AROUSAL PATTERNS AND STIMULUS BARRIER FUNCTIONING IN SCHIZOPHRENIA

by

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Submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy
under the Executive Committee of the Graduate
School of Arts and Sciences
Columbia University

1976

ABSTRACT

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Recent research into arousal in schizophrenics points to the existence of two reciprocally functioning systems whose normal balance is disrupted--resulting in two different arousal patterns: one hyperaroused and the other hypoaroused.

The present research further examined these arousal patterns and their relationship to stimulus barrier--viewed as a complex, active ego function.

There were two experimental groups of schizophrenics--inpatients with a history of chronic psychiatric hospitalization (averaging 11.9 years), and outpatients with no more than one year accumulated psychiatric hospitalization; and a control group with no history of any psychiatric difficulties. There were 20 subjects in each group.

Each subject was tested twice, six weeks apart, with the following procedures being administered in order:

1. Beck Depression Inventory (Beck et al., 1961).
2. Stimulus Barrier Interview (Bellak et al., 1973).
3. GSR characteristics were measured in response to a series of 15 tones of moderate intensity.

4. Structured Clinical Interview (Burdock & Hardesty, 1969).

5. Taylor Scale of Manifest Anxiety (1953).

In addition, demographic, pharmacological, and institutionalization data were gathered from the patients' records.

As predicted, all schizophrenics exhibited one of two patterns: an overresponder pattern, with an elevated skin conductance level, no habituation within the test period, and a high incidence of spontaneous fluctuations of conductance (and, in the case of outpatients, abnormally high response amplitudes); and an underresponder pattern, with a depressed skin conductance level, either no responses at all or an isolated trial-one response, and a low incidence of spontaneous fluctuations. These patterns were markedly different from the normal habituation pattern exhibited by all of the controls: moderate baseline levels, with 3-8 orienting responses, followed by habituation to criterion.

The predicted correlation between response pattern and Stimulus Barrier Ratings was found to be highly significant --with overresponders being rated more pathological.

An exploration of the factors relating to these response patterns found that outpatient overresponders were more anxious (both by clinical observation and their own report), while underresponders were rated higher on a scale of Lethargy-Dejection, but did not report more subjectively experienced depression. In the inpatient sample, these differences seemed to be obscured by the effects of insti-

tutionalization. Differences in medication, hospitalization, and clinical picture were insignificant, except for a trend towards chronic undifferentiated schizophrenics being underresponders.

As predicted, certain schizophrenics alternated between response patterns--although at no time did any schizophrenic exhibit a normal habituation pattern. Changers were more common among outpatients.

The alternation between patterns seemed to serve an adaptive function for schizophrenics--suggesting an attempt at modulation of stimulation otherwise not open to them. Changers exhibited less extreme skin conductance levels, less accumulated institutionalization and lower levels of psychotropic medication. All changers exhibited an initial trial-one response in their underresponder phase --also suggestive of a more modulated approach. They also appeared to be more affectively alive--being rated more pathological with respect to anxiety (both objectively observed and subjectively reported), Anger-Hostility, and Self-Depreciation. Nevertheless, changers were less pathological with respect to Incongruous Behavior, suggesting a more adaptive capacity to express their conflicts.

The results of the present research support the view that stimulus barrier dysfunction and maladaptive patterns of arousal are two ways of viewing the same phenomenon, and that this defect is deeply implicated in the etiology of schizophrenia--particularly with regard to the schizo-

phrenic's unstable ego boundaries and difficulty in maintaining a modulated relationship with his environment.

The results suggest that differential treatment according to response pattern is needed in helping schizophrenics to recompensate.

Further research--particularly of a longitudinal type--into the vicissitudes of response patterns in schizophrenics is strongly indicated.

ACKNOWLEDGEMENTS

I should like to thank Dr. Leah B. Lapidus, who was my dissertation advisor. Her enthusiastic support and her extensive familiarity with this area of research were invaluable to me in carrying out the present study.

Dr. Rosalea A. Schonbar, who has contributed so significantly to my development as a clinician, was instrumental in assisting and encouraging me to understand the more sweeping implications of this research.

I feel deeply indebted to Dr. Ronald Baken for assisting with the instrumentation of this research and helping me to maintain a high level of scientific rigor.

I also wish to thank Dr. Richard Lindeman, for his guidance in the analysis of the data.

To Drs. John Gruzelier and Christopher Gilbert I am most grateful for their leading me to the present research question and for their help and support throughout the study.

I wish to express my appreciation to the Central Bergen Community Mental Health Center, where much of the research was actually conducted, for providing me with research time and facilities with which to carry out the study. I am particularly grateful to its Director, Dr. Aristide H. Esser, whose support greatly facilitated my work.

I am particularly grateful to all the subjects who gave of their time to participate in the study, with no hope of any reward other than the possibility of helping to further our knowledge in this field.

Above all, I should like to thank Nancy Davenport, whose assistance, understanding, tolerance, and love enabled me to see the study through to a successful completion.

R. L. R.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

I--INTRODUCTION

It has long been accepted that maladaptive arousal patterns are inextricably bound up with schizophrenic processes. Nevertheless, the understanding of the nature of arousal dysfunctions in schizophrenia has proved to be an elusive and contradictory undertaking.

Psychophysiological opinion has fluctuated between a view of schizophrenics being hypoaroused and one of their being hyperaroused. Bernstein (1964 & 1970) found a generalized hyporesponsivity among schizophrenics in conjunction with a significantly faster habituation rate in comparison to controls. Conversely, Dykman and his associates (1968) found hyperresponsivity and slower habituation rates among schizophrenics. Similarly paradoxical findings have been reported with respect to virtually every measure and dimension of schizophrenic arousal, and any authoritative and unbiased account of this subject makes mention of these discrepancies (e.g., Lang & Buss, 1965; Broen, 1968; Lapidus & Schmolling, 1975; et al.).

Psychodynamically oriented, clinical investigations of schizophrenia until recently have tended to ignore the issue of subtle dysfunctions in arousal in favor of explorations of manifest symptomatology. As Cromwell (1975) points out in a recent historical overview of the literature concerning schizophrenia, this emphasis is important

and understandable, recognizing the necessity for clinicians to deal with the interpersonal and social interactions of schizophrenics and their milieu. Nevertheless, he suggests that such explorations have not been sufficient to the explanation of the etiological and prognostic questions underlying the symptoms themselves.

In recent years, increased interest in the specific adaptive functions of the ego has led many clinicians and dynamically oriented theorists such as Beres (1956), Engel (1962), Gediman (1971), and Bellak, Hurvich, and Gediman (1973) to undertake an examination of the role of arousal in schizophrenia. In particular, the concept of stimulus barrier--long relegated to the laboratories of the psychophysiologists--has begun to be explored by the psychodynamically oriented in relation to its implications in schizophrenic dysfunctions.

The present study has been designed to explore the relationship between psychophysiological arousal in schizophrenia and the role of stimulus barrier, conceived of in terms of an ego function. Therefore it is necessary first to review the literature concerning each of these areas before considering their proposed interrelationship.

General Theories of Arousal in Schizophrenia

In summarizing the history of research into the role of arousal in schizophrenia, the literature is here divided into three categories: anxiety-reduction theories, one-fac-

tor arousal theories, and two-factor arousal theories. These categories follow the division of the literature proposed by Lapidus and Schmolling in their more extensive review of arousal and schizophrenia (1975).

Anxiety-Reduction Theories

In 1894, Freud first explained the break with reality, which is so much a distinguishing facet of psychosis, as a process in which "the ego has fended off the incompatible [or, 'intolerable,' in the German editions] idea through a flight into psychosis" (p. 59).

Arlo and Brenner (1964) have suggested that Freud conceived of this process differently at different stages of his life. Freud's earlier conceptualization (1894, 1911, & 1914) of the process is one of the libidinal decathexis of the external world and a regression to a narcissistic stance, followed by a pathological attempt to reestablish contact with the external world through delusions and hallucinations. Later (1926 & 1932), when Freud had developed his structural theory and its more advanced understanding of the role of anxiety, the process could be viewed as a defensive alteration of the ego--much on the model of the neurotic process. According to either conception of the psychotic process, the objective is somehow to reduce intolerably high levels of anxiety.

The growing importance of ego psychology in psychoanalytic thought has resulted in a more sophisticated extension of Freud's admittedly rudimentary framework for

understanding schizophrenia. Beres (1956) and Bellak et al. (1973) systematically subdivided ego functioning into discrete areas that could be more precisely evaluated in describing the functioning of a given individual. Nevertheless, the fundamental understanding of schizophrenia remained (as in Hartmann, 1953) a flooding of the ego with deneutralized drives which was occasioned by high-anxiety states and resulted in a regressive deterioration of defenses.

Grinker and Spiegel (1945), who studied psychotic-like reactions in combat personnel, found that under the pressure of intense anxiety certain soldiers developed "a profound regressive reaction in which there is a considerable break with reality" (p. 327). In the most severe cases, not only was reality-testing temporarily lost, but the ability to differentiate between harmless and dangerous stimuli was impaired: All stimuli evoked anxiety as though they were signals of life-threatening situations. Grinker and Spiegel concluded that the only factor that separated these psychotic-like states from actual psychoses was the extreme reality of the stress which precipitated them, and not any observable clinical difference in symptomatology.

As summarized by Lapidus and Schmolling (1975), the anxiety-reduction theories share several basic assumptions: Those predisposed to become schizophrenics exhibit deficits in ego functioning and self-esteem; the overt psychosis is a reaction to some situation which is perceived as a threat

to personal integrity; there follows a disintegration of reality-based functioning and a reorganization on a level of more primitive, autistic functioning; the effect of this regressed functioning is to reduce anxiety, and in the process to increase self-esteem and to ameliorate the threat from some reality situation.

One-Factor Arousal Theories

As early as 1934, Elizabeth Duffy suggested the importance of considering emotion and intensity together under the rubric of arousal or activation. Since that time, many investigators (Malmo, 1959; & Hebb, 1955; et al.) have adopted the position that there exists a unitary continuum of arousal, ranging from sleep to emotional excitement; and that changes along this continuum are reflected in measures such as EEG, skin resistance, heart rate, etc. This psychological theory found neurophysiological support in the work done by Moruzzi and Magoun (1949) on the functioning of the ascending reticular activating system. Duffy (1962) reviewed the literature which developed in response to her arousal hypothesis and concluded not only that it was generally supported by the findings, but that psychiatric patients differed from normals on various measures that reflected arousal.

Hebb (1949 & 1955) proposed that a reformulation of the Yerkes-Dobson law applied in the case of the relationship between arousal and performance. He postulated an inverted-U relationship in which the effectiveness of response

improves from low to medium levels of arousal, but becomes increasingly disrupted at higher levels. Schmolling and Lapidus (1972) qualify this by suggesting that task complexity must be taken into account (finding that performance is disrupted at lower levels of arousal only in the case of complex tasks).

This conception of the effects of arousal is employed by many researchers to explain various schizophrenic dysfunctions. Tomkins (1962, 1963, & 1965), in his cognitive-affective theory of personality, concludes that rapid stimulation at high levels of arousal results in the prevalence of disorganizing affects (fear, distress, and anger) which, as Lapidus and Schmolling (1975) point out, are so common in schizophrenia. West (1962), assuming that a sustained level of sensory input is necessary for efficient brain functioning, concludes that hallucinations occur at both extremes of the arousal continuum--in accordance with the inverted-U relationship. Storms and Broen (1969) suggest that disorganizing levels of arousal in combination with lowered response strength ceilings account for the prevalence of maladaptive and interfering associations in schizophrenics by causing inappropriate selections from the patient's hierarchy of possible responses.

