77-12,758

REYNOLDS, Leslie Ann Beard, 1946-
GALVANIC SKIN RESPONSE PATTERNS OF AUTISTIC, SCHIZOPHRENIC, AND CONTROL GROUP CHILDREN TO SENSORY STIMULATION.

The University of Oklahoma, Ph.D., 1976
Education, psychology

THE UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

GALVANIC SKIN RESPONSE PATTERNS OF AUTISTIC, SCHIZOPHRENIC, AND CONTROL GROUP CHILDREN TO SENSORY STIMULATION

A DISSERTATION

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

DOCTOR OF PHILOSOPHY

BY

LESLIE ANN BEARD REYNOLDS

Norman, Oklahoma

1976

GALVANIC SKIN RESPONSE PATTERNS OF AUTISTIC,
SCHIZOPHRENIC, AND CONTROL GROUP CHILDREN
TO SENSORY STIMULATION

APPROVED BY

O. J. Rupiper

Robert L. Curry

Charlyce King

Lloyd V. Williams

DISSERTATION COMMITTEE

ACKNOWLEDGMENTS

This dissertation has not been a solitary effort, but a melding of various sources. The author is grateful to Dr. Omer J. Rupiper, major professor and committee chairman, for his continual guidance and enthusiasm throughout the period of the doctoral program. I also wish to thank Dr. Robert L. Curry, Dr. Charlyce King, and Dr. Lloyd P. Williams for their aid and encouragement during the doctoral program.

Appreciation is extended to Dr. Kathleen Huff, Dr. Donald Marburg, Ms. Maureen Ivey, Ms. Judy Bell, and Dr. Beverly Sutton, staff at the Children's Psychiatric Unit, who helped formulate ideas and ably assisted with the completion of this study. Mrs. Mary Savage is recognized for her aid in the preparation of this manuscript. Additionally, Mr. Dee E. Wheeler is gratefully acknowledged for his necessary and helpful technical and instrumentation assistance.

Finally, I wish to thank my family for their warm support and encouragement during my tenure as a graduate student. A special debt of gratitude goes to Patterson Bond for his gentle patience and continued faith in me.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

GALVANIC SKIN RESPONSE PATTERNS OF AUTISTIC, SCHIZOPHRENIC, AND CONTROL GROUP CHILDREN TO SENSORY STIMULATION

CHAPTER I

INTRODUCTION

Kanner, in 1943, utilized the term "Early Infantile Autism" to delimit a specific cluster of systematically observable behaviors which he noted in eleven children. He saw these behaviors as unique to a syndrome which had previously been unrecognized and therefore considered a variant of the diagnostic classification, "Childhood Schizophrenia" (DSM II, 1968). Since that time, many authors have attempted to determine both the causative and nosological considerations of the behaviors Kanner coined as "autistic disturbances of affective contact" (Kanner, 1943).

The description Kanner used to identify these children emphasized the following behavioral characteristics: inability to develop relationships with people, repetitive behaviors with a desire to maintain sameness, a delay in the acquisition of speech with non-communicative language skills, a good rote memory, and a normal physical appearance. Others (Despert,

1951; Bakwin, 1954) replicated Kanner's observations and concluded that such a disorder did exist; however, considerable controversy over causation, treatment, and diagnostic categorization had resulted.

In a comparative study, Rutter and Lockyer (1967) identified only three symptoms which were universal and specific to "Childhood Autism", as compared to the control group comprising other psychiatric disorders. These three symptoms were a profound and general failure to develop social relationships, language retardation with impaired comprehension, echolalia, pronominal reversal, and ritualistic or compulsive phenomena. The Clancy, Dugdale and Rendle-Short (1968) Checklist had become widely used and was therefore expanded into a concise format for use by the allied medical fields (1969). Most recently, Ornitz and Ritvo (1976) succinctly separated the behavioral symptoms of autism into five subclusters, including disturbances of perception, developmental rate, relating, speech and language, and motility.

Historically, autism was dealt with in reference to psychoanalytic interpretation, thereby inferring environmental factors as causative. This was accepted without question because there seemed to be no objective way to investigate the bizarre behavior of these children with the usual techniques. In the early 1960's, a number of research studies were initiated using physiological and psychological techniques to investigate childhood autism and/or schizophrenia

(Goldfarb, 1961; O'Connor & Hermelin, 1964, 1965). During the past decade, a dearth of clinical studies were conducted which suggested a cognitive defect of yet undetermined organic rather than environmental origin.

Although much of the present clinical research into autism was directed toward further understanding of the more or less accepted research findings that autism operated as the result of organic etiology, there were still those authors whose emphasis remained analytic and environmentally oriented (Franknoi & Ruttenburg, 1971; Bettleheim, 1974) and/or who viewed autistic disturbance as a behavioral variant of childhood schizophrenia (Bender, 1969). Recently, other authors had emphasized behavioral change through remedial or corrective education (Ferster, 1966; Lovaas, Koegel, Simmons, & Long, 1973). Even Kanner (1971a) had modified his original view of autism to include environmental manipulation of a remedial nature rather than placing emphasis on a totally inborn phenomenon aggravated by faulty parenting.

The consistent finding that children variously labeled autistic, schizophrenic, or psychotic exhibited a marked disinterest in their environment, despite differences in historical and symptomatic patterns, had been noted. Varying degrees of unresponsiveness to stimulation in the absence of known sensory impairment had generated experimental reports focusing on responsivity to sensory stimulation emphasizing behavioral (Goldfarb, 1956; Rutter, 1968, 1971, 1974) as well as physiological unresponsiveness (Grey-Walter, 1964). In addition,

differential responsiveness to various sensory modalities had been reported by Goldfarb (1956), Hermelin and O'Connor (1964a, 1964b) and Hutt and Hutt (1968). The verbal and motor reports of this clinical group yielded little consistency, consequently there had been a recent trend toward the use of electrophysical recordings to gain information regarding physiological responsiveness.

The galvanic skin response (GSR) had been employed as an objective and reliable physiological measure of attentional and orienting behaviors. Although there were many and varied theories concerning the GSR, perspiration was apparently the main factor. In the presence of a stimulus a person perspired, thus increasing the amount of salt and other electrolytes which were brought onto the skin. The greater the amount of perspiration on the skin, the lesser the resistance to the flow of electricity between the two electrodes. Conversely as a stimulus dissipated, the perspiration decreased (Venables & Christie, 1973). Research with both autistic and schizophrenic children had been prohibited because of the severe communication handicap of this clinical group. Therefore, a more objective measure of response to stimulation was needed. Through the use of the GSR, the degree to which the central nervous system registered the stimulus and produced an autonomic response could be directly measured. The application of such a measure to this clinical group could, therefore, yield highly useful information.

In summary, this research study was an attempt to investigate to which sensory stimuli autistic and schizophrenic children responded, as measured by the GSR.

Statement of the Problem

The psychoanalytic interpretation of the etiology of Early Infantile Autism, based on clinical observation alone, remained important only as the historical antecedent to the recent application of research principles. Findings which supported an organic basis for many of the deficits associated with this disorder led researchers to explore possible areas of central nervous system (CNS) disruption, as well as which perceptual channels would more effectively allow for the acquisition of learning for this clinical group. It was not known to what stimuli these children specifically responded or which stimuli were capable of promoting learning and overt behavioral change. Therefore, the problem of this study was an attempt to explore to what sensory stimuli autistic children primarily attended. The secondary problem of this study was an attempt to determine whether the GSR, as an index of behavioral activation, was utilizable to determine to what sensory modalities each child appeared to respond consistently, thus making the specific limitations for the acquisition of learning more objectively measurable. In this way, perhaps each child diagnosed autistic or schizophrenic could achieve his highest level of performance in functioning in the home, the school, or the therapeutic

milieu. At the very least, by determining individual preference or sensory reception levels, prosthetic environments could be designed for the individual child, according to his maximum abilities.

Statement of the Purposes

The purpose of this study was to investigate differences between autistic, schizophrenic, and control group children in reactivity to repeated sensory stimuli, using electrodermal changes (GSR) as the dependent variable. Further, this study attempted to determine whether the GSR could be utilized to more objectively differentiate children diagnosed "Childhood Schizophrenic" from those diagnosed "Early Infantile Autistic."

Relevant Literature on the Application of the Galvanic Skin Response to Autistic and Schizophrenic Children

In one of the earliest published works dealing with the application of the galvanic skin response (GSR) Darrow (1929) found that the immediate reflex effect of sensory stimulation could be measured by the use of electrodermal reactivity. He further noted that the galvanic reflex followed excitation immediately without requiring that the stimuli have acquired meaning to the subject, as the GSR was more responsive to sensory rather than ideational stimuli. More recently, physiological and biofeedback laboratory research had consistently shown the GSR to be a highly responsive

indicator of emotional stress (Barland & Raskin, 1973).

The GSR had been utilized by researchers to measure such physiological parameters as anxiety levels in adults (Mandler, Mandler & Uviller, 1958; Lader & Wing, 1964); the effect of auditory and visual stimulation in schizophrenic and normal adults (Venables, 1960); and response patterns of normal versus psychotic adults (Paintal, 1951; Brown, 1974). In children, the GSR had been used as a measure of hyperactivity (Satterfield and Dawson, 1971), but there had been few studies employing this parameter with autistic and schizophrenic children. The most notable of these was Bernal and Miller (1971) in which it was noted that schizophrenic children of the autistic type produced a consistently lower magnitude GSR both to initial sensory stimulus and to the highest intensity stimulus as compared to normals. Thus, the correlation between theories of "over-arousal" and self-stimulation were not supported by these findings, nor was there evidence that the schizophrenic children were more aroused than the normals in electrodermal function. It was theorized that response magnitude, rather than response pattern, was a characteristic difference in schizophrenic versus normal children's GSR. This study utilized only visual and auditory stimulus presentation; since both of the stimuli represented distal receptors, a comparison of receptor preference for either near or far receptor use by autistic-type schizophrenic children was not possible.

Related research utilizing electroneurophysiologic parameters to measure sensory stimulation and response patterns of autistic and autistic-like children had been conducted by Grey-Walter, Aldridge, Cooper, O'Gorman, McCallum, & Winter (1971) and Small (1971). Grey-Walter, et al. sought to discover how patterns of interaction in polygraphic records of response to visual, auditory, and tactile stimuli presented alone and in combination, interacted and varied with the following in normal and disturbed children (thirty disturbed subjects, thirteen of whom were diagnosed as autistic): age, attitude, mental development and social maturity. The general hypothesis underlying this series of experiments was that in conditional adaptation the brain mechanisms acted as computers of contingent significance and that this action was reflected in the interaction patterns of brain responses evoked by associated stimuli. Results yielded the following: 1) although autonomic responses to various stimuli were recorded, disturbed children's nonspecific responses to visual stimuli were absent in one-third of the subjects and also absent to auditory stimuli in three-fourths of the subjects. These same subjects also showed pronounced autonomic excitement and anxious behavior. 2) Disturbances of sensorimotor experience in younger children appeared to set up a state of exaggerated autonomic excitement, associated with anxiety, which could interfere continuously with the establishment of stable interactive adaptations. 3) Of the disturbed

children, many were chronically distracted by internal excitement while others were incapable of preliminary emotional engagement. Because of this finding, the consideration of some simple fissure between the cortex and hypothalamus was considered. This perpetual state of autonomic dissociation and disintegration could be transiently induced in normal people by fright or embarrassment, but appeared to be the pattern for the disturbed subjects.

Small (1971) investigated average sensory responses in neurophysiological functioning and slow potential shift as measured by the EEG in five matched autistic-normal pairs of children. In data obtained from a series of over seventy-eight recording sessions, the preliminary observations suggested that there were identifiable differences between the cerebral evoked responses of autistic and normal children, even though the experimental subjects were considered neurologically intact at the time of the experiment. Visual evoked responses of the experimental subjects were of lower amplitude, while the auditory evoked responses of these subjects appeared less complex. That is, fewer negative and positive peaks were found in the records of the experimental subjects as compared with the controls. Background EEG frequencies also appeared to be faster in the experimental subjects. When stimuli of two different modalities were presented, a "scrambling" or marked variability of the visual evoked response occurred in the autistic children.

