mother [or whoever is present as familiar person], but you must not talk with each other. You may use your right hand to turn the pages of the book on the table when reading or looking at the pictures; or, if you like, you may play with the puzzle on the table, but you may use your right hand only. These wires are to show how many drops of sweat your fingers make. They do not hurt. You won't feel anything. Because we do not want you to be bothered by noise, we'll put these earphones on your head. You may now and then hear a beep in them, but that does not mean anything and you need not pay attention to it.

After this the actual recording began. After an adaptation period of 5 minutes, 24 acoustic stimuli were offered, followed by another adaptation period of 5 minutes to conclude the recording.

RESULTS

Visual inspection of the data taught us that the mean EOR indices of the various groups differed only over the trials S_1 through S_8. Therefore, only the data on the first eight trials are presented and analyzed. First, the number of spontaneous fluctuations, responsiveness, habituation rate, and EOR indices of the four groups will be compared. Finally, the covariance between the number of spontaneous fluctuations before stimulation and the EOR variables in each group will be investigated.

Comparison of the Groups

Spontaneous Fluctuations. No differences between the groups were found (autistic children: $\bar{x} = 6.25$, $SD = 5.05$; normal children: $\bar{x} = 7.94$, $SD = 4.62$; child psychiatric patients: $\bar{x} = 7.33$, $SD = 3.84$; and mentally retarded children: $\bar{x} = 5.08$, $SD = 3.71$).

Responsiveness. According to Patterson and Venables (1978), the population was divided into four subgroups: *nonresponders,* who did not respond at all to the first trial; *fast habituators,* who habituated within two trials; *habituators,* who were habituated at trial 8, and *nonhabituators* (see Figure 1). The autistic group was differentiated from the normal group, the child psychiatric patients, and the mentally retarded children on the basis of a significantly greater number of nonresponders (chi square 8.7, $df = 1$, $p < .01$; chi square 7.5, $df = 1$, $p < .01$; and chi square 5.1, $df = 1$, $p < .05$, respectively) (see Figure 1).

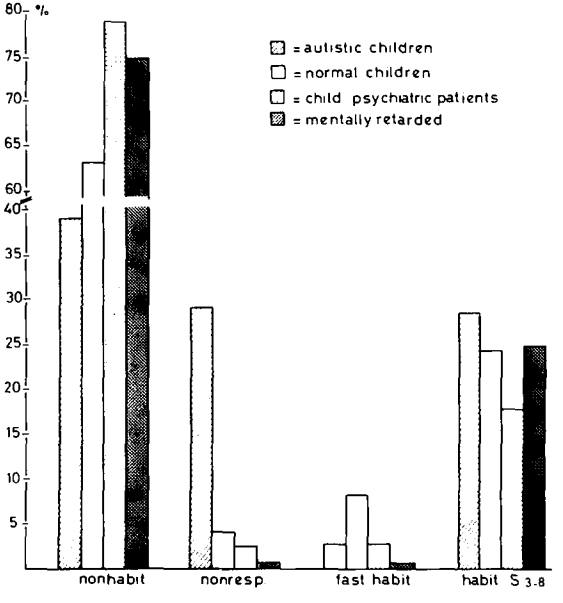

Figure 1. The percentage of nonresponders, fast habituators, habituators, and nonhabituators in the four groups.

Habituation Rate. The mean habituation rates of the groups did not differ.

EOR Indices. Table II offers a survey of the means and standard deviations of the amplitude, latency, recovery time, and recovery rate of the groups on each trial. Analysis of multivariance (Finn, 1976) did not reveal any significant difference between the groups.

Covariance Between Spontaneous Activity and EOR Variables

In order to investigate the covariance between the number of spontaneous fluctuations before stimulation and the EOR variables, each group was split into a subgroup with a high number of spontaneous fluctuations (H) and a subgroup with a low number of spontaneous fluctuations (L). It was decided to take the mode of the number of spontaneous fluctuations in the whole population ($N = 138$) as a cutoff point. So the children with six (= the mode) or more spontaneous fluctuations a minute belonged to the H groups; children with fewer than six spontaneous fluctuations a minute belonged to the L groups. Table III shows the definitive composition of the groups after dichotomization in L and H.