Perhaps the most important of the researchers utilizing the one-factor arousal theory is Mednick. He proposed (1958) that the preschizophrenic is an anxiety-prone individual, whose high drive level results in heightened

stimulus generalization and remote associations. In a later longitudinal study of children with a high risk for schizophrenia, Mednick (1966) found the hyperresponsiveness and quick latencies predicted by his earlier theory. However, he found a highly significant difference in recovery time in the direction of faster recovery times for the high-risk children--the reverse of what he had predicted.

The assertion that schizophrenics become disorganized at arousal levels that are either above or below those of optimal functioning has made a valuable contribution to the understanding of schizophrenic dysfunction and of its relation to arousal. Nevertheless, the earlier mentioned conflicting findings are not simply explained by the inverted-U relationship, and a further extension of the theoretical framework is necessary before they can be accounted for.

Two-Factor Arousal Theories

By the late 1960's, it became apparent to various researchers that it was not useful to attempt to treat arousal as if it were a single, one-dimensional drive. Claridge (1967) pointed to the fact that at least two descriptive dimensions were necessary to account for the role of arousal in psychological dysfunction: arousability (which, in the present study is termed responsivity), or the degree to which the individual can be aroused by stimulation; and arousal level, referring to the prevailing

state of activation in an individual at a particular time.

Claridge (1967) hypothesized the existence of two interacting systems, one governing tonic arousal and the other an arousal-modulating system. The latter, seen as responsible for the dimension of responsivity, was measured by Claridge utilizing drug sedation thresholds (i.e., tolerance for barbiturate medications). The subject's general level of tonic arousal was measured by a test of spiral after-effect (i.e., a measure of the extent of the illusion of apparent motion induced by fixation of a previously rotated spiral). His findings led to the conclusion that in psychosis the normal equilibrium between these two systems is lost and dissociation of these systems occurs in one of two directions:

> (a) towards a state in which there is poor modulation of sensory input and poor inhibitory control over tonic arousal or (b) towards a state where increased excitability of the modulating system leads to excessive inhibition and a reduction in tonic arousal. (Claridge, 1967)

These two patterns are seen as underlying what he terms active and retarded psychoses.

As McGhie (1970) points out, such an understanding helps to clarify the apparently paradoxical findings that schizophrenics are over and under aroused. The conclusion that there are two basic groups of schizophrenics, divided precisely along these lines, obviates the problem.

The actual physiological correlates of the rather unusual measures (sedation threshold and spiral after-effect) employed by Claridge have been called into question (McGhie,

1970; Lapidus & Schmolling, 1975). Nevertheless, his fundamental assertion that a more complex interplay of arousal systems underlies psychotic dysfunction, and that this interaction results in divergent patterns of maladaptive arousal is recognized as a most significant advance.

A similar understanding is proposed by DesLauriers and Carlson (1969) in their attempt to explicate the finding that certain autistic children are extremely hypoactive while others are hyperactive. They conclude that the answer is to be found in an imbalance between the two systems of arousal which Routtenberg (1968) had proposed: Arousal System I, or the ascending reticular activating system; and Arousal System II, part of the limbic-midbrain system. DesLauriers and Carlson (1969) concluded that these two arousal systems normally function in an inhibition-disinhibition balance, and that it is the dissolution of this balance which is responsible for the two divergent activity patterns they were seeking to explain in autistic children.

In extensive research into the nature of Galvanic Skin Response (GSR) in schizophrenics, Gruzelier (1973) has demonstrated two distinct response patterns to auditory stimuli. One group, whom he termed responders, exhibited high skin conductance levels, a high incidence of spontaneous fluctuation of skin conductance level, high response amplitudes, and short latencies and recovery times. The

second group, termed nonresponders, demonstrated an absence of orienting responses, low skin conductance levels, and a low incidence of spontaneous fluctuations.

Gruzelier and Venables (1975) conclude that the absence of orienting responses cannot be attributed to peripheral or attitudinal factors, as in another phase of the same experiment the great majority of the non-responders did exhibit responses to tones when the tones were given signal value instead of being presented as neutral stimuli (Gruzelier & Venables, 1973). This is consonant with the observations of Luria and Homskaya (1970). Because responses can be evoked in nonresponders under these conditions, and because certain of the non-responders exhibit an isolated trial-one response even to neutral stimuli, Gruzelier (1975) has concluded that the terms "responder/nonresponder" are something of misnomers. In this study, therefore, these groups will henceforth be referred to as overresponders and underresponders, respectively.

Gruzelier (1973) accounts for these findings by means of a two-factor theory of arousal similar to those examined by Lapidus and Schmolling (1975). Gruzelier, however, places the locus of both systems within the limbic forebrain. He proposes that there is normally a reciprocal balance between the amygdala and hippocampus, influencing phasic, tonic, and general behavioral arousal. He concludes that in schizophrenia this balance disintegrates:

When the amygdala becomes dominant, phasic orienting response becomes accentuated and slow to habituate, tonic activity is accentuated, and there is an increase in behavioral arousal; when the hippocampus is dominant, phasic orienting activity either ceases to exist or habituates quickly, tonic levels are reduced, and there is a decrease in behavioral arousal. A great deal of evidence is presented to support the neurophysiological assumptions of this position (Douglas, 1967; Douglas & Pribram, 1966; et al.).

It is not the intent of the present research to ascertain the nature of the neurophysiology responsible for the phenomenon under consideration, or to test the various theories relating to actual brain foci and neurological systems involved. What is of immediate concern is that some such two-factor arousal system, with a normal balance of reciprocal inhibition-disinhibition, is apparently disrupted in schizophrenia, and that the resulting imbalance produces two disparate maladaptive patterns of arousal. What the two-factor arousal theories discussed above have in common is that they all predict a dichotomous division of the general schizophrenic population that is able to account for what nad previously appeared to be contradictory and paradoxical findings regarding the arousal characteristics of schizophrenics as a whole.

The Development of the Stimulus Barrier Concept

Freud's Elucidation of the Concept

The concept of stimulus barrier (Reizschutz) which first appeared in Freud's writings (1892 & 1895) was that of a protective shield necessary for the survival of the organism in a stimulus-charged environment. He elaborated upon the idea more fully in "Beyond the Pleasure Principle":

> Living substance is suspended in the middle of an external world charged with the most powerful energies; and it would be killed by the stimulation emanating from these if it were not provided with a protective shield against stimuli. (1920, p. 27)

The stimulus barrier was seen as functioning in both a receptive and protective mode. It had to allow for certain stimuli to be admitted into the organism so as to permit adaptive interaction with the external world. Nevertheless, Freud felt its more important function was the exclusion of all nonessential stimuli and the protection against potentially traumatic impingement upon the organism.

This idea of stimulus barrier is most directly connected to Freud's consideration of traumatic neuroses. He defined as traumatic any stimulus so powerful as to break through the stimulus barrier: "It seems to me that the concept of trauma necessarily implies...a breach in an otherwise efficacious barrier against stimuli" (1920, p. 29). Freud described the response to such a traumatic impingement as follows:

> Cathectic energy is summoned from all sides to provide sufficiently high cathexis of energy in the

> environs of the breach. An 'anticathexis' on a grand scale is set up, for whose benefit all the other psychic systems are impoverished, so that the remaining psychical functions are extensively paralyzed or reduced. (1920, p. 30)

Freud (1926) later returned to this idea, claiming that such traumatic rendings of the stimulus barrier were likely to have been the causes of early, primal repressions.

Although the essence of this conception was that of a passive screen designed to filter potentially damaging stimuli, certain more far reaching implications were seminally present. The role of the stimulus barrier in admitting necessary stimuli was acknowledged, even though it was minimized. Despite the fact that Freud repeatedly denied the possibility that the stimulus barrier might have any role in mediating internally produced stimuli (i.e., in dealing with instinctual drives), he noted (1926) the basic similarity in the two processes. Moreover, in his final mention of the phenomenon in "An Outline of Psychoanalysis," he hinted at the extent of the importance of the stimulus barrier as a precursor of the ego:

> Under the influence of the real external world around us, one portion of the id has undergone a special development. From what was originally a cortical layer, equipped with the organs for receiving stimuli and with arrangements for acting as a protective shield against stimuli, a special organization has arisen which henceforward acts as an intermediary between the id and the external world. To this region of our mind we have given the name of ego. (1940, p. 145)

Later Extrapolations from Freud's Conception

Perhaps the first extension of the stimulus barrier

concept concerned its implications for the regulation of internal as well as external excitation. Holt (1948) and Hartmann (1950) were quick to come to the position--logically suggested, albeit nominally denied, by Freud himself--that the stimulus barrier served as a protection against forces from within the psyche as well as from without. More recently, Winnicott (1958) and Benjamin (1965) have concurred with this position.

Fenichel, in his compendium of Freudian psychodynamics (1945), further elaborated on the role of stimulus barrier in traumatic neuroses:

> The excitation already at hand has to be mastered before new stimuli can be accepted. The organism develops different ways of protecting itself against too great a quantity of stimulation (Reizschutz). Refusing to accept new stimulation is a primitive means of re-establishing such protection after it has been broken down by trauma. (p. 118)

More importantly, he focused on a second basic longing in human existence. Freud had concentrated on the tendency of the psychic apparatus to reduce stimulation and tension to the lowest obtainable level (the Nirvana principle). Fenichel, noting the alternation in infants between states of waking hunger and sleep, added to this longing for tension reduction the concept of longing for objects.

The drive towards objects, so important in current object relations theory, also plays a pivotal role in later thinking about the stimulus barrier phenomenon. The concept of stimulus hunger, first enunciated by Buerger-Prinz and Kaila (1930), has become widely accepted

by theoreticians of various persuasions (Wolff, 1960; Engel, 1962; Rappaport, 1967; et al.) as an approach to explaining the interaction between the seeking out of sensory stimulation on the one hand and the filtering of it on the other. As put forth by Bellak (1963) and Wallerstein (1967), adaptive stimulus barrier functioning must provide the organism with an optimal amount of stimulation--not merely protect it against a destructive excess of stimulation.

It has been mentioned that the concept of stimulus barrier received the greatest attention in Freud's work in connection with the issue of traumatic neuroses. The conception of a traumatic breach in an individual's stimulus barrier has also been greatly expanded in recent years. Kris (1956) and Khan (1963) have espoused what has become a widely accepted notion that cumulative, ongoing strain on the individual is at least as significant a problem for an individual's stimulus barrier to cope with as is sudden, piercing trauma.

Thus, the concept of stimulus barrier advanced from its rather limited position in Freud's conception to the point where it was considered to be a quite complex mechanism functioning with regard to both internal and external stimulation, reacting differentially in both a receptive and a protective fashion in such a way as to achieve an optimum level of stimulation, and carrying on its functioning in an ongoing way as opposed to coming into

play only in intensely threatening situations.

Stimulus Barrier as an Active Ego Function

It has already been noted that Freud, in his later considerations (1940) of the stimulus barrier phenomenon, recognized the relationship of the role played by this mechanism to the developing structure of the ego. Moreover, it is of great significance that Freud continually attributed the cause of the development of the ego to the need to deal with the interface between the organism and the external world--an interface that takes place at the very location of the stimulus barrier.

Nevertheless, in the view of modern ego psychology, demonstrating the relationship between this phenomenon and the development of the ego does not suffice to justify the inclusion of stimulus barrier among the functions of the ego. Such an inclusion would imply that the phenomenon provided some form of mastery, and, as Helen Gediman (1971) pointed out in her exhaustive review of the literature concerning stimulus barrier as an ego function, "Any consideration of mastery must imply active, if not 'volitional,' efforts on the part of the ego" (p. 251).