In spite of the fact that there was a good deal of research into various uses for the GSR, only a very small portion of it related to psychotic populations and virtually no studies, with the exception of Bernal & Miller, dealt with receptivity of this clinical group to sensory stimulation as measured by GSR. The reported studies which investigated the neurophysiological deviations of childhood autism in the presence of sensory stimulation were pilot projects and were subject to further empirical study. Consequently, the above mentioned literature reflected the relevant studies which dealt directly with these aspects of the research.

Definition of Terms

Galvanic Skin Response (GSR). An instrument used for the recording of the autonomic parameter of electrodermal resistance or conductance on the surface of the skin. The GSR is an electrophysiological recording which measures the degree to which the nervous system has registered a stimulus and an effector reaction has occurred.

Sensory Receptors.

A) Contact (near) receptors. Those receptors of sensory stimuli associated with touch, taste, and/or smell.

B) Distal (far) receptors. Those receptors of sensory stimuli associated with vision and hearing.

Subject Diagnosis.

A) Control Group. Hospitalized children whose diagnosis was non-psychotic, non-organic, and whose intellectual functioning fell within the normal range. DSM II (1968)

psychiatric diagnosis including Behavior and Personality Disordered classifications.

B) Schizophrenia, childhood type. Cases in which schizophrenic symptoms appear before puberty. The condition may be manifested by autistic, atypical, and withdrawn behavior not classifiable under other types of schizophrenia (DSM II, 1968).

C) Early Infantile Autism. Inability to develop relationships with people; repetitive behaviors with a desire to maintain sameness; a delay in the acquisition of speech with non-communicative language skills; a good rote memory; a normal physical appearance (Kanner, 1943).

The diagnosis of infantile autism was applied when at least seven of the following criteria were present and the symptoms began within the first three years of life:

1) Great difficulty in mixing and playing with other children.
2) Acts as if deaf--does not react to speech or noise.
3) Strong resistance to any learning--either new behavior or new skills.
4) Lack of fear about realistic dangers, e.g., may play with fire.
5) Resist change in routine--the smallest change may produce disproportionate anxiety.
6) Prefers to indicate needs by gestures, speech may or may not be present.

7) Laughs and giggles for no apparent reason.

8) Not cuddly as a baby.

9) Marked physical overactivity.

10) No eye contact, persistently looks past or turns away from persons, especially when spoken to.

11) Unusual attachment to a particular object or objects.

12) Spins objects, especially round ones.

13) Repetitive and sustained odd play, e.g., rattling stones in a can.

14) Standoffish manner, treats persons as objects rather than as persons.

(Clancy, Dugdale, & Rendle-Short, 1968).

Null Hypotheses

H_{01} - There is no significant difference in the amplitude of GSR responses of hospitalized children diagnosed autistic, schizophrenic, or control group to various sensory stimuli.

H_{02} - There is no significant difference in sensory receptor preference of hospitalized autistic, schizophrenic, or control group children as measured by the GSR.

CHAPTER II

DESIGN OF STUDY

Selection of the Subjects

The sample was comprised of five hospitalized children diagnosed Childhood Schizophrenic (DSM II, 1968), five hospitalized children diagnosed Early Infantile Autism (Clancy, Dugdale, & Rendle-Short, 1968) (Appendix B), and five hospitalized non-psychotic control group children. All experimental subjects were matched by sex and age with range in age from 7 years 7 months to 16 years and a mean chronological age of 10 years 9 months. The ratio of three boys to two girls in the autistic and schizophrenic groups coincided with research findings delineating these clinical groups (Hingtgen & Bryson, 1971; Rimland, 1964).

The hospitalized control group children were matched by sex and (as far as possible) by age to the experimental groups. In addition, the control group children were non-organic, non-psychotic, and fell within the normal range of intelligence as measured by the WISC-R. Mean chronological age for control group children was 11 years 6 months. All schizophrenic subjects fell within the mild to moderate mental

retardation range as measured by the WISC-R. All autistic subjects were untestable with the WISC-R, but on the Vineland Scale of Social Maturity they scored in the moderate to severe range of social functioning.

Description of the Instruments

A Stoelting Polyscribe portable electronic Galvanic Skin Response instrument, model #22770, with a self-contained brush recording unit was used. Finger electrodes were placed on the left ring and index finger of each subject after the application of EKG Sol (Burton, Parsons, & Co.) to enhance skin conductance potential.

The sound attenuated experimental room measured 6 x 7 feet and contained a 2 x 4 foot experimental booth. A 28 x 12 inch desk area on each of the booths two sides was separated by a partial screen. This screen served as both a shield to prevent stimulus contamination and contained an 8 x 8 inch opaque screen with a 2 x 8 inch space at its base. Finger electrodes were made stationary at the left side base of this screen. The booth housed a storage area on the side accessible to the examiner. A white noise generator (Grason Stadler #455B) placed in this space remained in operation at 70 decibels during acclimation and experimental phases to avoid contamination of the subjects response by extraneous noise. A 3 x 3 foot table placed behind the booth held the GSR instrument, which was blocked from the subjects' view by a 3 x 2 foot screen.

Additional visible equipment in the room included two identical chairs and a floor to ceiling curtain placed at the end of the booth. The curtain was used to deter subjects from examining experimental objects, and from visual distraction by the entrance and exit door. All walls were blank and at all times the room furnishings remained unchanged. Temperature in the room was controlled and varied no more than from 74 to 76 degrees. Light intensity was held constant at 60 watts diffused light.

Procedure for Collecting Data

All experimental subjects were first familiarized with the experimental procedures in a standardized manner. Primary reinforcement with M&Ms as a reward followed the subjects' demonstrated ability to 1) come into the experimental room, 2) sit in the designated chair, 3) examine the mock electrodes placed on the desk, 4) sit relatively still, 5) hold their left hand in place on an outline of a hand, and 6) remain seated quietly with the mock electrodes attached to their fingers as their left hand remained stationary on a hand outline. Control subjects were first familiarized with the experimental room; at the time of data collection these subjects were instructed to keep their left hand as still as possible with the electrodes attached and direct their attention to the various presentations the examiner would make. Primary reinforcement followed each session for all subjects. For experimental subjects reinforcement often followed the presentation of each stimulus complex as well.

Each testing session constituted the randomized presentation of stimulus in the five primary sensory modalities with five consecutive presentations in each condition, interspersed with no stimulus intervals. All stimulus conditions were presented for three seconds, followed by a ten second no stimulus interval, while GSR was being continuously recorded. Three testing sessions per subject were presented during an eight day period. In addition it was at times necessary to present experimental subjects more than five presentations in some modalities due to movement artifacts; presentations were therefore made until five scorable GSR responses per modality were obtained. At times when extraneous elements were responsible for large resistance drops in GSR, testing was momentarily discontinued until GSR recording had returned to baseline.

During acclimation and actual data collection sessions, the subject and examiner were seated in stationary chairs in the booth facing each other. An observer marked off stimulus presentation intervals on all GSR print-outs. Gross subject movement, talking, and self-stimulation were also noted on the print-out to delineate artifact responses from stimulus responses. Another observer stood beside experimental subjects to observe for movement and insure that subjects remained in their seat. This observer was quite familiar with experimental subjects, as recent findings consistently suggested that biofeedback responses by autistic and schizophrenic, as well as normal subjects were favorably influenced to a significant

degree by examiner familiarity (Small, 1971; Brown, 1974). Posture of all subjects was standardized, as pilot research indicated that GSR responsivity was significantly influenced by body position (Wheeler, 1976).

The GSR instrument was set on automatic recording mode for all presentations so that all GSR reactions of each subject took place from an arbitrarily and consistently assigned baseline. In this way the strength of every reaction to experimental stimulation (and otherwise) was directly comparable to the strength of the other GSR reactions on each protocal for each subject. Since medication on experimental subjects was not discontinued, no readable GSR tracing could be obtained except under the self-centering mode. A standardized level of sensitivity for adjusting the gain (amplitude) of the pen was set at 3500 ohms for all subjects in all sessions. At the beginning of each day's data collection, the GSR was calibrated to insure standardized instrument responsivity; at the beginning and end of each session a 1-K response for each subject was also obtained. This served to further standardize instrument responsivity by measuring the relative amplitude of each subject's response to a 1000 ohm change in resistance.

Following are the sensory stimuli utilized and their mode of presentation:

Visual--A 75 watt light bulb encased in a shield was placed behind the opaque screen. The light was flashed on

for three seconds, off for ten seconds for a total of five times each session.

Auditory--As white noise (static) was emitted continuously, a pure tone at the intensity of 80 decibels, 1000hz (Breur & Keur sound level meter) was presented for three seconds, followed by a ten second no stimulus presentation. Five such presentations were made for each of the three sessions.

Olfactory--Identical Culturette (Scientific Products) capped swab sticks, one saturated with musk (Gildard, 1972), the other with water, were presented manually to each subject to smell for alternating periods of three seconds with ten seconds between stimulus intervals. Additionally, the culturette tubes were airtight so that contamination of odor/no odor intervals did not occur.

Gustatory--A single drop of a 2% saline solution (Gildard, 1972) was presented on each subject's tongue by a 1.0 to 5.0 mg oral medication dropper, followed by a ten second no stimulus presentation. This sequence was presented five times with alternating presentations of a single drop of distilled water by an identical dropper and drawn from an identical bottle.

Touch--Each subject was instructed to place his right hand, palm up, through the opening in the base of the partial/opaque screen. The subject's index finger was stroked lightly in one continuous motion with a #120 soft natural fiber brush (1/2" Artista, Binny & Smith Co.) for a duration of three

seconds. This was repeated five times, interspersed with ten second no stimulus intervals while the subject's hand remained in position under the screen.

Treatment of the Data

GSR print-outs were analyzed according to amplitude of GSR response during stimulus presentations. A scorable GSR was defined as a skin resistance drop of 375 ohms or greater occurring within one to ten seconds after stimulus onset.[1] A scoring template was used to mark off each ten-second response interval and assign a numerical value to the scorable response within that interval. This number value was derived from assigning each resistance drop of 375 ohms the numerical value of one (Appendix C).

Additional scoring criterion applied to the polygraphic records included the following treatment. When the maximum resistance drop was reached in response to the stimulus and the pen arm began its descent toward baseline, an additional resistance drop occurring in the ten second interval was not scored if the pen had returned half the distance to baseline as compared to the initial resistance drop in that interval. Conversely, when the initial resistance drop did not return by half the distance to its arbitrary baseline, the amplitude of the second resistance peak was scored. In other words, the first or second response

---

[1]This unit of electrical resistance per line is standardized by the GSR instrument used (Stoelting Co.)

in each ten second interval that met this criterion was considered a valid response and all other responses within that interval were not scored. The additional "responses" that occurred were assumed to be the result of intrinsic, spontaneous autonomic activity within the subject and therefore not a response to experimental manipulation (Appendix C).

CHAPTER III

RESULTS

For each subject five GSR responses per modality were averaged from each of the three recording sessions, thus obtaining a mean response level for each subject in each sensory modality. A "subject mean" was then derived for each subject in each modality by averaging the means from the three sessions (Table 1). Each "subject mean" was grouped by diagnosis of Autistic, Schizophrenic, or Control, and by stimulus modality, then rank-ordered by modality (Table 1). Sums of ranks of amplitude scores were analyzed by the Kruskal-Wallis non-parametric analysis of variance (Spence, Underwood, Duncan, & Cotton, 1968) across three groups in five modalities (Table 2). Results yielded no significant differences among groups in response to any of the sensory stimuli. Survey of these results suggested that visual stimulation produced the greatest variation in response across the three groups ($p = .10$). The patterns of response across groups to the auditory, olfactory, gustatory, and touch presentations suggested that all three groups responded similarly to stimulation in these modalities as

Table 1

Mean GSR Amplitude of the Response to Each Sensory Modality by Subject and the Accompanying Rank

Subjects	Sensory Modality									
	Vision		Audition		Olfaction		Gustation		Touch	
	$\bar{X}$	R	$\bar{X}$	R	$\bar{X}$	R	$\bar{X}$	R	$\bar{X}$	R
Autistic	14.00	8	19.95	15	13.60	9	21.47	15	18.60	12
	15.47	11	19.67	14	14.69	10	15.73	13	12.40	7
	14.80	10	15.40	9	13.40	7	9.00	7	12.47	8
	8.07	4	9.33	3	13.47	8	4.60	3	11.00	6
	5.80	2	8.33	2	4.00	2	11.00	10	6.40	3
Schizophrenic	19.47	14	17.00	12	20.20	14	5.93	4	19.80	14
	11.60	6	9.60	4	16.00	12	6.47	5	8.53	4
	14.47	9	15.13	8	6.93	3	12.00	11	10.00	5
	18.40	13	10.07	5	10.13	5	7.60	6	13.73	9
	20.13	15	18.87	13	14.80	11	17.60	14	17.33	11
Control	13.13	7	16.56	11	18.87	13	9.87	9	15.13	10
	10.47	5	16.47	10	12.07	6	3.33	1	5.40	1
	16.47	12	14.33	7	20.80	15	9.07	8	20.20	15
	7.47	3	11.47	6	9.60	4	13.20	12	19.47	13
	4.73	1	4.67	1	3.60	1	3.47	2	5.87	2

measured by the GSR (p = .53 to p = .88). Therefore, the null hypothesis of no significant difference in GSR amplitudes of hospitalized children diagnosed autistic, schizophrenic, or control group to various sensory stimuli was not rejected. Additionally, the null hypothesis of no significant difference in sensory receptor preference of the three groups was not rejected.