Table II. Survey of the Means and Standard Deviations of Amplitude, Latency, Recovery Time, and Recovery Rate of the Groups on Each Trial

	Autistic children		Normal children		Child psychiatric patients		Mentally retarded children	
EOR indices	$\bar{X}$	*SD*	$\bar{X}$	*SD*	$\bar{X}$	*SD*	$\bar{X}$	*SD*
Amplitude								
S_1	2.43	3.06	2.03	1.97	2.21	2.27	1.99	1.00
S_2	1.02	1.23	1.17	1.18	.73	1.16	.95	.83
S_3	.61	.96	.66	.82	.63	.64	.49	.50
S_4	.83	.90	.69	.93	.84	.72	.58	.53
S_5	.56	.65	.46	.62	.75	1.06	.62	.62
S_6	.71	.95	.37	.43	.38	.42	.38	.40
S_7	.66	.68	.29	.45	.67	.88	.59	.75
S_8	.77	.96	.38	.47	.45	.70	.22	.32
Latency								
S_1	1.07	1.02	1.61	.72	1.56	.67	1.63	.55
S_2	1.25	1.24	1.15	.86	1.16	1.26	1.13	1.05
S_3	1.35	1.52	1.17	1.04	1.18	1.13	1.32	1.41
S_4	1.37	1.19	1.10	1.02	1.67	.90	1.50	1.05
S_5	1.47	1.50	.90	.94	1.06	1.16	1.40	1.16
S_6	.92	1.10	1.25	1.24	1.45	1.29	1.21	1.03
S_7	1.19	1.10	.80	1.05	1.25	1.08	1.20	.96
S_8	.92	1.11	1.23	1.23	.99	1.11	.85	1.18
Recovery time								
S_1	4.90	4.58	6.79	3.84	6.11	3.88	8.47	2.73
S_2	2.94	3.64	4.55	4.08	2.13	2.72	3.25	3.83
S_3	2.19	3.58	3.28	3.67	2.04	2.73	2.69	3.59
S_4	1.76	2.43	2.45	3.31	2.49	2.54	2.79	3.00
S_5	2.06	3.26	2.97	2.73	1.37	1.86	2.22	1.99
S_6	1.63	2.77	1.92	2.70	2.12	2.80	2.51	3.06
S_7	2.73	3.71	1.73	3.01	2.07	2.65	2.69	3.08
S_8	1.85	3.20	1.79	2.59	1.45	2.52	1.72	3.06
Recovery rate								
S_1	.20	.28	.23	.41	.24	.25	.15	.15
S_2	.28	.44	.18	.26	.15	.20	.22	.26
S_3	.20	.35	.16	.28	.18	.22	.12	.17
S_4	.28	.36	.18	.29	.27	.26	.15	.18
S_5	.20	.28	.10	.15	.19	.23	.14	.13
S_6	.21	.34	.10	.13	.11	.19	.09	.12
S_7	.16	.22	.07	.12	.14	.17	.11	.14
S_8	.23	.37	.12	.16	.14	.22	.06	.09

For each of the eight subgroups, responsivity, habituation rate, and EOR indices on each trial were computed. Table IV shows the means and standard deviations of the EOR indices for each subgroup.

In the autistic group, responsivity on the first trial (chi square 50.7, $df = 1$, $p < .0001$) of the H subgroup exceeded that of the L subgroup; habituation was slower ($t = 2.93$, $df = 3$, $p < .01$); amplitudes were larger ($F = 5.10$, $df = 1, 33$, $p = .031$); recovery times were shorter ($F = 9.90$, df

Table III. Composition of the Groups After Dichotomization in L and H

Groups	N	Spontaneous fluctuations		Chronological age	
		Mean	*SD*	*Mean*	*SD*
Autistic H	21	9.9	4.4	8.6	2.6
Autistic L	14	2.8	1.9	9.0	3.4
Normal H	27	9.2	3.3	8.2	1.6
Normal L	18	3.4	1.9	8.7	1.9
Child psychiatric patient H	25	9.5	3.0	9.8	1.8
Child psychiatric patient L	13	2.3	1.4	10.3	1.8
Mentally retarded H	8	9.5	2.9	20.1	1.8
Mentally retarded L	12	3.2	1.9	18.7	2.5

= 1, 33, p = .003); and recovery rates exceeded those of the L subgroup (F = 10.29, df = 1, 33, p = .003).