Waelder (1967) attributed just such an adaptive function to stimulus barrier in describing it as an active regulator of the organism's dealing with the onslaught of traumatic stimuli in either of two diverse ways (alloplastic and autoplastic responses).

Hartmann (1950), too, insists on the active nature of

the stimulus barrier and further suggests that the phenomenon represents an early area of autonomous functioning of the preliminary stages of the ego upon which later defensive functioning is modelled:

> Freud has often pointed to the analogy between defense actions against drives and the means by which the ego avoids danger from without.... Here I want to emphasize that it is indeed tempting to consider very early processes in the autonomous area as forestages of later defense against both inner and outer dangers.... I want to point to Freud's statements concerning what he calls protective barrier against stimuli, in its possible relation to ego development....those tendencies do not originate in the id but in the autonomous preliminary stages of ego formation. It might well be that the ways in which infants deal with stimuli ...are later used by the ego in an active way. (p. 125)

That the stimulus barrier functions in both a passive, threshold mode and an active, integrating mode was the conclusion of Brody and Axelrad (1966). Benjamin (1965), in addition to positing both modes of functioning, claimed to have found neurophysiological evidence for the emergence of the active, integrating aspects of stimulus barrier in EEG pattern changes in infants between the ages of one and two months.

The concept of stimulus barrier as an ego function achieved its most highly developed formulation in the work of Gediman (1971):

> Stimulus barrier may be reformulated as a complex ego function measurable along a dimension of adaptiveness-maladaptiveness. It refers to those structures and functions which enable a person to regulate amounts of inner and outer stimulation so as to maintain optimal homeostasis and adaptation. The 'receptive' and 'protective' functions referred to by Freud...include both sensory thresholds and

> also the organization of sensory experience. (p. 254)

This conception of stimulus barrier is incorporated into the work of Bellak et al. (1973), who went on to develop a means for the quantification of the adaptiveness of an individual's functioning in this area.

Stimulus Barrier Dysfunction

Early in his writings, Freud (1895) recognized that in infancy the role of the mother was related to the functioning of the stimulus barrier. This theme became particularly important for the object relations theorists (Winnicott, 1958 & 1965; Guntrip, 1961 & 1968; et al.) who viewed inadequate mothering as a failure to protect the infant against damaging impingements from without or to provide an appropriate level of positive stimulation--which is to say, a failure to serve a stimulus barrier function to augment the infant's own developing functioning. Failures at this stage are seen as leading to less adaptive stimulus barrier functioning in the individual's later life.

Constitutional factors may also play a crucial role in determining the adaptiveness of an individual's stimulus barrier functioning, as has been suggested by Anna Freud (1967). Such assertions are consonant with the older, threshold conceptions of stimulus barrier.

Two major facets of stimulus barrier functioning have been delineated--those of sensory threshold and of

integration of sensory experience--and it is logical to assume that two types of dysfunction are possible. As Gediman (1971) has noted:

> Apparently all people are endowed congenitally with threshold potentials for stimuli in all sensory modalities, and thus bring to bear in their total response repertoire something called the 'state' of the organism. But the ego, in its totality of developmental vicissitudes and multiple functions, is responsible for the eventuation of congenitally determined thresholds in each person's unique mode of responding to stimuli by organizing and integrating his sensory experience. So there are ego response measures other than absolute or differential thresholds for stimuli, which determine the status of stimulus barrier as an ego function. (p. 250)

Thus it has been observed by Engel (1962) that certain individuals with constitutionally low thresholds exhibit a "heightening" of stimulus barrier, leading to a defensive withdrawal and a reduction of incoming stimulation. On the other hand, as Goldfarb (1961) has pointed out in regard to schizophrenic children, hypersensitivity may at times be linked to failures in the ability to integrate experience rather than to lowered thresholds.

Arousal and Stimulus Barrier Dysfunctions in Schizophrenia

As has previously been discussed, maladaptive patterns of arousal have long been implicated in schizophrenia. The clinical research conducted by Bellak et al. (1973) found significant stimulus barrier dysfunction was also present in schizophrenics. As both of these approaches

appeared to be exploring related aspects of a schizophrenic's functioning in relation to stimulation, albeit from quite different vantage points, it appeared potentially profitable to examine their possible interrelationship.

The most productive of recent thinking concerning the role of arousal in schizophrenia has been shown to be based on two-factor theories (Claridge, 1967; Gruzelier, 1973; et al.). These theories propose that two normally balanced systems, which act in a reciprocal inhibition-disinhibition manner in the arousal patterns of healthy individuals, become dissociated in schizophrenia. The resulting imbalance, depending upon its dominant direction, produces either of two schizophrenic subtypes: the responders or nonresponders of Gruzelier; the active or retarded psychotics of Claridge.

The two facets of stimulus barrier functioning that have been discussed--what Gediman (1971) referred to as the threshold components, or "state" of the organism on the one hand, and the organizing and integrating, or active response elements on the other--suggest a possible parallel to the tonic and phasic components of the two-factor arousal theories. Nevertheless, just as the present study did not attempt to explore further the actual psychophysiological underpinnings of these arousal theories, it cannot claim to lead to any definite elucidation of the possible relationships between the components of stimulus

barrier and the specific arousal systems. It did, however, predict that there would be two distinct patterns of schizophrenic stimulus barrier functioning reflective of the two different schizophrenic arousal patterns.

Andrew (1975), in an extensive review of the literature dealing with arousal, has underscored the dangers inherent in defining arousal too broadly, and has insisted upon the importance of choosing measures that focus on specific aspects of arousal. The aspects of arousal that appeared to be most germane to the present study were orienting response activity and a baseline measure of activation.

The orienting response was first described by Pavlov (1941), and was extensively studied by the more recent Russian psychophysiologist, E.N. Sokolov. This phenomenon, which takes the form of a momentary increase in arousal level (as reflected in various physiological systems) in reaction to a novel stimulus, was described by Sokolov (1960) as representing a most basic interaction of the organism with its environment, indicative of the admission into the awareness of the organism of some input concerning its environment.

It was decided that an examination of arousal patterns that combined both orienting response characteristics and baseline arousal levels--measured, following the methodology of Gruzelier (1973) and Gruzelier and Venables (1972, 1973, 1974, & 1975), in terms of electrodermal activity--would

provide the optimal opportunity for further elucidation of the schizophrenic's most basic levels of responsivity. It was felt that such an examination would provide both direct information about arousal patterns and comparative data concerning stimulus barrier.

II--PURPOSES AND HYPOTHESES OF THE PRESENT STUDY

At the heart of the present study are two rather different approaches which are both designed to measure an individual's reactivity to his environment. One involves a psychophysiological experiment designed to obtain information about the autonomic patterns of the participant's orienting responses to simple stimuli as reflected in the GSR pattern he exhibits. The other consists of a structured psychological interview designed to assess the adaptive and coping characteristics, as well as the level of sensory thresholds, that make up an individual's stimulus barrier--when stimulus barrier is viewed as an adaptive ego function.

It is proposed that these two approaches are related, and that they are both similarly efficacious in the examination of the maladaptive arousal patterns found in schizophrenic pathology. The broader understanding of stimulus barrier (as proposed by Bellak et al., 1973) and the recently identified psychophysiological patterns of arousal in schizophrenia would appear to provide a midground between two rather different--and seemingly disparate--approaches to schizophrenia research. The new conception of stimulus barrier moves towards areas of active ego response and integration, and thence into more psychodynamic realms; while the intent of Gruzelier (1973) is to identify specific

brain foci implicated in schizophrenic dysfunction, and thereby to concentrate on the neuropsychological aspects of these conditions. Nevertheless, stimulus barrier is an idea just recently expanded from a purely sensory threshold concept into a dynamic one, and it still has its roots deep in psychophysiology; while, on the other hand, researchers and clinicians alike are beginning to recognize that GSR and the arousal systems that underlie it are related to and reflect long-term behavioral patterns (Edelberg, 1972), and furthermore, that these autonomic responses are open to direct therapeutic intervention (as in the growing fields of biofeedback and relaxation training).

The present study was also designed to explore further the nature of the maladaptive arousal patterns in schizophrenic conditions. The issue of whether the overresponder/underresponder differentiation is stable over time for individual schizophrenics was systematically examined. Gruzelier (1973), in addition to suggesting that the overresponder condition might degenerate into an underresponder mode over the life course of a schizophrenic, mentioned that there were at least some instances of change in both directions, at times apparently relating to clinical state or environmental pressures. His research made no provision to examine such fluctuations systematically, however. The present research was designed to ascertain whether schizophrenics remain in a single pattern or move back and forth between the two patterns characteristic of schizophrenics in

general.

One of the two experimental groups in the present study was composed of schizophrenics being treated on an outpatient basis, all of whom had little or no history of psychiatric hospitalization. These criteria were chosen in contradistinction to those of the noninstitutionalized samples in the studies of Gruzelier and Venables (1972, 1973, & 1974). In these earlier studies, chronically institutionalized patients were compared to those with a shorter history of hospitalization (less than five years), but who nevertheless were inpatients at the time of testing. The decision to examine outpatients in the present study was made in order to explore the arousal patterns in a schizophrenic group not as yet studied by any of the current researchers. It was further hoped that in comparing such a sample to a long-term institutionalized sample (such as the inpatient sample in the current study) the differences attributable to institutionalization itself would be more easily identifiable.

The hypotheses of the present study are as follows:

1. Schizophrenics exhibit either an overresponsive or underresponsive pattern of GSR when compared to a normal control group.

2. The GSR patterns of schizophrenics are differentially related to ego strength ratings of their stimulus barrier functioning.

3. In comparison to the stability of the normal

habituation pattern, the overresponder/underresponder differentiation changes more over time.

In addition to the hypotheses, an exploration of the relationships between the overresponder/underresponder differentiation and several other variables was undertaken. It had been suggested (Gruzelier, 1973) that while this differentiation was not related to traditional subdiagnostic categories of schizophrenia, it did correlate with certain facets of overall clinical picture. Gruzelier found that his noninstitutionalized overresponders were rated high on clinical scales (Wittenbourn, 1968) of manic, anxious, and belligerent behavior, and that coincident with, but not confined to, this pattern is a clinical picture of heightened schizophrenic excitement and active symptomatology. Noninstitutionalized underresponders received low ratings of behavioral arousal, and this pattern was coincident with a clinical picture of dulled affect, loss of interest, and reduced behavioral activity (and, in chronically institutionalized patients, with passive, malleable, or deteriorated behavior). Although Gruzelier's findings in these areas were less than conclusive, the conclusions reached by Lapidus and Schmolling (1975) would also predict generally similar correlations. Thus the present study examined the overresponder/underresponder differentiation against the background of the participants' anxiety, mood, and behavior--both subjectively reported and clinically observed.

III--METHOD

Subjects

There were two experimental groups, each consisting of 20 schizophrenic patients. The outpatient group was composed of subjects drawn from the outpatient departments of a community mental health center in New Jersey. These patients had no more than one year of total accumulated psychiatric hospitalization (with a mean length of hospitalization of 4.1 months); they had never been hospitalized consecutively for more than six months; and they had not been hospitalized within the three months prior to testing. The inpatient group was drawn from the chronic wards of a New Jersey State Psychiatric Hospital. These patients were currently institutionalized, with their present hospitalization beginning at least four years prior to the testing. They had an average of 142.6 months of accumulated psychiatric hospitalization. (See Table 1.)