A summary of the group mean amplitude scores from the GSR to the sensory modalities for all three groups is presented in Figure 1. The most striking feature of the electrodermal data was that the schizophrenic and control groups response to gustatory stimuli was of a lower amplitude than to all other sensory stimuli, while the autistic group responded the least to visual and olfactory stimuli. Schizophrenic subjects as a group scored the highest mean amplitude score across all groups to visual stimuli (Figure 1). Response to touch stimuli yielded the most consistent GSR amplitudes by all three groups (p = .88) while response to visual stimuli yielded the greatest difference in response by group (p = .10).

Figures 2, 3, and 4 depict response means of the autistic, schizophrenic, and control subjects to sensory stimulation in five modalities. While schizophrenic and control subjects showed less preference for gustatory stimuli, within the autistic group this preference was determined by individual pattern rather than by a trend within the diagnostic category. Additionally, autistic subjects response means

TABLE 2

Kruskal-Wallis One-Way ANOVA by Ranks of GSR Amplitude Scores Across Three Groups in Five Modalities

Modality	N	df	H	P
Vision	15	2	4.58	.10
Audition	15	2	.38	.83
Olfaction	15	2	.42	.81
Gustation	15	2	1.28	.53
Touch	15	2	.26	.88

to gustatory stimuli and schizophrenic and control subjects response means to olfactory stimuli yielded the largest range of responses of all groups across the entire stimulus complex.

The loss of evoked GSR's to sensory stimulation due to movement occurred most frequently in the autistic group. Infrequent movement responses were recorded for the schizophrenic group and only rarely for the controls. The potential for biasing the data due to the heavy preponderance of movement in the autistic group was controlled for by the additional presentation of sensory stimuli when movement artifacts were present. Under this circumstance only, additional stimuli were presented until five scorable responses per modality were obtained.

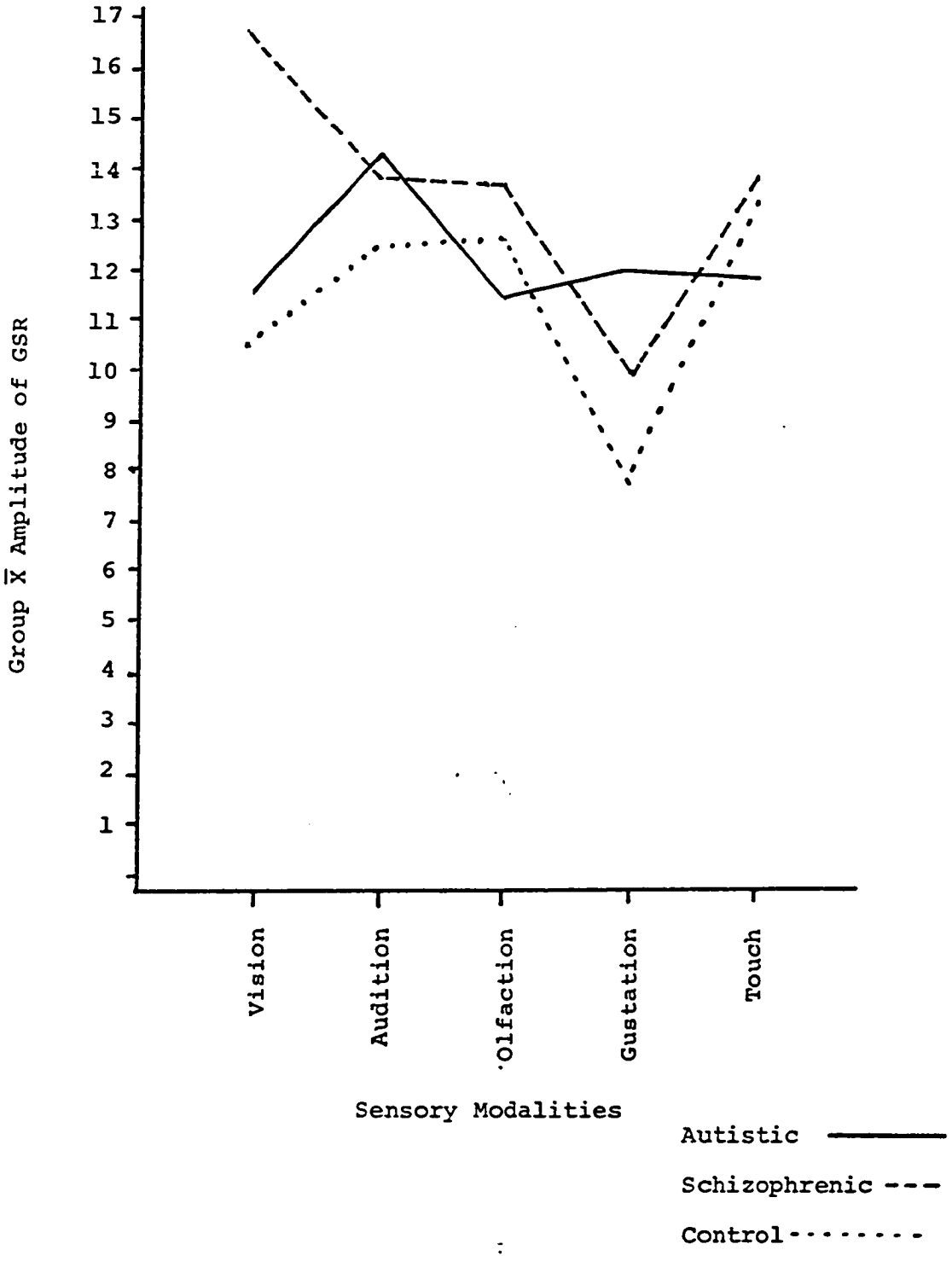


Figure 1. Group mean amplitudes to GSR by sensory modalities.

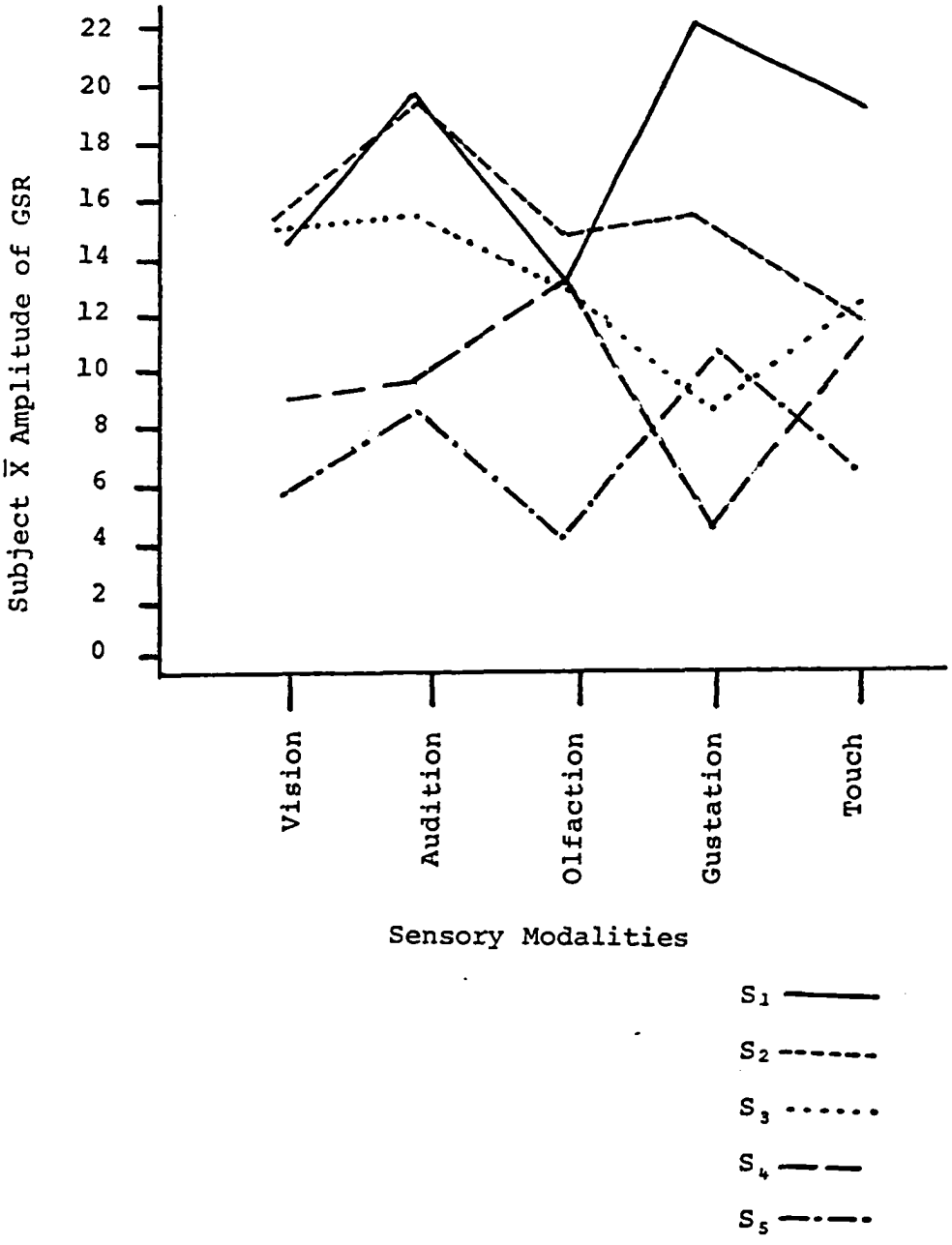


Figure 2. Response means of autistic subjects to sensory stimuli.