In the group of normal children, as well as in the child psychiatric group, the subgroups H and L differed on two variables. In both groups, recovery times for H were shorter than those for L (F = 18.27, df = 1, 43, p = .002 and (F = 5.03, df = 1, 36, p = .23, respectively) and recovery rates of H exceeded those of L (F = 6.15, df = 1, 43, p = .017 and (F = 7.94, df = 1, 36, p = .007, respectively).

In the mentally retarded group, only recovery time appeared to be shorter for H (F = 7.05, df = 1, 18, p = .016).

In summary, comparing subgroup H with subgroup L in autistic chlidren, five out of six EOR variables differ significantly, whlie in other children differences are found for two EOR variables at the utmost. Contrary to our prediction, it must be concluded that the number of spontaneous fluctuations covaries with EOR variables in the various groups in different ways.

In our opinion, these findings have implications for the way in which EOR variables of various groups of children should be compared. If in autistic children electrodermal orienting responses under conditions of extreme high or extreme low spontaneous activity behave in another way than the EORs of other children do, the way in which we compared the mean EOR indices of the groups (see above) might have produced false positive or false negative results! So we decided to reanalyze our data and compare the mean EOR variables of the four groups under H as well as under L. (Table III shows the composition of the subgroups.)

EOR Variables Compared Under L

Only one variable differentiated the various L subgroups: The autistic L group was significantly more frequently nonresponding on the first trial than the normal L group (chi square 4.12, $df = 1$, $p < .05$), the child psychiatric L group (chi square 5.83, $df = 1$, $p < .02$), and the mentally retarded L group (chi square 5.98, $df = 1$, $p < .02$).

EOR Variables Compared Under H

No differences in responsivity and habituation rate between the H subgroups were found. Amplitude differentiated the autistic children from the mentally retarded ($F = 5.20$, $df = 1, 27$, $p = .16$); latency differentiated the autistic children from the normal children ($F = 6.15$, $df = 1, 47$, $p = .017$). Mean recovery rate of the autistic group exceeded that of the normal group ($F = 9.16$, $df = 1, 45$, $p = .004$), that of the child psychiatric patients ($F = 5.85$, $df = 1, 44$, $p = .019$), and that of the mentally retarded group ($F = 6.57$, $df = 1, 27$, $p = .016$).

Summarizing the results of the comparison of the mean EOR variables of the four groups, we conclude that the response pattern of the autistic children differs from that of all the other children in two ways; Under L, autistic children are more often nonresponding at the first trial, and under H, they show a faster recovery rate of the EOR.

DISCUSSION

We mentioned already that several authors have speculated about the arousal levels of autistic chlidren. Some authors suggest that autistic children are chronically hyperaroused (Hutt & Hutt, 1968). Others state that at least some of the autistic children are hypoaroused (Des Lauriers & Carlson, 1969). Empirical research on this issue has produced conflicting results, mainly because operationalizing the concept of arousal into measurable parameters has many pitfalls (Lacey, 1967). If it is justifiable to use a number of fluctuations as an index of arousal of the electrodermal system, it must be concluded that our data don't support either the hyper- or hypoarousal hypothesis: The number of spontaneous fluctuations in skin conductance does not differentiate the four groups.

Recently several authors have studied the way in which adult schizophrenic patients electrodermally respond to auditive stimuli, using an ex-

Table IV. Survey of the Means and Standard Deviations of Amplitude, Latency, Recovery Time, and Recovery Rate of the Subgroups on Each Trial