All of the subjects were adult males. Only patients unambiguously diagnosed as schizophrenic were included in the experimental samples. Patients with schizo-affective disorders were not included in the samples. Where a patient's records suggested that there was a question of organic brain impairment, chronic alcoholism, or drug addiction, he was excluded from the study. None of the

Table 1

Mean Age, Number of Hospitalizations, and Length of Accumulated Hospitalization by Sample

Group[a]	Age		Number of Psychiatric Hospitalizations		Length of Accumulated Psychiatric Hospitalization (in months)	
	M	SD	M	SD	M	SD
Outpatient	31.8	8.92	1.0	.89	4.1	4.34
Inpatient	34.2	9.19	3.5	1.57	142.6	1.44
Control	32.4	9.04	0	0	0	0

[a] $n = 20$ for each group.

schizophrenics had been lobotomized or had received electroconvulsive therapy or insulin coma treatments. Subdiagnoses were obtained from the patients' charts. The representation of subdiagnoses in the two experimental groups is shown in Table 2.

The control group consisted of 20 normals with no psychiatric history. They were drawn from the professional and nonprofessional male employees of two New York religious institutions, and included clergymen, teachers, administrators, guards, janitors, telephone operators, etc. They were selected after the experimental groups had been formed, and an attempt was made to match them to the experimental groups with respect to age. It was not possible to match the control and experimental groups for education, marital status, and occupation, as schizophrenics are characteristically different from normals for these variables. Moreover, there is no evidence that such variables affect the psychophysiological measures used in the present study.

Materials and Procedures

Participants were all tested by the experimenter in similar settings: a quiet room with a comfortable arm chair for the subject and two chairs for the experimenter (one chair facing the subject and offset slightly to his right, and another, along with the GSR equipment, positioned directly behind the subject and used only during the GSR segment of the session). Room temperature was maintained at

Table 2

Frequency of Schizophrenic Subdiagnoses in the Outpatient and Inpatient Groups

Diagnosis	Outpatient	Inpatient
Hebephrenic	1	2
Catatonic	0	1
Paranoid	8	7
Acute	2	0
Residual	1	0
Chronic Undifferentiated	8	10

70° F ±2 .

Each subject was tested on two separate occasions, six weeks apart. The procedures were identical for both sessions. The time required for each session ranged between 40 and 70 minutes.

At the initial session a brief description and explanation of the procedures was given to the participant. This verbal material was virtually a verbatim repetition of the information included in the consent form (see Appendix A), which the participant was then asked to sign.

Participants were then administered the following procedures in order:

1. The subject was asked to fill out the Beck Depression Inventory, Short Form (Beck, Ward, Mendelson, Mack, & Erbaugh, 1961). The form is a one page questionnaire, in which the participant is asked to pick the one of four statements in each of the 13 categories that best describes his feelings at that particular moment in time. The maximum score for the entire scale is 39. The test yields a single numerical score that is reflective of an individual's subjectively perceived level of depression. The following are the accepted estimates for various levels of depression: 0 - 4, none or minimal; 4 - 7, mild; 8 - 15, moderate; 16+, severe.

2. Electrodes were attached to the volar surface of the distal phalanges of the first and second fingers of the right hand. The area was first cleaned with isopropyl

alcohol and then dried. An adhesive insulating tape ring with a hole 1 cm in diameter was then applied to the area to expose only a standardized area for electrode contact. Silver/silver chloride electrodes, 1 cm in diameter, were used together with Beckman electrode paste. The electrodes were held in position by means of plastic adhesive tape.

3. The stimulus barrier assessment interview was then administered. This interview is section nine of the 12 part interview for the clinical assessment of ego functions developed by Bellak et al. (1973). The stimulus barrier assessment is composed of 14 areas of inquiry into the subject's sensory thresholds and adaptive and coping mechanisms for dealing with stimulation. In each area specific questions are provided, and the type and extent of probing or prompting for further information are strictly delineated.

The interview is scored by rating the information obtained about the subject's stimulus barrier functioning against a scale of seven defined modal stops, numbered 1 to 7. When a subject's level falls between two of the defined stops on the scale, a .5 intermediate rating is introduced. The final score is computed by multiplying the scale number by 2 and subtracting 1--thereby producing a rating between 1 and 13, without any fractional steps.

The Bellak interview has been extensively validated, and it has been demonstrated that interviewer effects and interobserver variability are minimal. (Interrater reliability has been calculated by product-moment correlation as

.61 for a mixed sample; it is considerably higher when neurotic subjects are not included--reaching levels between .65 and .81.) To avoid introducing any rater bias into the evaluation of the stimulus barrier interview, the evaluation took place apart from the scoring of the other information about an individual participant. The stimulus barrier interviews were scored no fewer than ten at one time, and the identity of the subject was not known to the rater at the time of scoring.

The ego function scale can be used to describe a subject's highest, lowest, characteristic, and current levels of functioning. For the purposes of this study, only current functioning was measured.

4. Headphones were placed over the subject's ears and attached to an audio signal generator. The headphones were Superex model ST-PRO-B. The audio signal generator was designed and built by the Speech Research Laboratory of Teachers College, Columbia University.

5. Skin conductance was measured by a Lafayette Instrument Company GSR Amplifier (Model 7601 TP), operating in a DC mode with a 24 μA subject circuit. GSR characteristics were recorded directly on a Lafayette Instrument Company Polygraph (Model 7603-1A SP).

As suggested by Venables and Martin (1967) and Lykken and Venables (1971), GSR characteristics were expressed in terms of conductance. Following the methodology of Gruzelier (1973), skin conductance characteristics were defined

as follows:

a. Orienting response: an increase in conductance greater than .05 μmhos between 1 and 5 seconds after the orienting stimulus.

b. Amplitude: the highest point reached within 4 seconds of response onset, minus the level of skin conductance at response onset.

c. Latency: the time elapsed between stimulus onset and the onset of the response.

d. Recovery time: the time elapsed from peak amplitude and the amplitude halfway to return to the level of conductance at response onset.

e. Spontaneous fluctuations: responses with an amplitude higher than .05 μmhos and occurring during the tone sequence, but not between 1 and 10 seconds after each orienting stimulus.

f. Skin conductance level: the average level measured at five intervals during the tone sequence (at 0, 2, 4, 7, and 9 minutes into the sequence).

The subject was instructed to find a comfortable position, to move about as little as possible, and to remain silent during the tone sequence. He was told that there was nothing he had to do except relax and listen to the tones.

At this point the experimenter moved to the GSR equipment located out of sight behind the subject. A period ranging from 2 to 5 minutes was allowed for the subject's skin conductance level to stabilize and for the amplifier to

be adjusted.

The habituation sequence was then begun. It consisted of 15 1000 Hz, 1 second tones at an intensity of 75 dB SPL (re: ANSI, 1969). The tones were presented binaurally at preset intervals ranging between 24 and 60 seconds. The pattern and length of the intervals in the sequence were identical for each subject. The pattern was established prior to the beginning of the study using computer produced random numbers (within a predetermined range of 20 to 60 seconds). The tones were therefore not predictable from the point of view of the subject. A schedule for the timing of the tones in the sequence is found in Appendix B.

6. The subject's hearing was screened using the audio signal generator. A single tone (1000 Hz, 1 second) at an intensity of 18 dB SPL (re: ANSI, 1969) was presented monaurally to each of the subject's ears--first right and then left. The subject was asked to indicate when he heard a tone. Any subject unable to detect the tone in both ears was excluded from the study.

The experimenter returned to his original position facing the subject. The electrodes were then removed and the subject was provided with a paper towel with which to remove any remaining electrode paste from his fingers.

7. The Structured Clinical Interview (Burdock & Hardesty, 1969) was administered to each participant. The Structured Clinical Interview (SCI) is a psychological research technique for the assessment of levels of psycho-

pathology. It was used in this study to provide an objectively obtained measure of the various areas and degrees of psychopathology that might affect the major differentiations under examination.

The Structured Clinical Interview includes an interview protocol which sets forth a series of specific questions to be asked of the interviewee in a fixed order. In addition to the major questions, specific secondary probes are delineated for use when the original question fails to produce an appropriate response. No spontaneous questioning or prompting is permitted at any time. The Structured Clinical Interview also contains an inventory of 179 discrete items representing possible verbalizations, attitudes, or actions that might be evoked by the uniform stimulus of the protocol. The experimenter makes a yes/no judgement concerning each of these items during the course of the interview. Any positively rated item is included in the raw score of one of the ten pathology scales of the Structured Clinical Interview. The ten raw scores are then transformed into standardized scores ranging from -1.00 to 7.00, with zero being the mean score for normals on each subtest.

The 10 areas of psychopathology assessed by the Structured Clinical Interview are the following:

a. Anger-Hostility: belligerence, irritability, jealousy, hatred of associates, violent actions or threats, repeated legal difficulties, etc. (reflected either in verbalizations or behaviors).

b. Conceptual Dysfunction: disturbances of concept formation or retention, or disturbances of orientation, memory, attention, and concentration.

c. Fear-Worry: reported or displayed apprehensiveness, nervousness, or anxiousness.

d. Incongruous Behavior: seemingly contradictory modes of expression and unusual activities (e.g., incongruous emotional response, rituals, smells of urine or feces, fleeting expressions, bizarre actions or sounds).

e. Incongruous Ideation: contradictory emotions, inappropriate or flat affect, and delusions.

f. Lethargy-Dejection: lack of energy, loss of interest or enjoyment, pessimism, motor retardation, etc. (reflected physically as well as emotionally).

g. Perceptual Dysfunction: presence of hallucinations.

h. Physical Complaints: reported somatic difficulties.

i. Self Depreciation: feelings of guilt, inferiority, or worthlessness.

j. Sexual Problems: difficulties whose genesis is in the subject's sexual attitudes or behaviors.

The Structured Clinical Interview has been shown to have a high interrater reliability with mimimal interviewer effects. (Interrater reliability has been repeatedly calculated as being between .77 and .92.) It is of particular

value in the present research, as the Structured Clinical Interview has been shown to be highly sensitive to short-term fluctuations in clinical state.

8. Finally, the Taylor Scale of Manifest Anxiety (1953) was administered. The Taylor is a one page questionnaire in which the subject is asked to respond either true or false to a series of 28 items. All responses in the keyed direction are scored as 1, and their total represents the score for the test. The Taylor score provides a relative measure of a subject's level of anxiety--either directly experienced or somatically expressed.

9. The following information was obtained from the patient's clinic or hospital records. In the case of the control group it was obtained from the subject's direct reports:

a. Age.

b. Education.

c. Marital status.

d. Occupation.

e. Diagnosis. Controls were arbitrarily assigned a designation of zero, indicating no diagnosed psychiatric condition. The subdiagnoses of the schizophrenics in the study used the categories set forth in the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association (DSM-II), and were nominally scaled by the decimal fraction indicative of that

subtype in the DSM-II.

f. Length of psychiatric hospitalizations, in months (the total accumulated time as an inpatient).

g. Number of psychiatric hospitalizations.

h. Medication. The types of dosages of all psychotropic medications received by patients in the study were recorded. All patients included in the sample had no change in medication between the first and second testing sessions. Patients receiving psychotropic medications other than antipsychotic medication, tricyclic antidepressant medication, or anti-Parkinsonian medication (e.g., those taking lithium carbonate, antianxiety agents, or MAO-inhibitors) were excluded from this research. A scale of milligram-equivalent dosages for the antipsychotic medications taken by subjects in the sample was derived from the figures of Shader and Jackson (1975). A similar scale for the milligram-equivalent dosages of the anti-Parkinsonian medications was derived from a publication by Prien (1973). These scales are presented in Appendix C. Recent studies (Schildkraut & Klein, 1975) have indicated that there is such a marked variation among individuals in the rates of metabolism of antidepressant drugs, that there is no meaningful way of establishing equivalent dosages across

patients--even for the same drug. Nevertheless, the two antidepressants taken by patients in the current study, imipramine and doxepin, are seen as being equivalent with regard to their general potency (Prien, 1973; Schildkraut & Klein, 1975) and thus were both assigned a milligram-equivalent dosage of 100 for the purposes of this research. The total milligram-equivalent daily dosage for each of the three categories was computed for each subject.