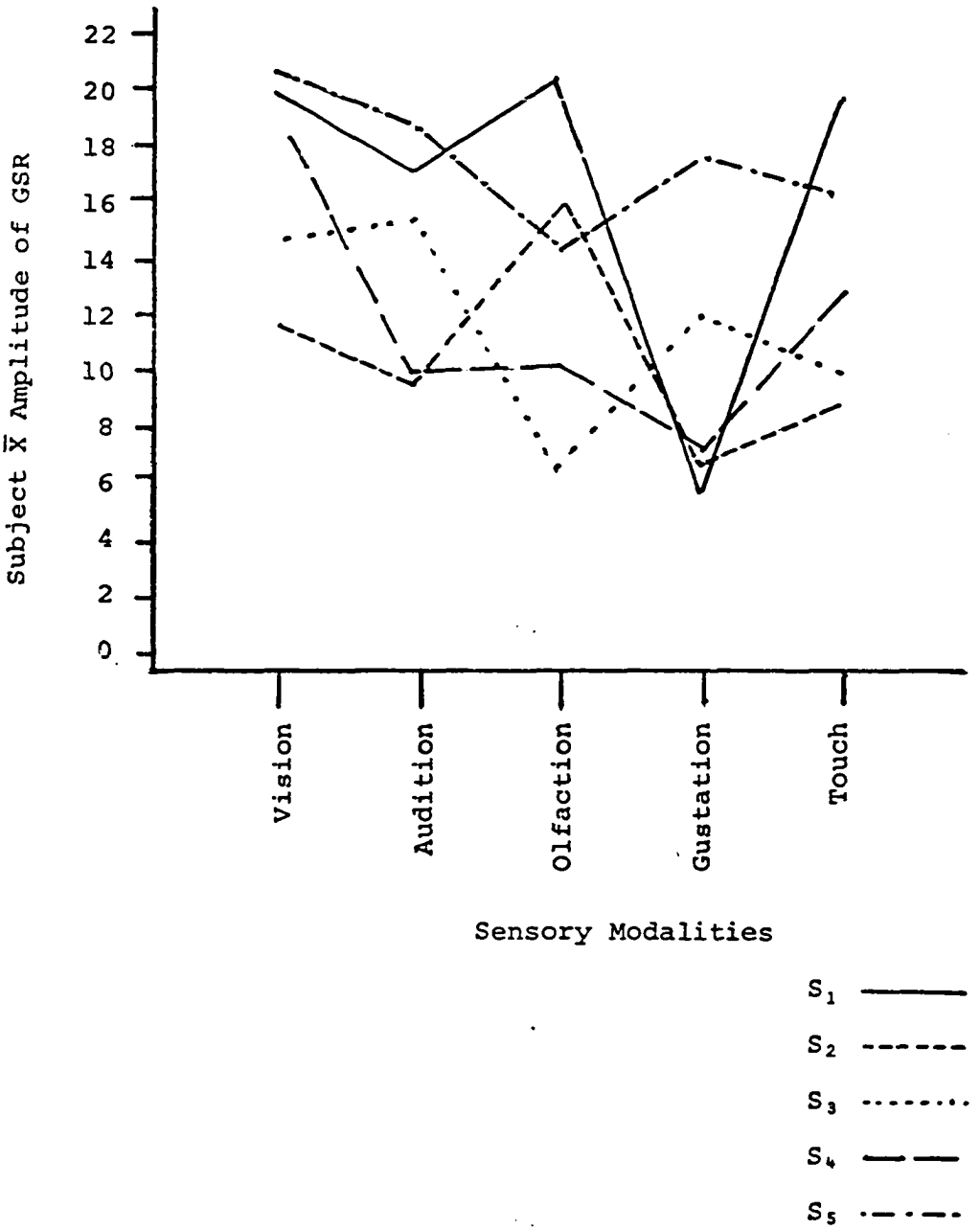


Figure 3. Response means of schizophrenic subjects to sensory stimuli.

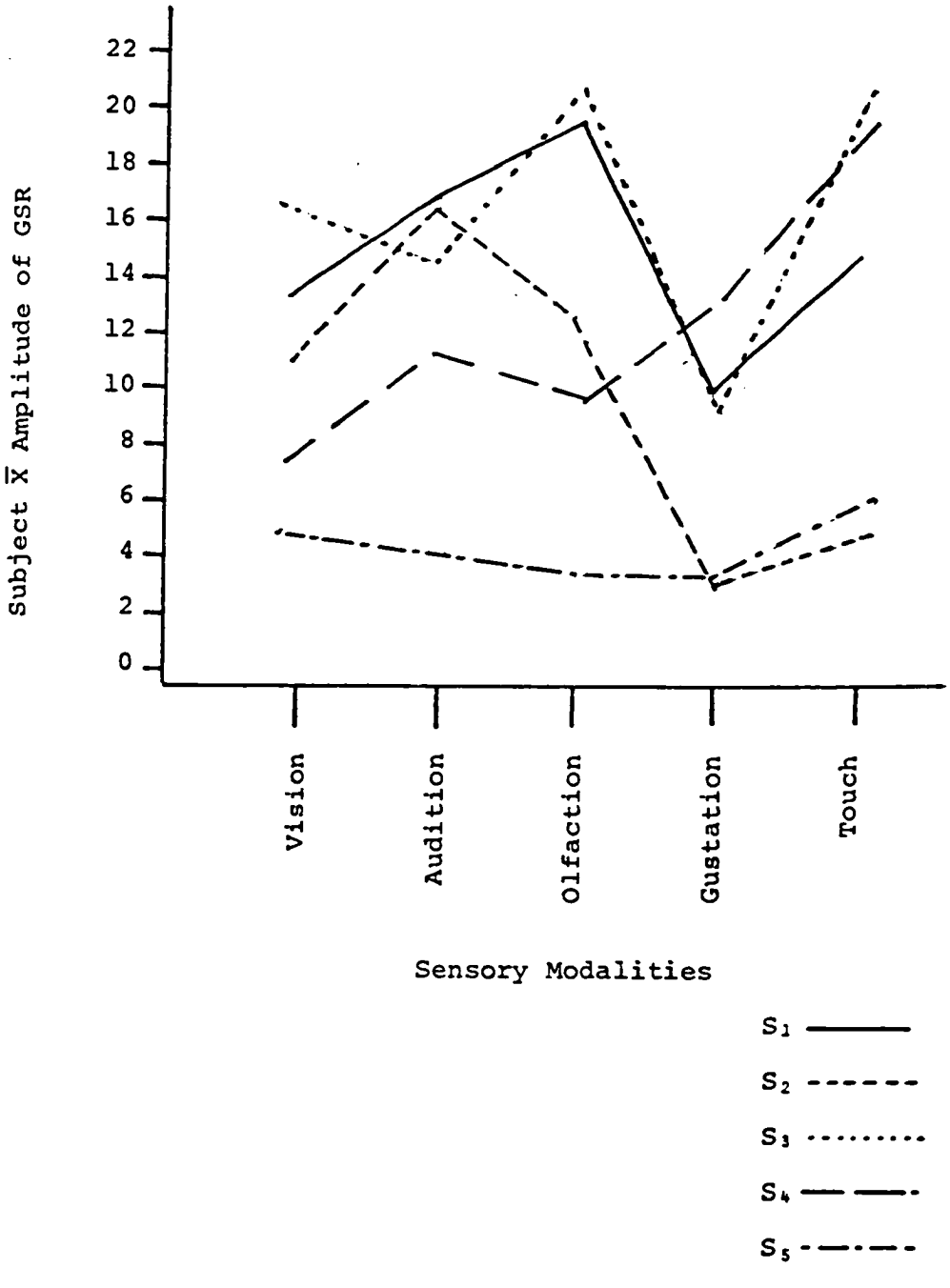


Figure 4. Response means of control subjects to sensory stimuli.

Table 3 illustrated the measured level of intellectual functioning of the autistic group, their mean amplitude responses and rank-order by each sensory modality. In the autistic group the highest functioning subjects scored the lowest average rank in response across the stimuli; the lowest functioning autistic subjects scored the next lowest rank average across the stimuli. Within the schizophrenic and control groups there was no discernable pattern in average rank of scores across modalities as a result of the level of individual intellectual functioning (or potential).

In summary, it would appear that autistic, schizophrenic and control group children matched for age and sex responded to sensory stimulation in the five primary modalities in a statistically similar manner when electrodermal change (GSR) was utilized as the dependent variable.

Table 3

Vineland Scale Social Quotient, Mean Amplitude Response, and Rank-Order of Autistic Subjects by Sensory Modality

Vineland Social Quotient	Vision		Audition		Olfaction		Gustation		Touch	
	$\bar{X}$	R	$\bar{X}$	R	$\bar{X}$	R	$\bar{X}$	R	$\bar{X}$	R
20	8.07	4	9.33	3	13,47	8	4.60	3	11.00	6
26	15.47	11	19.67	14	14.69	10	15.73	13	12.40	7
28	14.00	8	19.95	15	13.60	9	9.00	7	18.60	12
35	14.80	10	15.40	9	13.40	7	4.60	3	12.47	8
55	5.80	2	8.33	2	4.00	2	11.00	10	6.40	3

CHAPTER IV

DISCUSSION

Although statistical analysis of the data failed to show any significant differences in response patterns across groups, qualitative appraisal of the GSR print-outs for each diagnostic classification yielded highly individual patterns of response between each group of diagnosed autistic, schizophrenic, and control children. The controls began to habituate to the sensory stimuli rapidly, which yielded progressively lower amplitude responses and eventually zero responses in most modalities. However, neither the schizophrenic nor autistic subjects showed this ability to habituate to the stimuli. Protocols of control subjects appeared rather uniform; response latency was rapid; there were few spontaneous recordings in the between stimulus intervals; and the resistance peaks rose rapidly and smoothly, then returned to the arbitrary baseline.

Schizophrenic subject protocols contained many more spontaneous GSR peaks which were jagged and erratic. Between stimulus recordings were often of a higher amplitude than recordings based on sensory stimulation. There were copious

double-response artifacts on the schizophrenic protocols. It appeared, from inspection, that when the schizophrenic subjects were responding to sensory stimulation and were "tuned in" to the experimental procedure, their response peaks were of a lower, less erratic amplitude than other peaks. The duration of response peaks was extended; also, the smooth peak noted in the control subjects polygraphic records was angled off for the schizophrenic subjects. Pen movements also yielded motions which swept toward the left of center. In short, the schizophrenic protocols as a whole were characterized by much vaso-motor instability with higher resistance drops to spontaneous autonomic activity than to experimental presentations. Double response patterns indicated that the secondary response to stimulus was of a consistently higher magnitude than to evoked GSR. This pattern decreased somewhat as the subjects became more comfortable with the testing procedure. Perhaps the double response pattern and the between stimulus GSR activity was a result of both organic involvement and a function of a higher level of arousal due to anxiety. With extended recording sessions and a larger number of subjects this spurious observation could be more readily documented.

GSR records of the autistic subjects also contained double response artifacts; however, the ratio of these secondary responses was considerably higher within the schizophrenic group. When double responses did occur, they were of higher magnitude than the response measured to the sensory

stimulation. As a group the autistic subjects displayed continuous spontaneous GSR activity that was of such high intensity that many of the entire protocols resembled movement artifact responses, when there had been no physical movement at all. The autistic polygraphic records were characterized by massive vaso-motor instability. There were also many and jagged peaks, longer durations of response to the stimulus than in the schizophrenic group, and with few exceptions, the responses to experimental stimulus were of a lower magnitude than any of the spontaneous recording.

It would appear that in the experimental group, the subjects were able to respond to the sensory stimulation in statistically similar ways to the control group. However, in this study the between stimulus interval recordings yielded vast differences among the three groups in resting phase GSR. Conversely, Bernal and Miller (1971) found that autistic-type schizophrenic subjects produced a consistently lower magnitude GSR to varying intensities of visual and auditory stimulation when compared to matched normal subjects. They stated as a result that the schizophrenic subjects were not "over-aroused" and that it was response magnitude rather than response pattern that was the characteristic difference in schizophrenic versus normal children's GSR patterns.

The present investigation would support theories which posed innate defects in the physiological arousal mechanisms of both autistic and schizophrenic children. This CNS interruption purportedly interfered with the ability to process

incoming stimuli in meaningful ways. Both the autistic and schizophrenic groups appeared to lack ability to maintain the perceptual constancy that was observed in the control subjects. Experimental subjects appeared to present random underloading and overloading of CNS functions to the extent that no habituation to the stimuli could be maintained. This suggested that these subjects could have viewed the same sensory perception differently each time it was presented. Ornitz and Ritvo (1968) and Ornitz (1969) suggested that this type of random perceptual confusion was compatable with both autism and childhood schizophrenia. These authors suggested the presence of an abnormal physiological state involving degrees of excitation, facilitation, and inhibition of information processing. The present investigation supported this finding, particularly in view of the observed disparity of GSR in between-and within-stimulus recordings.

Ornitz, Ritvo and Brown (1969) postulated a malfunction of the homeostatic regulation of sensory input in both clinical groups. Present investigation also supported this theory; although random perceptual confusion was noted in both groups, the autistic protocols by visual inspection alone could be separated out from the schizophrenic protocols due to the increased vaso-motor instability seen on all autistic records. Additionally, autistic subjects appeared to selectively respond to sensory stimuli to the extent that stimuli in one modality were alternately not registered at the autonomic level (or "tuned out") and then registered at

a high amplitude on subsequent response. For the recording of five presentations in one sensory modality, several of the autistic subjects alternately over-responded as shown by a large resistance drop in GSR, or failed to respond at all, yielding a flat pen recording for that particular presentation.

Hutt and Hutt (1965, 1968) noted an abnormally high level of arousal in schizophrenics. The stereotyped behaviors exhibited by this clinical group were said to have an arousal reducing function of blocking sensory input. In the current study both autistic and schizophrenic subjects were characterized by an abnormal level of arousal, but it was the autistic subjects, not the schizophrenics, who showed the ability to block sensory input. Observation of the behavior of both experimental groups did suggest that the level of anxiety in both groups was initially high but with increased familiarity with the experimental procedure, the sterotypes of the autistics diminished somewhat. The double-response pattern of the schizophrenics also decreased as experimental procedures became more familiar.