	Autistic children				Normal children				Child psychiatric children				Mentally retarded children			
	L		H		L		H		L		H		L		H	
EOR indices	*X*	*SD*	*X*	*SD*	*X*	*SD*	*X*	*SD*	*X*	*SD*	*X*	*SD*	*X*	*SD*	*X*	*SD*
Amplitude																
S_1	1.08	1.35	3.38	3.51	1.73	1.80	2.25	2.09	1.93	1.66	2.35	2.55	2.07	1.24	1.87	.52
S_2	.56	.50	1.33	1.47	.74	.78	1.48	1.32	.84	.91	.67	1.28	1.09	.92	.75	.67
S_3	.15	.25	.92	1.13	.37	.47	.87	.95	.48	.62	.70	.65	.44	.52	.57	.51
S_4	.43	.69	1.10	.94	.43	.63	.87	1.07	.72	.69	.98	.74	.71	.59	.39	.37
S_5	.44	.55	.64	.72	.27	.43	.68	.70	.38	.58	.95	1.21	.64	.73	.60	.43
S_6	.48	.78	.87	1.04	.37	.45	.37	.43	.29	.35	.43	.45	.27	.38	.55	.38
S_7	.63	.64	.64	.72	.23	.45	.33	.46	.31	.56	.85	.97	.47	.82	.75	.64
S_8	.52	.91	.94	.98	.21	.28	.50	.55	.26	.36	.55	.81	.17	.26	.30	.39
Latency																
S_1	.83	.93	1.23	1.06	2.00	.84	1.34	.49	1.86	.65	1.48	.63	1.75	.67	1.46	.25
S_2	1.13	1.30	1.33	1.22	1.24	.99	1.09	.77	1.99	1.42	.73	.93	1.13	1.05	1.12	1.11
S_3	.78	1.23	1.79	1.56	1.11	.98	1.21	1.09	.74	.76	1.48	1.23	1.15	1.25	1.56	1.69
S_4	1.13	1.45	1.53	.99	1.04	1.08	1.14	1.00	1.89	.95	1.36	.87	1.53	.89	1.46	1.31
S_5	1.39	1.66	1.52	1.43	.80	.88	.98	.99	.91	.99	1.14	1.25	1.22	1.28	1.66	.96

S_6	.73	1.01	1.04	1.16	.93	.99	1.48	1.36	1.28	.97	1.53	1.44	.96	.93	1.58	1.12
S_7	1.07	1.28	1.26	1.06	.64	1.05	.91	1.06	.87	1.01	1.44	1.07	1.13	1.18	1.29	.57
S_8	.58	.73	1.20	1.24	1.16	1.38	1.28	1.14	1.07	1.13	.94	1.11	.96	1.42	.69	.76
Recovery time																
S_1	4.34	4.75	5.27	4.53	8.17	3.14	5.83	4.04	6.81	3.88	5.74	3.91	9.24	2.35	7.38	3.00
S_2	4.10	4.43	2.17	2.87	5.81	4.51	3.68	3.68	3.17	2.19	1.59	2.85	4.64	4.40	1.16	1.04
S_3	2.23	4.05	2.17	3.34	3.93	3.93	2.83	3.49	2.60	3.40	1.75	2.33	3.58	4.37	1.48	1.49
S_4	1.87	2.79	1.69	2.23	3.61	4.35	1.65	2.08	3.83	3.11	1.79	1.91	3.17	3.27	2.23	2.67
S_5	3.74	4.64	.93	.91	2.72	3.71	1.61	1.71	1.23	1.40	1.45	2.08	2.17	2.12	2.38	1.91
S_6	2.48	4.14	1.06	1.03	2.56	3.23	1.47	2.21	2.26	2.68	2.05	2.92	2.48	3.13	2.56	3.16
S_7	3.92	4.44	1.93	3.00	2.27	4.01	1.35	2.08	1.09	2.04	2.58	2.82	2.75	3.18	2.68	3.12
S_8	1.78	3.56	1.94	3.02	1.83	3.15	1.76	2.19	2.28	3.24	1.03	2.00	2.22	3.79	.97	1.37
Recovery rate																
S_1	.05	.08	.30	.33	.22	.55	.24	.29	.15	.12	.28	.28	.15	.18	.15	.10
S_2	.17	.41	.35	.46	.08	.16	.26	.30	.16	.21	.14	.20	.19	.27	.27	.24
S_3	.02	.06	.32	.42	.04	.07	.24	.34	.08	.13	.24	.24	.06	.10	.20	.22
S_4	.08	.15	.42	.40	.07	.16	.26	.34	.16	.19	.33	.28	.18	.22	.10	.09
S_5	.07	.14	.28	.32	.03	.07	.15	.18	.08	.11	.24	.25	.11	.13	.18	.13
S_6	.08	.15	.30	.41	.06	.09	.13	.14	.05	.09	.13	.22	.05	.09	.14	.13
S_7	.09	.14	.21	.25	.03	.07	.09	.14	.10	.20	.16	.15	.07	.14	.17	.14
S_8	.08	.15	.32	.44	.08	.17	.14	.15	.05	.09	.18	.25	.04	.08	.08	.10