IV--RESULTS

The Data and Their Analysis

The statistical operations for this study were performed at the Computer Center at Teachers College, Columbia University. With the exception of the analyses of variance, which utilized the MANOVA program (Appelbaum, 1974), and the Kruskal-Wallis nonparametric analyses of variance, which utilized the IBM NAK-1 package, the SPSS programs were used. All the p-values given represent two-tailed probabilities except where one-tailed p-values are specifically indicated.

The following are the variables used in the study, along with their range of scores or units of measurement.

GSR Variables

1. Skin conductance level, in μmhos.
2. Response amplitude, in μmhos.
3. Response frequency.
4. Response latency, in seconds.
5. Recovery time, in seconds.
6. Number of spontaneous fluctuations of skin conductance.
7. Response pattern (0 = underresponsivity, 1 = overresponsivity, 9 = normal habituation).

Psychological Measures

8. Beck Depression Inventory (0 to 39).
9. Taylor Scale of Manifest Anxiety (0 to 28).
10. Bellak Stimulus Barrier Rating (1 to 13).
11. The Structured Clinical Interview (SCI) Anger-Hostility scaled score (-1.00 to 7.00).
12. SCI Conceptual Dysfunction scaled score (-1.00 to 7.00).
13. SCI Fear-Worry scaled score (-1.00 to 7.00).
14. SCI Incongruous Behavior scaled score (-1.00 to 7.00).
15. SCI Incongruous Ideation scaled score (-1.00 to 7.00).
16. SCI Lethargy-Dejection scaled score (-1.00 to 7.00).
17. SCI Perceptual Dysfunction scaled score (-1.00 to 7.00).
18. SCI Physical Complaints scaled score (-1.00 to 7.00).
19. SCI Self Depreciation scaled score (-1.00 to 7.00).
20. SCI Sexual Problems scaled score (-1.00 to 7.00).

Demographic Information

21. Age, in years.
22. Education (0 = grade school, 1 = partial junior high school, 2 = complete junior high school, 3 = partial high school, 4 = complete high school, 5 = partial vocational or technical school, 6 =

complete vocational or technical school, 7 = partial college, 8 = complete college, 9 = graduate education).

23. Marital status (0 = never married, 1 = married, 2 = separated, 3 = divorced).
24. Occupation (0 = never employed, 1 = unskilled, 2 = skilled, 3 = sales, 4 = management or technical, 5 = professional).
25. Diagnosis (0 = no psychiatric condition, 1 = hebephrenic, 2 = catatonic, 3 = paranoid, 4 = acute, 6 = residual, 9 = chronic undifferentiated; the missing numbers represent subdiagnoses of schizophrenia not included in the samples for this study).
26. Length of accumulated psychiatric hospitalization, in months.
27. Number of psychiatric hospitalizations.
28. Antipsychotic medication, mg-equivalent dose per day (see Appendix C).
29. Antidepressant medication, mgs per day.
30. Anti-Parkinsonian medication, mg-equivalent dose per day (see Appendix C).

Tests of the Hypotheses

Hypothesis 1. The Overresponder/Underresponder GSR Patterns of Schizophrenics

A normal subject, in reaction to a novel stimulus that is repeatedly presented, will exhibit a series of orienting

reactions, followed by a process of habituation through which the response to the particular stimulus will be extinguished. It was predicted that schizophrenics would not exhibit this normal pattern of orienting responses and subsequent habituation, but rather would exhibit either of two maladaptive GSR response patterns. One of these maladaptive patterns was termed the overresponder mode, and was operationally defined as a pattern in which orienting responses were initially exhibited, but in which habituation to criterion (viz., the absence of orienting response to three consecutive stimuli) failed to occur within the 15 trials of the tone sequence. The second maladaptive response pattern was termed the underresponder mode, and was operationally defined as a pattern in which either no orienting response was exhibited or only an isolated first trial response occurred without any further orienting activity.

Response frequency. Of the forty schizophrenics in the study, approximately half (18 during the first session and 19 during the second) manifested the overresponder pattern. As indicated in Table 3, the mean response frequencies for this group were 13.64 and 14.14 for the outpatient and inpatient groups respectively for the first session, and 14.36 and 13.50 for the second.

The other group (22 during the first session and 21 during the second) were underresponders. Of the outpatients, approximately 75% of the underresponders exhibited an iso-

Table 3

Number of Subjects and Means and Standard Deviations for GSR Frequency, Skin Conductance Level, and Spontaneous Fluctuations, by Session

Group	n	Frequency		Skin Conductance Level[a]		Spontaneous Fluctuations	
		M	SD	M	SD	M	SD
First session							
Outpatient							
Overresponders	11	13.64	1.36	12.66	3.99	23.36	9.53
Underresponders	9	.78	.44	5.03	1.50	1.56	1.24
Inpatient							
Overresponders	7	14.14	1.22	12.30	4.05	20.86	5.15
Underresponders	13	.46	.52	4.16	2.27	1.62	1.80
Control	20	3.80	1.06	7.75	2.03	5.45	1.60
Second session							
Outpatient							
Overresponders	11	14.36	1.03	13.66	3.66	28.45	12.07
Underresponders	9	.67	.50	5.15	1.90	2.22	1.64
Inpatient							
Overresponders	8	13.50	1.31	12.50	3.52	19.12	6.66
Underresponders	12	.33	.49	3.56	1.52	1.67	1.75
Control	20	4.15	1.50	7.64	1.87	5.50	1.64

[a]Skin Conductance Level expressed in μmhos

lated trial-one response, while the remainder exhibited no orienting response at all. Less than half of the inpatient underresponders exhibited even an isolated trial-one response.

As indicated in Figures 1 and 2, there was a sharp contrast between the habituation pattern of the controls (all of whom initially exhibited 3-8 orienting responses followed by habituation to criterion) and the patterns of the schizophrenics--all of whom exhibited either an overresponder pattern (with a range of response frequencies of 11 to 15) or an underresponder pattern (with response frequencies of either 0 or 1). The comparison of the response frequencies of the overresponder, underresponder, and control groups, using the Kruskal-Wallis nonparametric analysis of variance, indicated differences significant at the $p < .001$ level for all samples (see Table 4).

These findings clearly support the hypothesized existence of two schizophrenic GSR patterns different both from each other and from the normal.

Skin conductance level. Group means and standard deviations are presented in Table 3, and the comparisons by the Kruskal-Wallis Test of each schizophrenic group and the control group are presented in Table 4. In every case the schizophrenic groups differed from the control group in the direction of overresponders having higher skin conductance levels and the underresponders having lower levels. All of the differences were significant at the $p < .001$ level.

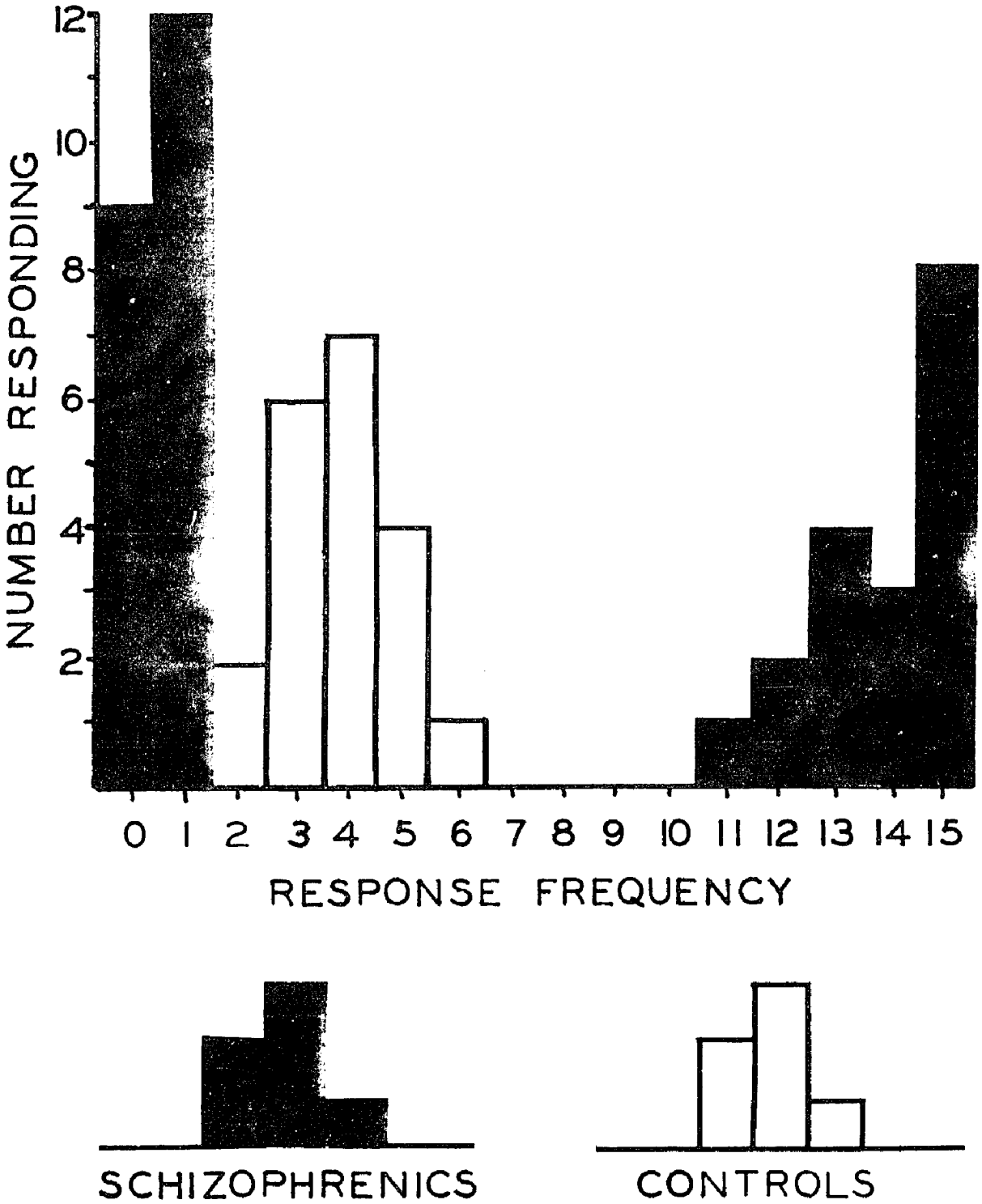

Figure 1. Distribution of response frequencies for schizophrenics and controls. First session.