The more current theories of autism and childhood schizophrenia which follow Bender's early (1947) statement that organic factors were causative, suggested various CNS disruptions. Goldfarb (1961), Schopler (1966), and Mahler (1965) had noted that schizophrenic children selectively experience and respond to their environment. Individual patterns noted in the autistic group in the current study

supported the observation of selective responding; however, the present study failed to support the theory that autistic and schizophrenic children rely mainly on touch, taste, and smell for perceptual orientation. GSR amplitude scores of the schizophrenic subjects suggested that since gustatory responses yielded the lowest autonomic recording and that for the autistic group olfaction and vision amplitude yielded the lowest response, these clinical groups did not show such a preference for proximal receptors.

Further, all three groups responded the most statistically similar to touch stimuli. The schizophrenic subjects scored the highest amplitude scores across all groups to visual stimuli, while Schopler (1966) had noted that schizophrenics showed significantly less visual preference (over tactile) than the same aged normals. GSR recordings for this group suggested that visual stimuli, at least on an autonomic level, were received with more impact than were any other modalities that were presented. In short, the present study failed to confirm theories of proximal receptor preference for the schizophrenic and autistic groups since these groups responded in statistically similar ways to the control subjects across all modalities as measured by the GSR. Frith (1970) noted that autistic children failed to be sensitive to experimental structures imposing their own sterotyped patterns. Present investigation suggested that these children did respond to experimental structure and with familiarity, decreased stereotypies.

There was the distinct possibility that sensory stimuli were indeed received by autistic and schizophrenic children, but that in the encoding process, sequential sensory inputs were not linked, thus discrimination and generalization learning failed to be incorporated into the cognitive process. Hermelin and O'Connor (1964a, 1964b, 1967) stated that autistic and schizophrenic children were more dependent than normal individuals on feedback from their motor responses to make sense out of their perceptions. In the presentation of gustatory and olfactory stimuli in the present study, there were stimulus intervals where presentations were made which contained an absence of smell or taste. Neither the schizophrenic nor autistic subjects maintained discrimination abilities in these presentations, using GSR amplitude as an indicator of arousal to the stimulus.

Hermelin and O'Connor (1964a) further suggested that only more complex perceptual tasks that depended on efficient information processing might be expected to be impaired. Thus, the more complex task of discriminating smell and taste from the absense of these stimuli would have been too complex a task. Also to be taken into consideration in the present investigation was the fact that these presentations were made manually by the examiner. Other stimulus modalities did not allow for the kind of anticipatory GSR that gustation and olfaction did. However, autistic subjects have been shown to lack emotional response to startle and delayed or absent attention to auditory and visual stimuli, as well as to

persons in the environment (Ornitz & Ritvo, 1976). It would have been inconclusive to cite lack of olfactory and gustatory discrimination and/or the emotional response of the experimental presentations as both necessary and sufficient reasons for the obtained data.

The present investigation demonstrated that in polygraphic recordings of the GSR, autistic, schizophrenic, and control subjects were able to respond to simple sensory stimulation in five modalities in statistically similar ways. The psychophysiological significance of any given GSR yielded in this study depended on the nature, extent, and intensity of the neural functions producing it. Thus, on occasion, the GSR may have been symptomatic of high level association and other cortically involved activities, but the presence of a GSR in response to sensory stimulation did not necessarily implicate higher level functions (Darrow, 1967). Perhaps one of the basic deficiencies underlying the autistic syndrome was not the avoidance (or lack of response) of sensory stimuli per se, but a deficit in the ability to make cross-modal, discrimination, and generalization associations necessary for further cognitive development in the areas of perceptual and language acquisition and in social relatedness and affective contact.

CHAPTER V

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary

The psychoanalytic interpretation of the etiology of Early Infantile Autism, based on clinical observation alone, remained important only as the historical antecedent to the recent application of research principles. Findings which supported an organic basis for many of the deficits associated with this disorder led researchers to explore possible areas of central nervous system (CNS) disruption, as well as which perceptual channels would more effectively allow for the acquisition of learning for this clinical group. It was not known to what stimuli these children specifically responded or which stimuli were capable of promoting learning and overt behavioral change. Therefore, the problem of this study was an attempt to explore to what sensory stimuli autistic children primarily attended. The secondary problem of this study was an attempt to determine whether the GSR, as an index of behavioral activation, was utilizable to determine to what sensory modalities each child appeared to respond to consistently, thus making the specific limitations

for the acquisition of learning more objectively measurable.

The purpose of this study was to investigate differences between autistic, schizophrenic, and control group children in reactivity to repeated sensory stimuli, using electrodermal changes (GSR) as the dependent variable. Further, this study attempted to determine whether the GSR could be utilized to more objectively differentiate children diagnosed "Childhood Schizophrenic" from those diagnosed "Early Infantile Autistic."

The null hypotheses set forth were: There is no significant difference in 1) evoked amplitude of GSR response, or 2) sensory receptor preference of hospitalized autistic, schizophrenic, or control group children as measured by the GSR.

The sample was comprised of five hospitalized children diagnosed Childhood Schizophrenic (DSM II, 1968), five hospitalized children diagnosed Early Infantile Autism (Rendle-Short, 1968), and five hospitalized non-psychotic, non-organic control group children matched by sex and age. Subjects were presented with random sensory stimulation in five modalities (vision, audition, olfaction, gustation, and touch) while GSR was continuously recorded. Five standardized presentations per modality were made; a total of three recording sessions per subject yielded fifteen scorable GSR protocols, three for each sensory modality.

GSR protocols were analyzed by amplitude of evoked autonomic response. The Kruskall-Wallis one-way ANOVA by

ranks was applied. Results yielded no significant differences among groups in response to any of the sensory stimuli. Survey of the results suggested that visual stimulation produced the greatest variation in response amplitude across groups, while touch stimulation produced the most similar response patterns across groups as measured by the GSR. Therefore, the null hypothesis of no significant difference in GSR amplitudes of diagnosed autistic, schizophrenic, or control group to the sensory stimulation was not rejected. Additionally, the null hypothesis of no significant difference in sensory receptor preference of the three groups was not rejected. However, qualitative appraisal of the GSR print-outs yielded highly individual patterns of response between each of the groups, and suggested that the autistic subjects showed a deficit in the ability to make cross-modal, discrimination, and generalization associations necessary for more normal cognitive development.

Conclusions

Within the past thirty years the syndrome of Early Infantile Autism was first identified, then observed, researched, medically treated, and neurophysiologically studied to the extent that it was concluded that autism did exist as a specific clinically and behaviorally defined syndrome. The source of this syndrome was manifest at birth or shortly thereafter and remained throughout the lifetime of the person. The current state of research strongly indicated that

an underlying neuropathophysiological process was involved, that no known factors in the psychological environment could have caused autism, and that no etiologically based treatment appeared to alter the course of this syndrome. The administration of psychotropic medications, which can alter the course of schizophrenia and/or psychotic ideation had no effect on the course of autism, except to reduce symptomatic behaviors such as hyperactivity and agitation. Therefore, prognosis remained currently poor for the diagnosed autistic patient.

The extreme lack of cooperation exhibited by most autistic children had limited the scope of further research into the possible neuropathophysiological processes underlying the syndrome. However, the development of adequate treatment programs which would allow each autistic child to function optimally in a prosthetic environment will to a large extent be determined by the continued application of empirical research to this interesting and unique clinical syndrome.

Recommendations for Further Study

Several recommendations for further study evolved from this investigation. The validity of utilizing the GSR as an index of behavioral activation in autistic groups could be established through further empirical research. Additionally, autistic children who have been trained to exhibit cooperative behavioral sets could be monitored over extended periods of time and in varied situations, in order

to establish more reliable polygraphic records for this clinical group. Extended GSR monitoring in a controlled academic environment would also establish whether the trend of individual response patterns noted in the present investigation would support or refute individualized instruction. It appeared in this investigation that each diagnosed autistic child exhibited sensory preferences and/or deficits on an individual basis, even though the common denominator of extreme vaso-motor instability was noted in all autistic GSR protocols.

Current research which supported an underlying neuropathophysiological process in the autistic syndrome could be further delineated by utilizing the GSR in programmed learning situations. Generalization, discrimination, transfer of training and cross-modal learning exercises could be developed and applied while the child is being monitored. In this way, the autonomically registered GSR response could be measured and the level of acquisition of concepts could then be more objectively measurable.

REFERENCES

Bakwin, H. Early infantile autism. Journal of Pediatrics, 1954, 45, 492-497.

Barland, G.H. and Raskin, D.C. Detection of deception. In W.F. Prokasy & D.C. Raskin (Eds.), Electrodermal activity in psychological research. New York: Academic Press, 1973.

Bender, L. Childhood schizophrenia: Clinical study of one hundred schizophrenic children. American Journal of Orthopsychiatry, 1947, 22, 40-55.

Bender, L. Childhood schizophrenia. Psychiatric Quarterly, 1953, 27, 663-681.

Bender, L. D-lysergic acid in the treatment of the biological features of childhood schizophrenia. Diseases of the Nervous System, 1966, 7, 43-46.

Bender, L. Childhood schizophrenia: A review. Journal of Hillside Hospital, 1967, 16, 10-22.

Bender, L. The nature of childhood psychosis. In J.G. Howells (Ed.), Modern perspectives in international child psychiatry. Edinburgh: Oliver & Boyd, 1969.

Bernal, M.E. & Miller, L.H. Electrodermal and cardiac responses of schizophrenic children to sensory stimuli. Psychophysiology, 1971, 7, 155-168.

Bettelheim, B. The empty fortress: Infantile autism and the birth of the self. New York: Free Press, 1967.

Bettelheim, B. A home for the heart. New York: Alfred A. Knopf, 1974.

Brown, B.B. New mind, new body. New York: Harper & Row, 1974.

Bryson, C.Q. Systematic identification of perceptual disabilities in autistic children. Perceptual and Motor Skills, 1970, 31, 239-246.

Clancy, H., Dugdale, A., & Rendle-Short, J. Diagnosis of infantile autism. Developmental Medicine and Child Neurology, 1969, 11, 432-442.

Cohen, D. Childhood autism and atypical development. Taboroff Memorial Lecture, 1975. Salt Lake City, Utah: University of Utah School of Medicine.

Creak, M. Schizophrenic syndromes in childhood. Developmental Medicine and Childhood Neurology, 1964, 4, 530-535.

Darrow, C.W. Differences in the physiological reactions to sensory and ideational stimuli. The Psychological Bulletin, 1929, 26 (4), 185-201.

Darrow, C.W. Problems in the use of GSR as an index of cerebral function. Psychophysiology, 1967, 3 (4), 389-396.

DeMyer, M.K., Churchill, K.W., & Pontius, W. A comparison of five diagnostic systems for childhood schizophrenia and infantile autism. Journal of Autism and Childhood Schizophrenia, 1971, 1, 175-189.

Despert, J.L. Some considerations relating to the genesis of autistic behavior in children. American Journal of Orthopsychiatry, 1951, 21, 335-350.

DSM-II. Diagnostic and Statistical Manual of Mental Disorders (2nd ed.) Prepared by the Committee on Nomenclature and Statistics of the American Psychiatric Association. Washington, D.C.: American Psychiatric Association, 1968.

Eisenberg, L. The course of childhood schizophrenia. AMA Archives of Neurology and Psychiatry, 1957, 78, 69-83.

Eisenberg, L. The classification of childhood psychosis reconsidered. Journal of Autism and Childhood Schizophrenia, 1972, 2 (4), 338-342.

Ferster, C.B. Positive reinforcement and behavioral deficits of autistic children. Child Development, 1961, 32, 437-456.

Ferster, C.B. The repertoire of the autistic child in relation to principles of reinforcement. In L. Goilschalk & A.H. Auerbach (Eds.), Methods of research in psychotherapy. New York: Appleton-Century-Crofts, 1966.

Franknoi, J., & Ruttenberg, B.A. Formulation of the dynamic economic factors underlying infantile autism. Journal of the American Academy of Child Psychiatry, 1971, 10 (4), 713-738.

Frith, U. Studies in pattern detection in normal and autistic children. Journal of Experimental Child Psychology, 1970, 10, 120-135.