perimental procedure that is comparable with the procedure we described above. Bernstein and Taylor (1979), Bernstein et al. (1981), and Patterson and Venables (1978) found that 23 to 70% of the schizophrenic population did not respond electrodermally to auditive stimuli of mild or moderate intensity. This applied to both chronic and acute patients regardless of drug status or sex. The studies of Ax and Bamford (1970), Gruzelier and Venables (1972), and Zahn, Carpenter, and McGlashan (1976) revealed shortness of skin conductance response *recovery* to be typical of schizophrenic patients as well as of children "at risk" for schizophrenia (Mednick et al., 1978).

In our opinion, the response pattern of autistic children shows some resemblance to the way schizophrenics respond electrodermally to auditive stimuli. However, most of the studies just mentioned investigated patients with IQ scores within the normal range, while the mean IQ score of the autistic children in our population was at a subnormal level. White (1974) made clear that some EOR variables covary with IQ scores, so definitive conclusions about similarities between autistic children and schizphrenic patients cannot be drawn. In order to solve this problem, we selected from our original population a subgroup of autistic children with IQ scores of 80 and above. The subgroup consisted of 12 autistic children with a mean performance IQ score of 91 ($SD = 6.5$). Five of them belonged to the L subgroup and 7 belonged to the H subgroup. Analysis of the EOR data of the subgroup of autistic children with IQ scores within a normal range revealed the following results:

1. Sixteen percent of the autistic population did not respond to the first trial.
2. The autistic group was differentiated from the normal children as well as from child psychiatric patients on mean *amplitude* under H ($F = 7.37$, $df = 1, 32$, $p = .011$ and ($F = 4.89$, $df = 1, 30$, $p = .035$, respectively).
3. The autistic group was differentiated from the normal children as well as from child psychiatric patients on mean *recovery rate* under H ($F = 7.24$, $df = 1, 32$, $p = .011$ and ($F = 5.48$, $df = 1, 30$, $p = .026$, respectively) as well as under L ($F = 6.49$, $df = 1, 21$, $p = .019$ and ($F = 5.89$, $df = 1, 16$, $p = .028$, respectively).
4. Mean *recovery time* of the autistic group under L was shorter than that of the normal group ($F = 7.27$, $df = 1, 21$, $p = .014$).

Although several investigators have suggested that childhood autism may be the earliest manifestation of schizophrenia (Goldfarb, 1961; Ornitz, 1969), at this moment most authors maintain that childhood autism is not the same disease or cluster of diseases as schizophrenia), but rather is a

clearly circumscribed nosological entity (Kolvin, 1971; Rutter, 1972, 1978). Nevertheless, some authors (James & Barry, 1980) have recently suggested that early onset psychosis and schizophrenia have in common an abnormal neurophysiological reactivity to stimuli expressed in the activity of phasic autonomic response mechanisms. Our findings support these speculations: Autistic children and schizophrenics at least show a remarkable resemblance in the way they respond electrodermally to auditive stimuli of moderate intensity.

At the start of this study it was predicted that autistic children would show EORs characterized by small mean amplitude, long mean recovery, and rapid habituation, suggesting that these children are in a pathological way "closed" for environmental stimuli. These predictions have been refuted by the results obtained. When the groups are matched optimally, and autistic children with IQ scores of 80 and above are compared with normal children and with child psychiatric patients, it becomes clear that mean recovery rate of the autistic group exceeds those of both other groups under H as well as under L. These results suggest that autistic chldren, particularly when their electrodermal system is highly activated, are abnormally open to environmental stimuli, supporting the hypothesis of Bergman and Escalona (1949) that children suffering from childhood psychosis are unusually sensitive to all kinds of stimuli and prone to become sensorily overloaded.