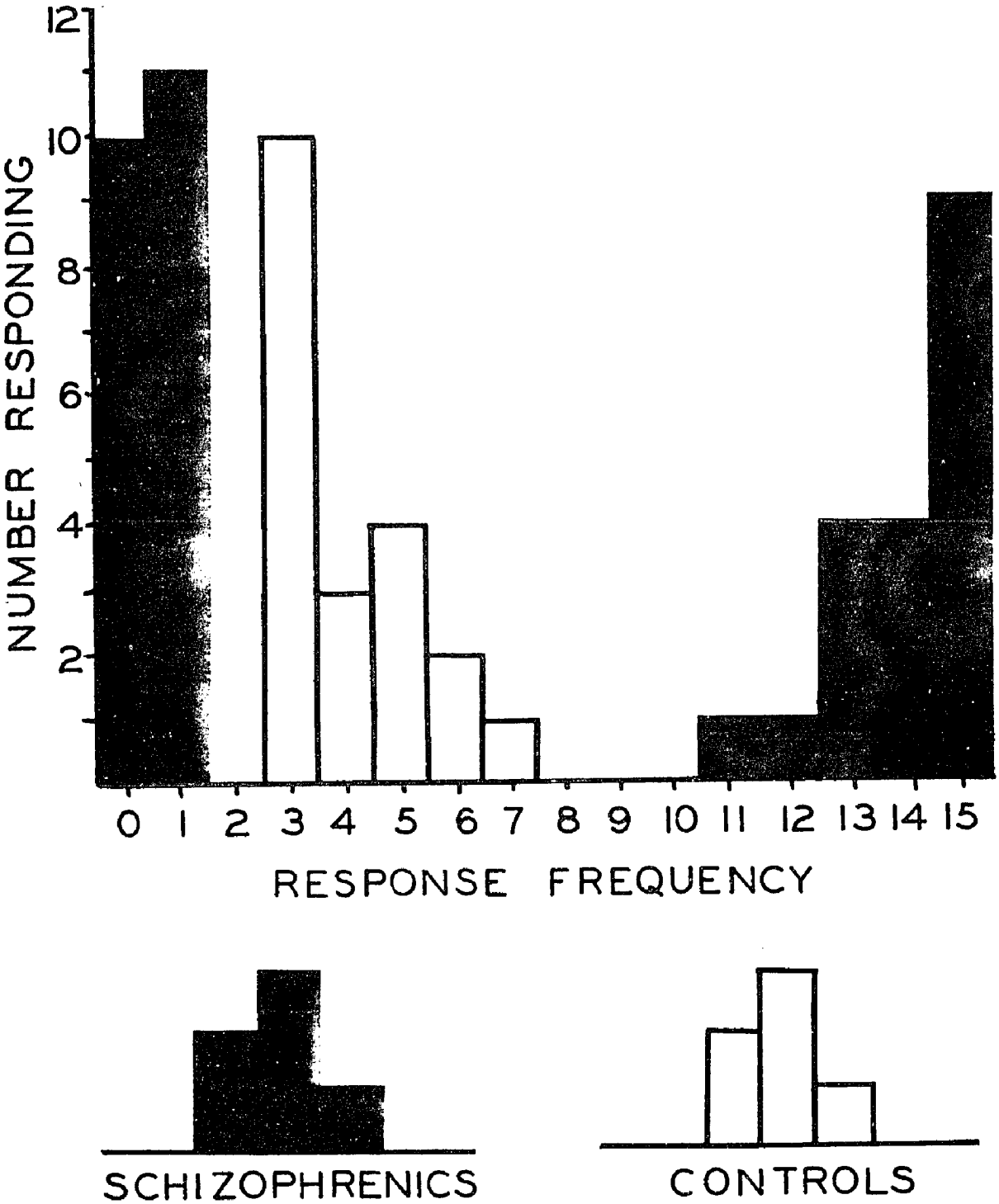

Figure 2. Distribution of response frequencies for schizophrenics and controls. Second session.

Table 4

Kruskal-Wallis Analysis of Variance for the Effect of Response Pattern on Response Frequency, Skin Conductance Level, and Number of Spontaneous Fluctuations

Sample[a]	H-Values for Each Analysis		
	Frequency	Skin Conductance Level	Spontaneous Fluctuations
First session			
Outpatient	32.89*	21.18*	31.96*
Inpatient	32.60*	20.47*	28.87*
Second session			
Outpatient	32.89*	24.28*	30.33*
Inpatient	32.78*	26.03*	30.43*

Note. The df=2 in each case.

[a]For each sample the control group was compared with the overresponder and underresponder groups comprising that experimental sample.

*p<.001

Spontaneous fluctuations. Group means and standard deviations are given in Table 3. The overresponder groups exhibited a higher incidence of spontaneous fluctuations than the control group, while the underresponder groups exhibited a lower incidence. All of the differences were significant at the level $p < .001$ (see Table 4).

Response amplitude. Response characteristics (viz., amplitude, latency, and recovery time) were compared only for the overresponder and control groups, because the underresponders either did not exhibit any response at all, or they exhibited only an isolated initial response.

Group means and standard deviations for response amplitudes are presented in Table 5. Comparisons were done using the Kruskal-Wallis tests with $df = 1$. For the second session, as predicted, the outpatient overresponders had a significantly higher response amplitude than did the controls. $H = 6.55$, $p < .01$. The corresponding comparison for the first session, while failing to reach a significant level of difference, approached significance in the predicted direction, $H = 2.47$, $p < .1$. The inpatients were not significantly different from the controls: first session, $H = .06$, ns; second session, $H = .00$, ns.

Response latency. Group means and standard deviations are presented in Table 5. Using the Kruskal-Wallis test ($df = 1$), no significant differences were found between the latency times of overresponders and controls: outpatients, first session - $H = 1.52$, ns; outpatients, second session -

Table 5

Group Means and Standard Deviations for GSR Amplitude, Latency, and Recovery Time of Outpatient and Inpatient Overresponders and Controls by Sessions

Group	Amplitude (μmhos)		Latency (seconds)		Recovery Time (seconds)	
	M	SD	M	SD	M	SD
First session						
Outpatient	.92	.72	1.73	.44	5.15	2.12
Inpatient	.51	.21	2.09	.43	2.14	.73
Control	.49	.20	1.90	.39	5.12	2.19
Second session						
Outpatient	1.05	.80	1.95	.81	4.11	1.79
Inpatient	.46	.19	2.05	.37	2.62	1.36
Control	.47	.24	1.92	.42	4.96	1.92

$\underline{H}$ = .07, ns; inpatients, first session - $\underline{H}$ = .88, ns; inpatients, second session - $\underline{H}$ = .47, ns.

Recovery time. Group means and standard deviations are shown in Table 5. Significantly faster recovery times were found in the inpatient overresponders than in the control group: first session, $\underline{H}$ = 11.20, $\underline{p} < .001$; second session, $\underline{H}$ = 7.82, $\underline{p} < .005$ (Kruskal-Wallis Test, $\underline{df}$ = 1). No significant difference was found between the outpatient overresponders and the controls: first session, $\underline{H}$ = .14, ns; second session, $\underline{H}$ = 1.24, ns (Kruskal-Wallis Test, $\underline{df}$ = 1).

Hypothesis 2. The Relationship between the Overresponder/Underresponder Differentiation and Stimulus Barrier Ratings

Group means and standard deviations are presented in Table 6. In the analysis of variance performed on the data, all of the schizophrenic groups were found to be significantly different from the control group in the predicted direction of lowest Bellak ratings (indicative of most pathological stimulus barrier functioning) for the overresponder schizophrenics, less pathological ratings for the underresponder schizophrenics, and highest ratings for the controls (see Table 7). All the differences were significant at the $\underline{p} < .001$ level.

As predicted, the Bellak Stimulus Barrier Ratings were very highly correlated with the overresponder/underresponder differentiation. In the multiple regression analyses performed on the non-GSR variables with the overresponder/underresponder dichotomy as the dependent variable, the

Table 6

Group Means and Standard Deviations for Bellak Stimulus Barrier Ratings

Group	Bellak Stimulus Barrier Score M	SD
First session		
Outpatient		
Overresponders	4.55	1.23
Underresponders	6.89	1.17
Inpatient		
Overresponders	3.57	1.72
Underresponders	6.23	1.69
Control	8.95	1.43
Second session		
Outpatient		
Overresponders	4.36	1.12
Underresponders	6.78	.83
Inpatient		
Overresponders	3.75	1.28
Underresponders	6.58	1.44
Control	9.10	1.52

Table 7

Analysis of Variance for Effect of Response Pattern on Bellak Stimulus Barrier Rating

Sample[a]	F(2,37)	Mean Square	p less than
First session			
Outpatient	41.22	69.70	.001
Inpatient	33.62	82.70	.001
Second session			
Outpatient	48.44	81.04	.001
Inpatient	40.62	85.88	.001

[a]For each sample the control group is compared with the overresponder and underresponder groups comprising that particular experimental sample.

Bellak scale was uniformly the best predictor of group membership (with simple correlations significant at the $p < .001$ level). Moreover, the Bellak score accounted for more than half of the total variance in every regression equation except for the first session of the inpatient group. (See Tables 19-22.)

Hypothesis 3. The Relative Instability of the Overresponder/Underresponder Differentiation over Time

Eight of the outpatient schizophrenics changed from one of the maladaptive arousal patterns in the first session to the other maladaptive pattern in the second: four of the first session overresponders exhibited underresponder patterns during the second session, and four of the first session underresponders became second session overresponders. Three of the inpatient schizophrenics shifted patterns: two first session underresponders becoming overresponders during the second session, and one first session overresponder becoming an underresponder. None of the controls exhibited anything other than a normal habituation pattern (viz., exhibiting three to eight orienting responses before habituating to criterion) during either of the two sessions.

As predicted, there was a significantly higher proportion of schizophrenics than normals who changed: outpatients, $z = 3.16$, $p < .001$, one-tailed; inpatients, $z = 1.80$, $p < .041$, one-tailed (computed by the test of difference between two proportions, Bruning & Kintz, 1968).

Tests of Additional Relationships

The Relationship between the Overresponder/Underresponder Differentiation and Psychological, Pharmacological, and Demographic Variables

Having established the existence of two dichotomous patterns of responsivity among schizophrenics (Hypothesis 1), it remained to examine how these two subgroups compared on the other measures obtained during the study.

To permit the computer analysis of the great amount of data involved in this exploratory segment of the present study, certain of the assumptions of the available statistical procedures had to be partially compromised. The distributions of certain of the measures suggest the applicability of nonparametric procedures for which computer programs were not available. Furthermore, an analysis of variance would have been preferable to the proliferation of *t*-tests used in this section. To compensate for the lack of homogeneity of variance, the *t*-tests utilized separate variance estimates, with a proportionate reduction in the degrees of freedom in each case. To reduce the likelihood of a Type I error (from the proliferation of *t*-tests), more stringent criteria were applied in the rejection of the null hypotheses: A result was seen as being significant only if it was found to be generally significant across both testing sessions. Furthermore, the more conservative, two-tailed probabilities were employed, even where the predictability of the direction of the

differences would have allowed the use of one-tailed *p*-values.

Indices of subjective experience. Group means and standard deviations for the scores obtained on the Beck Depression Inventory and the Taylor Scale of Manifest Anxiety are presented in Table 8.

The analysis of the Beck scores yielded no significant differences between the overresponder and underresponder groups: outpatient, first session - t (16.04) = 1.24, ns; outpatient, second session - t (17.20) = .43, ns; inpatient, first session - t (10.84) = .90, ns; inpatient, second session - t (13.24) = .80, ns.

The group comparison of the Taylor scores indicated a highly significant difference in the outpatient sample, first session, in the direction of higher anxiety levels for the overresponders, t (17.42) = 3.97, $p < .001$. A similar trend was present in the outpatient second session, although the difference fell short of significance, t (17.96) = 1.94, ns ($p < .069$). There were no significant differences in the inpatient sample: first session, t (8.32) = 1.05, ns; second session, t (12.11) = 1.05, ns.

Indices of observed pathology. The group means and standard deviations for the scores obtained on the 10 scales of the Structured Clinical Interview are presented in Table 9 for the outpatient sample, and in Table 10 for the inpatient sample. The t values for the overresponder/underresponder comparisons are shown in Table 11.