Gildard, F.A. Human senses. New York: John Wiley & Sons, Inc., 1972.

Goldfarb, W. Receptor preference in schizophrenic children. Archives of Neurology and Psychiatry, 1956, 76, 643-652.

Goldfarb, W. Childhood schizophrenia. Cambridge, Mass.: Harvard University Press, 1961.

Grey-Walter, W. Report on neurophysiological correlates of apparent defects of sensory-motor integration in autistic children. NIMH Research Fund Report, 1964.

Grey-Walter, W., Aldridge, U.J., Cooper, R., O'Gorman, G., McCallum, C., & Winter, A.L. Neurophysiological correlates of apparent defects of sensory-motor integration in autistic children. In D. Churchill, G. Alpern, & M. DeMyer (Eds.), Infantile Autism: Proceedings of the Indiana University Colloquium. Springfield, Ill.: C. C. Thomas, 1971.

Hermelin, B., & O'Connor, N. Crossmodal transfer in normal, subnormal, and autistic children. Neuropsychologia, 1964a, 2, 229-235.

Hermelin, B., & O'Connor, N. Effects of sensory input and sensory dominance on severely disturbed, autistic children and on subnormal controls. British Journal of Psychology, 1964b, 56, 455-460.

Hermelin, B., & O'Connor, N. Perceptual and motor discrimination in psychotic and normal children. Journal of Genetic Psychology, 1967, 110, 117-125.

Hermelin, B., & O'Connor, N. Measures of the occipital alpha rhythm in normal, subnormal, and autistic children. British Journal of Psychiatry, 1968, 114, 603-610.

Hermelin, B., & O'Connor, N. Psychological experiments with children. London: Pergamon, 1970.

Hingtgen, J.H., & Bryson, C.Q. Recent developments in the study of early childhood psychosis: Infantile autism, childhood schizophrenia, and related disorders. Rockville, Md.: National Institute of Mental Health, DHEW Publication No. (HSM) 71-9062, 1971.

Hingtgen, J.N., & Churchill, D.W. Identification of perceptual limitations in mute autistic childre: Identification by use of behavior modification. Archives of General Psychiatry, 1969, 21, 68-71.

Hingtgen, J.N., & Churchill, D.W. Differential effects of behavior modification in four mute autistic boys. In D. W. Churchill, G.D. Alpern, & M. DeMyer (Eds.), Infantile Autism: Proceedings of the Indiana University Colloquium. Springfield, Ill.: C.C. Thomas, 1971.

Hutt, C., & Hutt, S.J. Effects of environmental complexity upon sterotyped behaviors in children. Animal Behavior, 1965, 13, 1-4.

Hutt, S.J., & Hutt, C. Stereotype, arousal, and autism. Human Development, 1968, 11, 277-286.

Kanner, L. Autistic disturbances of affective contact. Nervous Child, 1943, 2, 217-250.

Kanner, L. Childhood psychosis: An historical overview. Journal of Autism and Childhood Schizophrenia, 1971a, 1 (1), 14-19.

Kanner, L. Followup study of eleven autistic children originally reported in 1943. Journal of Autism and Childhood Schizophrenia, 1971b, 1 (2), 119-145.

Koegel, R., & Wilhelm, H. Selective responding to the components of multiple visual cues by autistic children. Journal of Experimental Child Psychology, 1973, 15, 442-453.

Kolvin, I. Psychosis in childhood--a comparative study. In M. Rutter (Ed.), Infantile autism: concepts, characteristics, and treatment. London: Churchill, 1971.

Lader, M.& Wing, L. Habituation of the psycho-galvanic reflex in patients with anxiety states and in normal subjects. Journal of Neurology, Neurosurgery & Psychiatry, 1964, 24, 210-218.

Lovaas, O.I. Considerations in the development of a behavioral treatment program for psychotic children. In D. Churchill, G. Alpern, & M. DeMyer (Eds.), Infantile Autism: Proceedings of the Indiana University Colloquium. Springfield, Ill.: Charles C. Thomas, 1971.

Lovaas, O.I., & Koegel, R. Behavior therapy with autistic children. In C. Thoresen (Ed.), Behavior modification in education. Chicago, Ill.: The University of Chicago Press, 1973.

Lovaas, O.I., Koegel, R., Simmons, J.Q., & Long, J.S. Some generalization and followup measures on autistic children in behavior therapy. Journal of Applied Behavior Analysis, 1973, 6, 131-166.

Lovaas, O.I., & Schreibman, L. Stimulus overselectivity of autistic children in a two stimulus situation. Behavior Research and Therapy, 1971, 9 (4), 305-310.

Lovaas, O.I., Schreibman, L., Koegel, R., & Rehm, R. Selective responding by autistic children to multiple sensory input. Journal of Abnormal Psychology, 1971, 77, 211-222.

Mahler, M.S. On child psychosis and schizophrenia: Autistic and symbiotic psychosis. Psychoanalytic Study of the Child, 1952, 7, 286-305.

Mahler, M.S. On early infantile psychosis: The symbiotic and autistic syndromes. Journal of the American Academy of Child Psychiatry, 1965, 4, 554-568.

Mandler, G., Mandler, J., & Uviller, E. Autonomic feedback: The perception of autonomic activity. Journal of Abnormal and Social Psychology, 1958, 56, 376-383.

O'Connor, N., & Hermelin, B. Measures of distance and motility in psychotic children and severely subnormal controls. British Journal of Social and Clinical Psychology, 1964, 3, 29-33.

O'Connor, N., & Hermelin, B. Sensory dominance in autistic imbecile children and controls. Archives of General Psychiatry, 1965, 12, 99-103.

O'Connor, N., & Hermelin, B. Auditory and visual memory in autistic and normal children. Journal of Mental Deficiency Research, 1967, 11, 126-131.

O'Connor, N., & Hermelin, B. Cognitive deficits in children. British Medical Bulletin, 1971, 27 (3), 227-232.

Ornitz, E.M. Disorders of perception common in early infantile autism and schizophrenia. Comprehensive Psychiatry, 1969, 10 (4), 259-274.

Ornitz, E.M., & Ritvo, E.R. Neurophysiologic mechanisms underlying perceptual inconstancy in autistic and schizophrenic children. Archives of General Psychiatry, 1968, 19, 22-27.

Ornitz, E.M., & Ritvo, E.R. The syndrome of autism: A critical review. American Journal of Psychiatry, 1976, 6, 609-621.

Ornitz, E.M., Ritvo, E.R., & Brown, M.D. The EEG and rapid eye movements during REM sleep in normals and autistic children. Electroencephalography and Clinical Neurophysiology, 1969, 26, 167-175.

Paintal, A.S. A comparison of the GSR of normals and psychotics. Journal of Experimental Psychology, 1951, 41, 425-428.

Rank, B. Adaptation of the psychoanalytic technique for the treatment of young children with atypical development. American Journal of Orthopsychiatry, 1959, 19, 130-139.

Rendle-Short, J., Clancy, H.G., & Dugdale, A. Infantile autism. Medical Journal of Australia, 1968, 1, 921-922.

Rimland, B. Infantile autism. New York: Appleton-Century-Crofts, 1964.

Rimland, B. On the objective diagnosis of infantile autism. Acta Paedopsychiatricia, 1968, 35, 146-161.

Rutter, M. Psychotic disorders of early childhood. In A.J. Coppen, & A. Walk (Eds.), Recent developments in schizophrenia: A symposium. London: R.M.P.A., 1967.

Rutter, M. Concepts of autism: A review of research. Journal of Child Psychology and Psychiatry, 1968, 9, 1-25.

Rutter, M. The development of infantile autism. Psychological Medicine, 1974, 4 (2), 147-163.

Rutter, M. The description and classification of infantile autism. In D. Churchill, G. Alpern, & M. DeMyer (Eds.), Infantile Autism: Proceedings of the Indiana University Colloquium. Springfield, Ill.: Charles C. Thomas, 1971.

Rutter, M., & Bartak, L. Causes of infantile autism: Some considerations from recent research. Journal of Autism and Childhood Schizophrenia, 1971, 1 (1), 20-32.

Rutter, M., Bartak, L., & Newman, S. Autism--a central disorder of cognition and language? In M. Rutter (Ed.) Infantile autism: Concepts, characteristics, and treatment. London: Churchill, 1971.

Rutter, M., & Lockyer, L. A five to fifteen-year followup study of infantile psychosis I: Description of sample. British Journal of Psychiatry, 1967, 113, 1169-1182.

Satterfield, J., & Dawson, M. Electrodermal correlates of hyperactivity in children. Psychophysiology, 1971, 8 (2), 191-197.

Schopler, E. The relationship between early tactile experience and the treatment of an autistic and schizophrenic child. American Journal of Orthopsychiatry, 1964, 34, 339-340.

Schopler, E. Early infantile autism and receptor processes. Archives of General Psychiatry, 1965, 13, 327-335.

Schopler, E. Visual vs. tactile receptor preference in normal and schizophrenic children. Journal of Abnormal Psychology, 1966, 71, 108-114.

Schopler, E., Brehm, S., & Kensbourne, M. Effect of treatment structure on development in autistic children. Archives of General Psychiatry, 1971, 24, 415-521.

Small, J. Sensory evoked responses of autistic children. In D. Churchill, G. Alpern, & M. DeMyer (Eds.), Infantile Autism: Proceedings of the Indiana University Colloquium. Springfield, Ill.: C.C. Thomas, 1971.

Spence, J., Underwood, B., Duncan, C., & Cotton, J. Elementary statistics. New York: Appleton-Century, Crofts, 1968.

Stoelting Polyscribe Manual of Operating Instructions, Catalogue #224870B. Chicago: Stoelting Co., 1976.

Stroh, G., & Buick, D. Perceptual development and childhood psychosis. British Journal of Medicine and Psychology, 1964, 37, 291-299.

Venables, P.H. The effect of auditory and visual stimulation on the skin potential response of schizophrenics. The Brain, 1960, 83, 77-92.

Venables, P.H., & Christie, M.J. Mechanisms, instrumentation, recording techniques, and quantification of responses. In W.F. Prokasy & D.C. Raskin (Eds.), Electrodermal activity in psychological research. New York: Academic Press, 1973.

Wheeler, D.E. Notes on proper posture for a polygraph examination. In Stoelting Co. Catalogue #22498-B, 1976.

Wing, J.K. Diagnosis, epidemiology, aetiology. In J.K. Wing (Ed.), Early childhood autism: Clinical, educational, and social aspects. London: Pergamon Press, 1966.

Wing, L., & Wing, J. Multiple impairments in early childhood autism. Journal of Autism and Childhood Schizophrenia, 1971, 1 (3), 256-266.

APPENDIX A

REVIEW OF THE RELEVANT LITERATURE ON THE ETIOLOGY OF EARLY INFANTILE AUTISM

REVIEW OF THE RELEVANT LITERATURE ON THE ETIOLOGY OF EARLY INFANTILE AUTISM

Non-organic theories.

The psychoanalytic school had produced various dynamic formulations for a theory of infantile autism and/or childhood psychoses. Although all of these non-organic theories attributed the development of psychotic behavior to pathogenic interaction of parent and child, they differed in interpretation of the deviancy in this interaction. Critical periods of early development during which the child was unusually susceptible to parental mismanagement also varied with the particular stance.

Franknoi and Ruttenburg (1971) postulated that the autistic child had an inborn vulnerability to stimulation and an increased need for a sense of security. The mother, unable to respond to her infant's increased needs, caused the infant to shut himself off from his surroundings and engage in autoerotic and autoaggressive behavior. Their assumption, therefore, was that autism was a defensive position which the "congenitally vulnerable child" (p. 713) gradually resolved for himself as a way of coping with stress. The mother, under normal circumstances, assumed the

responsibility of providing her infant adequate protection from such trauma.

The overpossessive mother, according to Bettleheim's (1967, 1974) dynamic interpretation, anticipated her biologically intact child's every need and thereby removed the chance for the exploration, differentiation, and experimentation that allowed the child to interact with his world. The child thus gave up and withdrew, becoming autistic. Rank (1949) similarly postulated parental pathology which led to emotional deprivation of the infant as the etiological agent.