The functional significance of nonresponding at the first of a series of trials is still mainly a matter of speculation. Some researchers hold that nonresponsiveness indicates temporal lobe and limbic dysfunction (Gruzelier & Venables, 1972). However, several reports have demonstrated that schizophrenics, electrodermally unresponsive to a given innocuous stimulus, routinely become responsive if the same stimulus is made explicitly significant for them (Bernstein & Taylor, 1979; Gruzelier & Venables, 1973). On the basis of these observations, Bernstein postulated that nonresponsiveness reflects a difference in the way subjects evaluate the significance of stimuli; schizophrenics in particular allocate their attention differently than normal subjects.

Gruzelier and Venables (1973) reported that in normal subjects about 7% of the sample is found to be nonresponding, and the authors suggested that only nonresponsiveness coincident with abnormally low spontaneous electrodermal activity is indicative of pathology. Bernstein et al. (1981) recently discovered nonresponsiveness to be related to severity of psychopathology: "Nonresponding" schizophrenics showed more cognitive disorganization and more emotional withdrawal than responding schizophrenics.

In our study neither severity of psychopathology, operationalized in age of onset and number of positive scores on the Creak items, nor

chronological age, sex, performance IQ, and neurological symptomatology differentiated "nonresponding" autistic from other autistic children. Further analysis of our data indicated that "nonresponding" autistic children showed fewer spontaneous fluctuations in skin conductance before stimulation than the other autistic children ($t = 2.76$, $df = 33$, $p < .01$, two-tailed). Investigation of the responsiveness on trials 2 to 8 revealed that "nonresponding" autistic children showed a tendency to be hyporesponsive over those trials, as compared with other children matched on spontaneous electrodermal activity prior to stimulation. This tendency became statistically significant when autistic children nonresponsive at the first three trials ($n = 5$) were compared on responsiveness to trials 4 through 8 with other autistic children ($t = 2.19$, $df = 12$, $p < .05$, two-tailed), with child psychiatric patients ($t = 2.34$, $df = 17$, $p < .05$, two-tailed), with normal children ($t = 3.80$, $df = 15$, $p < .001$, two-tailed), and with mentally retarded children ($t = 4.55$, $df = 15$, $p < .001$, two-tailed), all matched according to spontaneous electrodermal activity prior to stimulation.

In view of these data, we assume "nonresponding" children to be abnormally inhibited in spontaneous as well as in provoked electrodermal reactivity, and we wonder whether this inhibition is a stable "trait" of the child concerned. Gruzelier and Venables (1972) regard "nonresponding" to be a "state-dependent" phenomenon, accompanying periods of inactive behavior of schizophrenic patients. In a pilot study in which we registered skin conductance reactivity to auditive stimuli on 3 consecutive days in 5 autistic children and 9 normal children (mean chronological age 10.8 years, performance IQ at or above normal level), we found that 6 out of 14 children (2 autistic children and 4 normal children) were nonresponding at one registration day and normal-responsive at another day. Apparently "nonresponding" is not a stable trait of the child concerned, and we support Gruzelier's opinion: "Nonresponding" should be considered a state-dependent phenomenon.

Several authors have described such a state of abnormal response inhibition occurring in human subjects overloaded with sensory stimuli. Lynn (1963), Jordan (1974), Buchsbaum (1976), Keuss and Orlebeke (1977), and Venables (1977) referred to this phenomenon as a "paradoxical reaction" that protects the organism from being overwhelmed by stimulation. We could imagine that such a paradoxical reaction produces "nonresponsiveness" to the first trial and, when inhibition increases, also to the first three or even to all trials.

If it is justifiable to regard "nonresponding" as the result of a paradoxical reaction, the following speculative hypothesis presents itself: When highly physiological aroused, autistic children are abnormally open to environmental stimuli and exposed to a stressing condition of information overload. The development of "nonresponsiveness" can then be explained

as a defensive strategy of the child minimizing the overload stress and at a behavioral level, resulting in a state of aloofness in which the child disregards, ignores, and shuts out most of the stimuli that come from outside.

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