Table 8

Group Means and Standard Deviations for the Beck Depression Inventory and the Taylor Scale of Manifest Anxiety

Group	Beck		Taylor	
	M	SD	M	SD
First session				
Outpatient				
Overresponders	7.18	4.14	16.45	4.43
Underresponders	4.67	4.77	8.67	4.30
Inpatient				
Overresponders	6.29	7.27	13.43	8.79
Underresponders	6.69	6.24	9.62	5.19
Control	1.50	1.15	5.75	1.89
Second session				
Outpatient				
Overresponders	5.82	5.36	14.36	7.12
Underresponders	4.78	5.38	8.67	6.04
Inpatient				
Overresponders	6.12	6.53	13.25	7.69
Underresponders	6.83	5.47	9.92	5.74
Control	2.25	1.29	5.60	2.06

Table 9

Group Means and Standard Deviations for Structured Clinical Interview Scores for Outpatient Sample

Scale	Overresponders M	Overresponders SD	Underresponders M	Underresponders SD
First session				
Anger-Hostility	1.59	1.23	.63	1.22
Conceptual Dysfunction	1.88	1.40	1.83	1.05
Fear-Worry	2.55	1.13	.66	1.12
Incongruous Behavior	1.22	1.68	.32	1.20
Incongruous Ideation	3.09	1.46	2.35	1.15
Lethargy-Dejection	.48	1.26	1.34	.46
Perceptual Dysfunction	1.56	1.51	.70	1.40
Physical Complaints	1.46	1.84	.91	1.53
Self Depreciation	1.08	1.00	1.28	.85
Sexual Problems	.64	1.48	1.40	1.42
Second session				
Anger-Hostility	1.28	1.03	.77	1.66
Conceptual Dysfunction	1.52	1.45	1.33	1.49
Fear-Worry	2.44	1.13	.73	1.20
Incongruous Behavior	1.27	1.46	.86	1.27
Incongruous Ideation	2.75	1.63	2.67	.42
Lethargy-Dejection	.55	1.09	1.15	.96
Perceptual Dysfunction	1.31	1.52	.91	1.36
Physical Complaints	1.02	1.63	1.07	1.78
Self Depreciation	1.03	.66	1.34	.37
Sexual Problems	1.21	1.59	.43	1.26

Note. Scores are standardized on the basis of 0 for normals. Higher scores indicate greater pathology.

Table 10

Group Means and Standard Deviations for Structured Clinical Interview Scores for Inpatient Sample

Scale	Overresponders M	Overresponders SD	Underresponders M	Underresponders SD
First session				
Anger-Hostility	.94	1.53	1.27	1.45
Conceptual Dysfunction	2.30	1.24	2.74	.86
Fear-Worry	.96	1.52	.48	1.34
Incongruous Behavior	2.08	1.29	2.15	1.74
Incongruous Ideation	2.52	2.10	2.57	1.52
Lethargy-Dejection	.77	1.66	.97	1.48
Perceptual Dysfunction	1.01	1.73	.63	1.19
Physical Complaints	.58	1.65	.52	1.40
Self Depreciation	.90	.78	.98	.91
Sexual Problems	1.96	1.10	1.23	1.39
Second session				
Anger-Hostility	1.28	1.60	.76	1.22
Conceptual Dysfunction	2.58	.49	2.51	.74
Fear-Worry	1.60	1.43	.56	1.36
Incongruous Behavior	2.05	1.12	2.13	1.40
Incongruous Ideation	2.90	1.52	2.08	1.70
Lethargy-Dejection	1.07	1.37	1.53	.96
Perceptual Dysfunction	1.90	1.61	.68	1.23
Physical Complaints	.46	1.57	.10	1.13
Self Depreciation	.91	.74	1.02	.88
Sexual Problems	1.66	1.32	1.43	1.41

Note. Scores are standardized on the basis of 0 for normals. Higher scores indicate greater pathology.

Table 11

t-Tests for Differences in Structured Clinical Interview Scales for Overresponder and Underresponder Groups

Scale	*t* Values for Overresponder/ Underresponder Differences	
	Outpatient	Inpatient
First session		
Anger-Hostility	1.75(17.25)	.50(9.71)
Conceptual Dysfunction	.09(17.88)	.83(9.18)
Fear-Worry	3.72(17.25)*	.70(11.10)
Incongruous Behavior	1.38(17.75)	.11(15.89)
Incongruous Ideation	1.27(17.99)	.06(9.51)
Lethargy-Dejection	1.59(13.03)	.27(11.24)
Perceptual Dysfunction	1.33(17.69)	.52(9.17)
Physical Complaints	.72(17.98)	.08(10.71)
Self Depreciation	.50(17.96)	.21(14.22)
Sexual Problems	1.18(17.48)	1.28(15.14)
Second session		
Anger-Hostility	.81(12.84)	.78(12.25)
Conceptual Dysfunction	.29(17.02)	.26(18.00)
Fear-Worry	3.24(16.69)*	1.61(14.66)
Incongruous Behavior	.67(17.90)	.14(17.29)
Incongruous Ideation	.16(11.56)	1.13(16.32)
Lethargy-Dejection	1.29(17.86)	.81(11.52)
Perceptual Dysfunction	.63(17.83)	1.82(12.30)
Physical Complaints	.07(16.53)	.55(11.81)
Self Depreciation	1.33(16.14)	.30(16.91)
Sexual Problems	1.23(17.99)	.37(15.88)

Note. *t* values were computed utilizing separate variance estimates. The numbers in parentheses represent degrees of freedom.

*$p < .005$

A highly significant difference was found in the outpatient sample on the Fear-Worry Scale during both sessions, in the direction of more pathology in the overresponder group. There was also a trend across both outpatient sessions in the direction of higher Lethargy-Dejection Scale scores for underresponders, although these differences fell short of significance.

None of the overresponder/underresponder comparisons in the inpatient sample were significant, nor did any trends that might have approached significance hold across the two sessions.

Medication differences. Group means and standard deviations for the milligram-equivalent daily dosages of the three classes of psychotropic medication are presented in Table 12. From the *t*-tests presented in Table 13, it may be seen that there were no significant differences or even consistent trends in the outpatient sample. In the inpatient sample, overresponders were found to be receiving a significantly higher daily dosage of anti-Parkinsonian medications ($p < .03$) for the first session, although this difference did not even approach significance during the second session. Inpatient overresponders tended to be on higher doses of antidepressant medications than their underresponder counterparts, although this trend fell short of significance (p less than .137 and .062 for the first and second sessions respectively).

Differences in psychiatric hospitalization. The group

Table 12

Group Means and Standard Deviations for Daily Dosage of Antipsychotic, Antidepressant, and Anti-Parkinsonian Medications

Group	Mg-Equivalent Daily Dosage of Medications					
	Antipsychotic		Antidepressant		Anti-Parkinsonian	
	M	SD	M	SD	M	SD
First session						
Outpatient						
Overresponders	18.91	24.79	1.00	2.49	4.55	6.99
Underresponders	16.22	15.76	0	0	2.67	5.20
Inpatient						
Overresponders	45.71	52.28	9.00	11.42	7.71	2.43
Underresponders	68.15	54.95	1.53	3.76	3.38	5.38
Control	0	0	0	0	0	0
Second session						
Outpatient						
Overresponders	18.36	24.63	.73	2.41	3.64	7.09
Underresponders	16.89	16.16	.33	1.00	3.78	5.24
Inpatient						
Overresponders	41.00	50.02	9.12	10.58	5.75	4.20
Underresponders	73.17	54.30	.83	2.89	4.33	5.52
Control	0	0	0	0	0	0

Note. See Appendix C for table of mg-equivalent dosages.

Table 13

t-Tests for Differences in Age, Length and Number of Psychiatric Hospitalizations, and Daily Mg-Equivalent Dosages of Antipsychotic, Antidepressant, and Anti-Parkinsonian Medications for Overresponders and Underresponders

Variable	*t* Values for Overresponder/ Underresponder Differences	
	Outpatients	Inpatients
First session		
Age	.75(17.78)	.31(12.78)
Length of Hospitalizations	.01(18.00)	.11(14.84)
Number of Hospitalizations	.29(16.00)	.12(8.31)
Medication		
Antipsychotic	.29(17.10)	.90(12.97)
Antidepressant	1.33(10.00)	1.68(6.78)
Anti-Parkinsonian	.69(17.88)	2.47(17.73)*
Second session		
Age	.46(16.68)	.25(16.25)
Length of Hospitalizations	.41(15.94)	.22(17.85)
Number of Hospitalizations	.28(17.98)	1.06(10.35)
Medication		
Antipsychotic	.16(17.30)	1.36(16.03)
Antidepressant	.49(13.88)	2.16(7.70)
Anti-Parkinsonian	.05(17.86)	.65(17.57)

Note. *t* values were computed utilizing separate variance estimates. The numbers in parentheses represent degrees of freedom.

*$p < .03$

means and standard deviations for number and length of psychiatric hospitalizations are shown in Table 14. The *t*-tests (see Table 13) revealed no significant differences between the overresponders and underresponders.

Age differences. The group means and standard deviations are presented in Table 14. There were no significant differences between the groups (see Table 13 for *t* values).

Differences in schizophrenia subdiagnosis. The distribution of subdiagnoses of schizophrenia in each group is shown in Table 15. It was found that overresponders and underresponders did not differ significantly with respect to subdiagnosis: outpatient, first session - χ^2 (4) = 5.86, ns; outpatient, second session - χ^2 (4) = 2.83, ns; inpatient, first session - χ^2 (3) = 3.23, ns; inpatient, second session - χ^2 (3) = 3.61, ns. Despite the overall lack of significance, it is worth noting that in every case the most often occurring diagnosis among underresponders was chronic undifferentiated. It was also interesting that for the outpatient sample the most commonly occurring diagnosis among overresponders was paranoid.

Demographic variables. The representation of the educational levels in the overresponder and underresponder groups is shown in Table 16. The differences were found to be nonsignificant: outpatient, first session - χ^2 (4) = .90, ns; outpatient, second session - χ^2 (4) = 1.63, ns; inpatient, first session - χ^2 (6) = 5.90, ns; inpatient, second

Table 14

Group Means and Standard Deviations for Age and Length and Number of Psychiatric Hospitalizations

Group	Age (in years)		Length of Accumulated Hospitalizations (in months)		Number of Hospitalizations	
	M	SD	M	SD	M	SD
First session						
Outpatient						
Overresponders	30.4	9.67	4.1	4.85	1.0	1.10
Underresponders	33.6	8.73	4.1	3.92	.8	.60
Inpatient						
Overresponders	35.1	9.21	140.3	63.33	3.4	2.15
Underresponders	33.7	9.52	143.8	77.92	3.5	1.27
Control	32.4	9.04	0	0	0	0
Second session						
Outpatient						
Overresponders	32.7	9.06	3.7	4.12	1.0	1.00
Underresponders	30.8	9.70	4.6	4.80	.9	.78
Inpatient						
Overresponders	34.9	8.82	138.5	58.19	4.0	2.00
Underresponders	33.8	9.80	145.2	81.50	3.2	1.19
Control	32.4	9.04	0	0	0	0

Table 15

Frequency of Schizophrenic Subdiagnoses in the Overresponder and Underresponder Groups

Diagnosis	Outpatient		Inpatient	
	Overresponder	Underresponder	Overresponder	Underresponder
	First session			
Hebephrenic	1	0	0	2
Catatonic	0	0	1	0
Paranoid	6	2	3	4
Acute	1	1	0	0
Residual	1	0	0	0
Chronic Undifferentiated	2	6	3	7
	Second session			
Hebephrenic	1	0	0	2
Catatonic	0	0	1	0
Paranoid	5	3	2	5
Acute	1	1	0	0
Residual	1	0	0	0
Chronic Undifferentiated	3	5	5	5

Table 16

Frequency of Educational Levels in the Overresponder and Underresponder Groups

Educational Level	Outpatient		Inpatient	
	Overresponders	Underresponders	Overresponders	Underresponders
First session				
Grade School	0	0	0	1
Some Jr. High School	0	0	0	2
Completed Jr. High	1	1	1	0
Some High School	1	1	3	5
High School Graduate	2	2	2	4
Some College	6	5	1	0
College Graduate	1	0	0	0
Graduate Education	0	0	0	1
Second session				
Grade School	0	0	0	1
Some Jr. High School	0	0	0	2
Completed Jr. High	1	1	1	0
Some High School	1	1	5	3
High School Graduate	2	2	2	4
Some College	7	4	0	1
College Graduate	0	1	0	0
Graduate Education	0	0	0	1

session - χ^2 (6) = 6.63, ns.