Ferster (1961, 1966) presented a dynamic, behavioral interpretation of autistic etiology in which parents failed to interact with their infant in such a way that the infant did not pair primary reinforcers with a wider range of behaviors. In this way, the parental responses of a social nature failed to become generalized and the child thus developed little or no appropriate social behavior. The repetitive, autistic behaviors were maintained because the self-stimulation provided continuous and immediate self-reinforcement.

Theories of infantile autism based on non-organic etiology were all similar in that the emphasis on parental pathology was viewed as the primary cause of behavioral deficits (deviations) in the child. The assumption was that this early interaction of infant and mother in particular, was so traumatic or severe that the child of biological integrity who was physically normal at birth was thus prevented from

establishing effective interaction with his social and physical environment.

Studies by Kolvin (1971) and Franknoi and Ruttenburg (1971) attempted to delineate which, if any, parental behaviors were identifiable as defects both necessary and sufficient to have caused autism. While Franknoi and Ruttenburg have continued to maintain a psychogenic approach, Kolvin's study concluded that there was no evidence of any such abnormalities in family functions or relationships. The only exception thus far identified when research techniques were used (rather than observation alone) was the consistent finding that the parents included a disproportionate number of professional, middle-class individuals (Rutter & Bartak, 1971; Kolvin, 1971). The incidence of familial pathology in studies by Rutter (1967), Rutter and Lockyer (1967), Rutter, Bartak, and Newman (1971), and Kolvin (1971) had in fact been found to be consistently lower in parents of autistic children (as defined separately from other childhood psychosis) in comparison to control groups. In addition, these dynamic and behavioral theories have been unable to account for the fact that other children subjected to severe environmental stress of a similar type and duration developed into normal adults.

In summary, the present state of research into etiological considerations of autism supported psychogenic, non-organic factors as important only in their effect on the course of the disorder, but not essential in the role of

causation; while the efficacy of employing a behavioral approach as an intervention technique remained under empirical and longitudinal investigation.

Organic-experiential theories.

Another global theoretical framework which explained the etiological considerations precipitous of the childhood psychoses stressed the interaction of organic factors with experiential events. Within this group, there were those theories which were similar to nonorganic, psychodynamic theories and emphasized pathogenic mother-child relationships, but also included organic factors. There were also those theories which placed major emphasis on organic components in stressing deviations inherent in the child for which the mother, in psychodynamic terms, failed to compensate.

Goldfarb (1956 and 1961) made a distinction between organic and nonorganic childhood schizophrenia on the basis of congenital brain damage and/or environmental inadequacy as causative of severe deficits in ego development. His study remained of historical importance in that it was among the first to identify systematically the presence of congenitally determined cognitive defects capable of causing clinical variants of the childhood psychoses. The central hypothesis of Goldfarb's (1961) study was that schizophrenic children experienced a failure in the development of the usual hierarchy of receptor preferences. His observations showed that schizophrenic children made use of contact or near receptors in preference to the use of distal, far

receptors. He further observed that schizophrenic children fragmented their perceptual world by excluding or selectively experiencing one part of the world in preference to another.

Schopler (1965, 1966, and Schopler, Brehm & Kensbourne, 1971) had found that the "hyposensitivity" in childhood autism and "hypersensitivity" of childhood schizophrenia was a result of a specific dysfunction in the arousal and inhibition of sensory processes. Schopler's (1966) study of receptor preference in schizophrenic children explored their visual versus tactile receptor preference and found that the schizophrenic children showed significantly less visual preference than the same aged normals, thus confirming Goldfarb's (1961) receptor hypothesis. This information was consistent with the view that infantile autism involved early sensory deprivation which resulted from an interaction between a constitutional deficiency which inhibited certain reticular arousal functions and mothering that tended to be understimulating. Schopler's earlier (1964) clinical observations suggested that schizophrenic children made poor use of visual and auditory cues and excessively relied on taste, touch, and smell for orientation to their environment.

Although Mahler (1952, 1965) placed primary emphasis on a constitutional vulnerability of the autistic child, the child's basic failure to associate the mother as an external love object resulted in the lack of development of basic ego functions and retraction of affective contact. Autism therefore became the mechanism by which the child attempted to

shut out potential sources of sensory perception, particularly those which required affective response. Mahler viewed autism as a defense against anxiety from environmental stress which acted in combination with the effects of central nervous system pathology to produce the syndrome of autism. She further stated that her clinical observations included findings similar to Schopler's (1965) with reference to the schizophrenic child's over-reliance on near receptor process.

Stroh and Buick (1964) were more specific in their emphasis on perceptual dysfunction and postulated that the constitutional and/or environmental deficits resulted in arrested perceptual development. Further, they postulated that autistic behaviors represented the psychotic child's attempt to adapt to the environment and integrate incoming stimuli within his very limited perceptual system.

In Kanner's earliest (1943) formulation of a theory of Infantile Autism he ascribed its etiology to an "innate ability, an inborn autistic disturbance of affective contact," (p. 217) but also cited the cold, rejecting parent as a primary factor. More recently, Kanner (1971a and 1971b) had placed less emphasis on parental rejection and regarded parents from the view of "mutuality," thus stressing their involvement in the therapeutic efforts, rather than remaining the "etiological culprits" of the psychodynamic approach. He recommended that parental involvement include instructional programs which can more effectively meet this child's needs. Kanner (1971b) felt that all experimental and

heuristic therapeutic endeavors in this vein should be checked by controlled longitudinal and followup studies.

Eisenberg (1957, 1972) supported Kanner's view of autism as an interaction of biochemical or genetic endowment and environmental experience. He further postulated splitting autistic children into two groups: a neurologically damaged population called symptomatic autism, and a group exhibiting "Infantile Autism" in Kanner's sense.

Lovaas applied behavioral principles of reinforcement theory to psychotic children's failures in the acquisition of symbolic rewards, as evidenced by their paucity of social behavior and lack of interaction or response to environmental stimulation. Lovaas approached these children through the framework of educational intervention, rather than etiological reference, and trained them to attend to social stimuli. He utilized personal contacts as symbolic rewards in association with the termination of pain (both aversive conditioning for self-destructive behaviors and pathological interactions), training for attending to social stimuli through primary food reinforcers, and teaching first imitative then meaningful, contextual speech (Lovaas, 1971). Perhaps Lovaas' techniques of employing behavior modification as an intervention technique in the treatment of psychotic children remained of such importance because it was among the only empirically measured remediation techniques which demonstrated significant effectiveness (Lovaas & Koegel, 1973).

An outstanding behavioral feature Lovaas noted in the educational treatment of psychotic and autistic children was their deviation from normal perceptual functioning, namely in responding to multiple stimulus inputs. This observation led a group of authors to undertake empirical hypothesis testing in this vein.

Lovaas, Schreibman, Koegel, and Rehm (1971) reinforced autistic, retarded, and normal children for responding to a simultaneously presented complex of stimuli of auditory, visual, and tactile cues. Once discriminations were established, elements of the complex were presented separately, yielding findings that autistics responded primarily to only one of the cues, normals responded uniformly to all three cues, and retardates functioned between the two extremes. Although the data of this experiment failed to support notions that any one sense modality was impaired in autistic children, it did indicate that this clinical group tended toward attention which was overselective.

A subsequent study of autistic and normal children by Lovaas and Schreibman (1971) which used a stimulus complex of an auditory and visual component yielded consistent results with regard to autistic children's demonstrated stimulus overselectivity.

Koegel and Wilhelm (1973) conducted an experiment expanding Lovaas and Schreibman (1971) hypothesis of selective attention by autistic children by introducing cards containing two visual cues and gained results consistent with

those previously cited. Results further indicated that autistic children experienced difficulty responding to multiple cues even when both cues were within the same sensory modality. These results were compared with results yielded by normally functioning children who did not demonstrate selective responding to the stimuli employed in this experiment.

Hingtgen and Churchill (1971) conducted an empirical study utilizing a direct and intrusive approach of reinforcement technique of four mute autistic boys. Post test scores indicated that there was a substantial increase in imitative behavior for all subjects. It was concluded that intensive imitative training with elevated motivational levels was effective in increasing the cooperative set of autistic children and in expanding their behavioral repertoires. However, certain low-level behaviors were not learned, suggesting as did Lovaas, Schreibman, Koegel, and Rehm (1971) that a fundamental disturbance in perceptual processes was present.

Theories of autism utilizing the interaction of organic and environmental factors as causative had broadened this area of research by including variables other than parental pathology which were subject to quantifiable, rather than observational, measurement. Yet, these theories failed to define either the nature of the underlying CNS vulnerability or the nature of the environmental trauma. Further, the degree to which each factor contributed to the pathology was undetermined. If only stress of undetermined amounts

produced autistic behavior in a child of congenital vulnerability, perhaps the frequency of the autistic syndrome would have been greater. For those children who exhibited thought and affective disorders, drug, milieu, and psychotherapy application yielded significant results, while children meeting the criteria for autism responded best to a combination of behavior therapy and special education (Schopler, et al., 1971; Lovaas, Koegel, Simmons, & Long, 1973).

Organic theories.

Bender (1947) was among the first authors to suggest pathology at every level of central nervous system functioning as the causative factor of abberant behaviors within the clinical group of the childhood psychoses. Bender (1953) maintained the nosological stance that all behavioral manifestations noted were organically determined variants of the clinical classification of childhood schizophrenia. Her strict organic view was further elaborated (1966, 1967) as an inherited vulnerability and a maturational lag at the embrionic level in CNS integration. Consequently, Bender's schizophrenic child was unable to perceive reality clearly and retreated to autistic like behaviors as a defensive maneuver of coping with the anxiety generated from CNS disruption. Bender, unlike the following researchers, had postulated her theory on the results of longitudinal clinical observations of a small group of heterogeneous children, most of whom did not meet the Kanner criterion for autism.

The recent application of research principles to the study of autism and/or childhood psychoses had led one group of authors to support a strict organic base for autism's etiology as a result of empirical investigation. Prior to the early 1960's, behaviors were explained almost entirely through clinical observation, with little empirical evidence generated as to the cause. The organic hypothesis of innate defects in the various physiological arousal mechanisms of this clinical group which interfere with the ability to process incoming stimuli was hypothesized by Hutt and Hutt, Rimland, Ornitz and Ritvo, and Hermelin, O'Connor, and Frith.

Hutt and Hutt (1965, 1968) suggested that a chronically high level of cortical arousal caused the autistic child to engage in stereotyped behaviors which served an arousal-reducing function by producing repetitive endogenous stimulation and blocking of sensory input. These authors noted further that stereotyped movements increased with increasing environmental complexity, i.e., novel situations, involvement with strangers, and novel objects introduced in a familiar environment.

Rimland's (1964, 1968) reticular formation theory postulated that a malfunction in neurogenic processes produced inadequate cortical arousal to incoming stimuli. This resulted in the autistic child's inability to relate any new stimuli to remembered stimuli. Further speculation of this theory was that the malfunction of the reticular formation was a result of genetic homozygous vulnerability in the

gene related to the ability to focus attention and ignore distraction. This inadequate level of arousal to incoming stimuli at the level of infancy disallowed associations of biological rewards with social, in particular maternal, relationship. Therefore, the acquisition of an increasingly more complex behavioral repertoire in the autistic child was inhibited.

The empirical and observational studies Ornitz (1969) and Ornitz and Ritvo (1968) conducted with psychotic children had led them to formulate a neurophysiological hypothesis concerning the etiology of this group of psychiatric disorders. In January 1968, they postulated that the developmental failures were associated with and explained by the psychotic child's inability to maintain constancy of perception. They further postulated that random underloading or overloading of CNS functions causes these children to perceive identical percepts differently each time they are perceived, with resultant random perceptual confusion. This abnormal physiological state involved degrees of excitation, facilitation, and inhibition of information and interacted with the hypo-hypersensitivity characteristic of this clinical group. Following REM sleep studies later that same year, these authors inferred a central pathophysiology of the vestibular system with a resultant break through into waking life of the phasic excitatory and inhibitory influences of REM sleep (Ornitz, et al., 1969).

Ornitz (1969) suggested that the results of clinical, psychological, psychophysiological, and neurophysiological investigations pointed to a malfunction of the homeostatic regulation of sensory input in both early infantile autism and in childhood schizophrenia. Ornitz concluded that a profound disturbance in perception was found in both clinical groups and that this perceptual dysfunction may underly the clinical pathology.

These findings based on physiological arousal did not support those of either the Hutts or Rimland who hypothesized different and specific direction of arousal, while Ornitz hypothesized that the deviation involved alternating fluctuations in arousal level. Further, Ornitz and Ritvo (1968), in preliminary findings, failed to support the Hutt's suggestion of autistic children's high arousal with increasing stereotypes which resulted from the exposure to more complex environments.

Hermelin and O'Connor (1968, 1970) also found results which tended to disprove the Hutt's high arousal postulation. These researchers found that in a resting state, autistic children did not differ significantly from either normal or subnormal controls in their cortical arousal state as measured by alpha blocking.

Rutter (1967), in summarizing his review of research on autism, stated that infantile autism was distinct from schizophrenia, that psychogenic factors or the presence of mental subnormality were not sufficient etiological agents,

and that the role of organic brain abnormality may have been a primary influence in most cases. This author regarded the etiological hypothesis of primary defects in terms of a language or coding problem as the most promising. He further suggested that many of the manifestations of autism were explicable in terms of cognitive and perceptual defects. Rutter's (1968) and Rutter, Bartak, and Newman (1971) systematic investigations of theories which hypothesized lack of stimulation, parental rejection, or intrapsychic conflict yielded results which were largely negative with regard to autism's origin as a psychogenic disorder.

Rutter and Bartak (1971) extended Rutter's hypothesis of autism's etiology to state that a cognitive defect constituted the primary handicap in autism, the noted social and behavioral abnormalities arouse as secondary consequences. Although the cause of this cognitive defect remained unknown, these authors felt that circumstantial evidence suggested the presence of some yet undetermined organic brain dysfunction.

In defense of this cognitive deficit, Rutter conducted a series of experiments focusing on the inherent language impairments evident in autism. Compiled results of these studies indicated a lack of imaginative play (Rutter & Bartak, 1971), absence of remarkable preverbal babbling (Rutter, et al., 1971), impaired language comprehension (Rutter, 1967), and a paucity of meaningful verbalizations (Rutter, 1970) in this clinical group, and therefore

suggested a cognitive language defect. Rutter (1974) stated the hypothesized biological basis of the cognitive defect in autism required continued research so that it could be ascertained whether autism was a single disease entity, a syndrome of biological impairment, or a collection of heterogeneous symptom influences, both biological and psychosocial.

The hypothesis of a deficit in the central processing of sensory input as an explanation for much of autistic children's behavior was supported by the experimental studies of Hermelin, O'Connor, and Frith. The failure of the development of a sensory hierarchy in psychotic children was noted in a study (Hermelin & O'Connor, 1964a) which utilized bimodal combinations of light, sound, and touch stimuli presented to psychotic, normal, and subnormal control children. All groups responded most frequently to light, although the establishment of a sensory-dominance hierarchy was not noted. As a continuation of the preceeding experiment, these authors (O'Connor & Hermelin, 1964) added the variable of variation in stimulus intensity in light and sound presented simultaneously. Results yielded a tendency for visual dominance by all groups, but independent of modality, high intensity affected responses. In another experiment (1964b) using similar neurophysiological subject groups, the ability to transfer from tactile to visual tasks was measured and yielded no significant results. However, significant crossmodality effects from visual to touch were demonstrated. Results tend to indicate that crossmodal transfer ability of the kind they measured did not depend on tact cortical structure.

O'Connor and Hermelin (1967) and Hermelin & O'Connor (1967) in experiments on visual discrimination ability did not differentiate psychotic and normal children when the discrimination was a very simple one, or when no instrumental response was required. However, psychotic children showed significantly briefer visual inspection times; as a consequence of their fixation time, they perhaps gained less information than would normal children who employed longer inspection times. Thus, only more complex perceptual tasks that depended on efficient information processing might have been expected to be impaired. This impairment, they suggested, might consist of a failure to make use of complex information from any one sense, or of a failure to integrate information coming from different sensory channels.

Frith (1970), in results of a study using autistic and normal children of comparable performance levels, indicated that the autistic children were insensitive to differences in experimental structures presented and tended to impose their own stereotyped patterns, therefore a consistency with the hypothesis of an input processing deficit as the etiological agent of childhood autism was suggested.

Thus, through an analysis of specific cognitive deficits in visual and auditory perception and communication, relations between language and thought, cross-modal coding, attention, arousal and memory, Hermelin and O'Connor (1970) concluded that evidence pointed to deficits in acquisition as consistent with the autistic syndrome. This acquisitive

impairment, they further hypothesized, was due in part to the autistic child's inability to focus attention on the relevant stimulus features presented in their various experiments. Although results of these studies were not easily summarized, O'Connor and Hermelin (1971) noted the importance of a central cognitive pathological disturbance in the autistic group, manifested by the inability of autistic children to encode stimuli meaningfully or process sequential sensory inputs to make discriminations in the absence of feedback from motor responses.

Hingtgen and Bryson's (1971) and Hingtgen and Churchill's (1969, 1971) hypotheses of inadequate short-term memory and cross-modal information processing capacities suggested that perceptual disabilities underly the intellectual and the language development of psychotic children and may have contributed to the development of bizarre behavior patterns. Their experimental results also suggested that the basic difficulty in infantile autism was not an avoidance of auditory and visual stimuli, per se, but rather a deficit in ability to make cross-modal associations necessary for further perceptual and language development.

Wing and Wing (1971) in a statement of clinical observation, noted that autistic children appeared to suffer from multiple impairments (with a variation in severity) affecting comprehension and use of speech and gesture, visual and auditory perception, posture, autonomic function, and control of skilled movements. When these symptoms

occurred together, the concomitant behavior patterns were typified in the childhood autism syndrome. These "multiple impairments" (p. 256) did not necessarily mean multiple etiologies. The Wings hypothesized that many different brain functions were affected by an organic lesion due to a single genetic, biochemical or anatomical abnormality (J. Wing, 1966). Refutation or confirmation of this multiple-handicap hypothesis depended upon the development of more adequate techniques for examination of the central nervous system and a better understanding of the neurophysiology of language reception and expression.

In an unpublished manuscript prepared for oral presentation, Cohen (1975) reported that in one of every three thousand live births worldwide, primary autism was noted. He stated that this incidence of autism was noted in every social class, and that in two sets of identical twins he had studied there had been a one hundred percent rate of concordance for autism. These isolated and global findings, Cohen suggested, gave much support for a congenital, organic, or metabolic abnormality in the uniform clinical picture of primary autism.

With the inclusion of organically based theories to delimit the etiology of the childhood psychoses, a variety of neurogenic involvements had been hypothesized. Included in these had been postulates of defects in arousal, sensory hierarchy, and integrative mechanisms. Recent experimental data concluded that although there was neurobiological damage

present in a large number of psychotic children (DeMyer, Churchill, & Pontius, 1971) until more sophisticated technological means of measuring specific physiological and biochemical parameters are developed, there was not sufficient evidence to support any one organic theory. Present consideration of data did, however, suggest strongly that there was a multiplicity of subtle forms of organic disease present which resulted in similar maladaptive behaviors associated with all subtypes of the childhood psychoses (Creak, 1964). Advanced experimental data was first required on the neurological processes involved in the acquisition of learning, language development, and developmental organic processes of normal children so that a measurable means of the deviancy from the norm were established.

Theories of infantile autism and the childhood psychoses which suggested "normal potential" had, with recent research and longitudinal study, been discounted. The current status of outcome from a severely disruptive childhood which involved psychotic/autistic manifestations indicated that although bizarre behaviors diminished and social relatedness increased, gross deficiencies in intellectual, perceptual, and language development remained (Kanner, 1971b; Rutter, 1974; Bryson, 1970; Hingtgen & Bryson, 1971). It would now appear that the "deficits" in social behavior first noted by Kanner were less a cause than a reflection of multiple developmental deviations of yet undetermined, organic origin.

APPENDIX B

CLANCY, DUGDALE, RENDLE-SHORT CRITERION FOR INFANTILE AUTISM (1968)

CLANCY, DUGDALE, RENDLE-SHORT CRITERION

FOR INFANTILE AUTISM (1968)

A diagnosis of infantile autism is considered when at least seven of the following criteria are present and the symptoms begin within the first three years of life. In decreasing order of frequency, the symptoms are as follows:

1. Great difficulty in mixing and playing with other children.
2. Acts as if deaf--does not react to speech or noise.
3. Strong resistance to any learning--either new behavior or new skills.
4. Lack of fear about realistic dangers, e.g., may play with fire.
5. Resist change in routine--the smallest change may produce disproportionate anxiety.
6. Prefers to indicate needs by gestures, speech may or may not be present.
7. Laughs and giggles for no apparent reason.
8. Not cuddly as a baby.
9. Marked physical overactivity.

10. No eye contact, persistently looks past or turns away from persons, especially when spoken to.
11. Unusual attachment to a particular object or objects.
12. Spins objects, especially round ones.
13. Repetitive and sustained odd play, e.g., rattling stones in a can.
14. Standoffish manner, treats persons as objects rather than as persons.

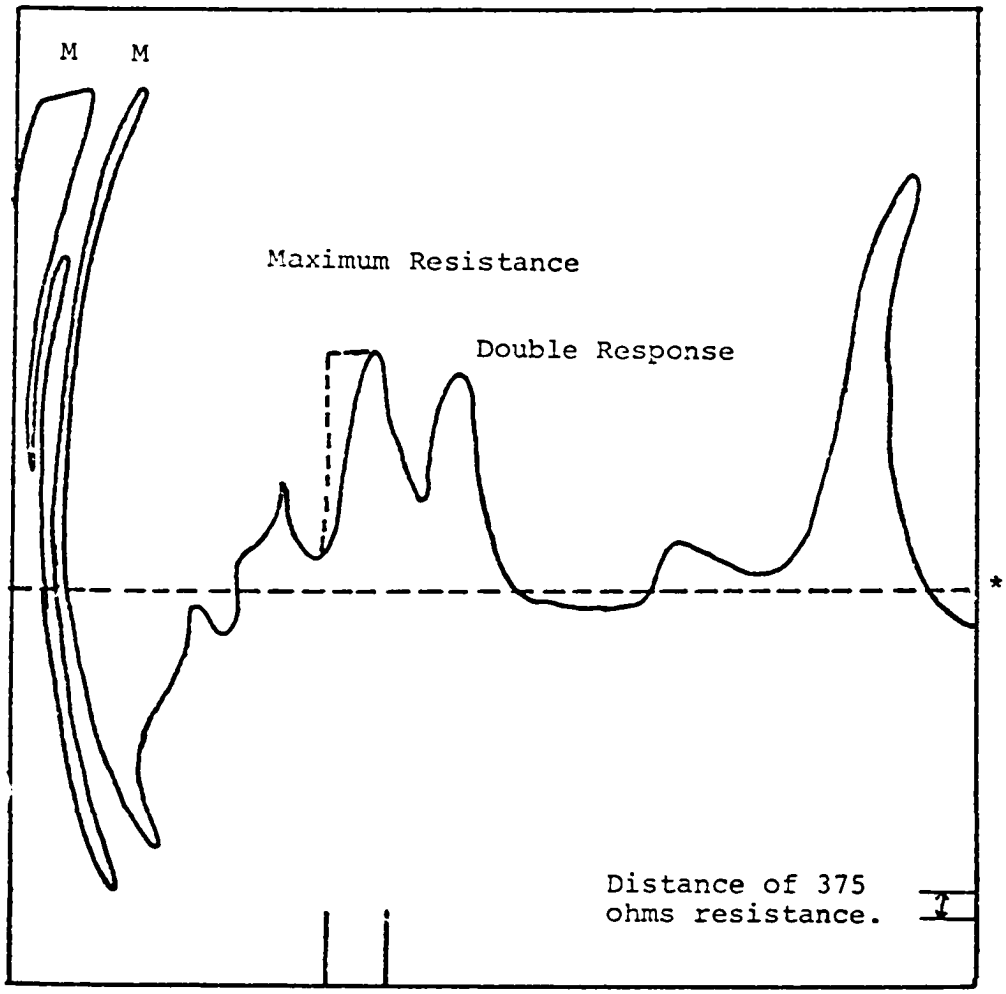
APPENDIX C

SCORABLE RESPONSES AND MOVEMENT ARTIFACTS ILLUSTRATED

GSR

SCORABLE RESPONSES AND MOVEMENT

ARTIFACTS ILLUSTRATED



*Arbitrary Baseline Sensitivity at 3500 ohms, Stoelting Polyscribe