The relation of marital status to the overresponder/underresponder differentiation (as set forth in Table 17) was found not to be significant; outpatient, first session - χ^2 (3) = 3.36, ns; outpatient, second session - χ^2 (3) = 2.89, ns; inpatient, first session - χ^2 (2) = 1.32, ns; inpatient, second session - χ^2 (2) = 1.51, ns.

The representation of occupational levels in the overresponder and underresponder groups is presented in Table 18. In this case, too, the relationship was found not to be significant: outpatient, first session - χ^2 (2) = 3.50, ns; outpatient, second session - χ^2 (2) = 1.88, ns; inpatient, first session - χ^2 (2) = 1.49, ns; inpatient, second session - χ^2 (2) = 2.93, ns.

Multiple regression analysis. Using the overresponder/underresponder differentiation as the criterion, multiple regression analyses were done including all of the non-GSR variables except educational level, marital status, occupational status, and subdiagnosis of schizophrenia. (These variables were omitted because their measurement was only on a nominal level and thus did not meet the required statistical criteria.)

As mentioned in the report of results of the tests of Hypothesis 2, the Bellak Stimulus Barrier Rating was found to be the number one predictor of group membership in each of the regression analyses. (See Table 19, outpatients, first session; Table 20, outpatients, second session;

Table 17

Frequency of Levels of Marital Status in the Overresponder and Underresponder Groups

Level of Marital Status	Outpatient		Inpatient	
	Overresponder	Underresponder	Overresponder	Underresponder
First session				
Single	10	7	6	10
Married	0	1	1	1
Separated	1	0	0	0
Divorced	0	1	0	2
Second session				
Single	9	8	7	9
Married	0	1	1	1
Separated	1	0	0	0
Divorced	1	0	0	2

Table 18

Frequency of Occupational Levels in the Overresponder and Underresponder Groups

Occupational Level	Outpatient		Inpatient	
	Overresponder	Underresponder	Overresponder	Underresponder
First session				
Never Employed	0	1	0	3
Unskilled	10	5	4	5
Skilled	1	3	3	5
Second session				
Never Employed	0	1	0	3
Unskilled	8	7	5	4
Skilled	3	1	3	5

Table 19

Multiple Regression Analysis of Overresponder/Underresponder Differentiation with Bellak Stimulus Barrier Rating, Psychological Scales, Hospitalization, and Medication, for Outpatients, First Session

Variable	Multiple R	R Square	R Square Change	Simple R
Variables individually accounting for at least 5% of total variance				
Bellak Scale	.73152	.53511	.53511	-.73152
Taylor Scale	.78986	.62388	.08877	.68227
SCI Scale 8	.82632	.68280	.05892	.16415
Anti-Parkinsonian Medication	.86585	.74969	.06689	.15554

Table 20

Multiple Regression Analysis of Overresponder/Underresponder Differentiation with Bellak Stimulus Barrier Rating, Psychological Scales, Hospitalization, and Medication, for Outpatients, Second Session

Variable	Multiple R	R Square	R Square Change	Simple R
Variables individually accounting for at least 5% of total variance				
Bellak Scale	.78388	.61446	.61446	-.78388
SCI Scale 5	.84330	.71116	.09669	.03393
SCI Scale 2	.87195	.76029	.04913	.06910
SCI Scale 10	.93273	.86999	.10970	.27142

Table 21, inpatients, first session; and Table 22, inpatients, second session.) In each case, this variable singly accounted for between 38% and 61% of the total variance. With the exception of the Bellak rating, no other single variable consistently contributed at least 5% of the variance in each equation. The milligram-equivalent dosage of anti-Parkinsonian medication figured significantly in all of the equations except for the outpatient, second session (where it was the last variable entered into the equation), although its simple correlation with pattern of responsivity approached significance only for the inpatients, first session. No other variable contributed as much as 5% to the analyses of both sessions of either the outpatient or inpatient samples.

The simple correlations performed as part of the multiple regression program further confirmed the significance of the relationship between response pattern and those variables which had been found to be significant according to the previously conducted *t*-tests. In the outpatient sample, the Fear-Worry Scale of the Structured Clinical Interview was significantly correlated with response pattern: first session, r (18) = .66, $p < .01$; second session, r (18) = .61, $p < .01$. The Taylor Scale of Manifest Anxiety was significantly correlated in the first session, r (18) = .68, $p < .001$, and approached significant correlation in the second session. In the inpatient sample, the relationship between response pattern

Table 21

Multiple Regression Analysis of Overresponder/Underresponder Differentiation with Bellak Stimulus Barrier Rating, Psychological Scales, Hospitalization, and Medication, for Inpatients, First Session

Variable	Multiple R	R Square	R Square Change	Simple R
Variables individually accounting for at least 5% of total variance				
Bellak Scale	.61819	.38216	.38216	-.61819
SCI Scale 10	.71260	.50780	.12564	.26999
Anti-Parkinsonian Medication	.75855	.57539	.06759	.42700
Antipsychotic Medication	.83473	.69678	.12139	-.20425
Length of Hospitalization	.87186	.76015	.06337	-.02386
SCI Scale 9	.93605	.87619	.11604	-.04674

Table 22

Multiple Regression Analysis of Overresponder/Underresponder Differentiation with Bellak Stimulus Barrier Rating, Psychological Scales, Hospitalization, and Medication, for Inpatients, Second Session

Variable	Multiple R	R Square	R Square Change	Simple R
Variables individually accounting for at least 5% of total variance				
Bellak Scale	.72678	.52822	.52822	-.72678
SCI Scale 4	.77851	.60608	.07786	-.03166
Antidepressant Medication	.81126	.65814	.05206	.52342
Anti-Parkinsonian Medication	.84120	.70761	.04947	.14350

and dosage of antidepressant medication (which had appeared only as a nearly significant trend in the t-test analysis) achieved significance: first session, r (18) = .4586, $p < .05$; second session, r (18) = .5234, $p < .02$.

The Relationship between the Changer/Nonchanger Differentiation and GSR, Psychological, Hospitalization, and Pharmacological Variables

The analysis conducted to examine Hypothesis 3 indicated the predicted existence of certain schizophrenics in both the outpatient and inpatient samples who switched from one schizophrenic GSR pattern to the other. Using the change status of GSR pattern as the criterion, the samples were divided into dichotomous groups: changers, who exhibited one schizophrenic GSR pattern during the first session and the other schizophrenic pattern at the time of the second session; and nonchangers, who exhibited the same GSR pattern during both sessions.

Group means and standard deviations by change status and response pattern are given for the outpatient sample in Table 23, and for the inpatient sample in Table 24.

A two-way analysis of variance was computed for change status and response pattern. All of the previously utilized variables were reanalyzed according to this division, with the exception of those demographic variables whose level of measurement did not permit such a procedure, and the GSR variables concerned with response characteristics (where such an analysis would result in comparisons in-

Table 23

Group Means and Standard Deviations for Outpatient Changers and Nonchangers, by Response Patterns, for All Continuous Variables Except Response Characteristics

Variable	Changers				Nonchangers			
	Overresponders (n=8)		Underresponders (n=8)		Overresponders (n=14)		Underresponders (n=10)	
	M	SD	M	SD	M	SD	M	SD
Skin Conductance Level	10.12	2.62	6.22	1.79	14.90	3.22	4.19	.84
Spontaneous Fluctuations	20.75	6.90	2.50	1.20	28.86	11.90	1.40	1.51
Beck Depression Inventory	6.00	2.88	6.12	4.19	6.79	5.60	3.60	5.40
Taylor Scale of Manifest Anxiety	15.00	7.27	13.12	4.42	15.64	5.23	5.10	1.37
Bellak Stimulus Barrier Rating	4.75	.71	6.50	1.07	4.29	1.27	7.10	.88
SCI Scales								
Anger-Hostility	1.84	.74	1.24	1.32	2.20	1.25	.27	1.40
Conceptual Dysfunction	1.92	1.60	1.28	1.58	1.58	1.32	1.82	.98
Fear-Worry	2.80	.53	1.16	1.06	2.32	1.31	.33	1.10
Incongruous Behavior	.36	1.56	.12	1.12	1.75	1.32	.97	1.24
Incongruous Ideation	3.84	.76	2.73	.47	2.39	1.61	2.33	1.06
Lethargy-Dejection	.90	.88	1.14	.99	.30	1.26	1.14	.50
Perceptual Dysfunction	2.58	1.11	.68	1.26	.78	1.28	.90	1.46
Physical Complaints	2.26	1.75	.53	1.72	.66	1.45	1.37	1.50
Self Depreciation	1.52	.48	1.47	.51	.78	.88	1.19	.73
Sexual Problems	1.44	1.56	1.31	1.49	.63	1.48	.59	1.30
Age	29.62	5.07	29.62	5.07	32.71	10.94	34.20	11.18
Length of Psychiatric Hospitalization	2.50	3.74	2.50	3.74	4.71	4.66	5.80	4.24
Number of Psychiatric Hospitalizations	.75	.71	.75	.71	1.14	1.17	1.00	.67
Medication								
Antipsychotic	10.75	10.22	10.75	10.22	23.14	28.69	21.20	17.83
Antidepressant	.38	1.06	.38	1.06	1.14	2.90	0	0
Anti-Parkinsonian	2.25	2.92	2.25	2.92	5.14	8.29	4.00	6.39

Table 24

Group Means and Standard Deviations for Inpatient Changers and Nonchangers, by Response Patterns, for All Continuous Variables Except Response Characteristics

Variable	Changers				Nonchangers			
	Overresponders (n=3)		Underresponders (n=3)		Overresponders (n=12)		Underresponders (n=22)	
	M	SD	M	SD	M	SD	M	SD
Skin Conductance Level	11.33	3.57	7.79	1.56	12.68	3.76	3.34	1.24
Spontaneous Fluctuations	18.00	6.93	3.00	1.00	20.42	5.81	1.18	1.74
Beck Depression Inventory	5.67	2.52	5.67	2.31	6.33	7.40	6.91	6.10
Taylor Scale of Manifest Anxiety	15.67	4.04	13.67	3.06	12.75	8.66	9.23	5.42
Bellak Stimulus Barrier Rating	4.00	1.00	5.33	1.53	3.58	1.56	6.54	1.54
SCI Scales								
Anger-Hostility	2.22	.76	1.82	.73	.84	1.56	.92	1.20
Conceptual Dysfunction	2.76	.48	3.03	.71	2.38	.97	2.58	.81
Fear-Worry	2.17	.53	.90	1.23	1.08	1.54	.47	1.36
Incongruous Behavior	2.17	.42	2.66	0	2.04	1.29	2.07	1.65
Incongruous Ideation	4.12	.36	2.84	.78	2.37	1.80	2.27	1.68
Lethargy-Dejection	.63	1.62	1.17	.94	1.01	1.49	1.25	1.32
Perceptual Dysfunction	1.81	1.57	0	0	1.41	1.75	.74	1.24
Physical Complaints	1.56	1.68	2.53	0	.25	1.48	.02	1.02
Self Depreciation	1.34	.47	1.34	.47	.80	.76	.96	.92
Sexual Problems	1.26	1.45	2.10	0	1.93	1.15	1.22	1.44
Age	29.00	5.29	29.00	5.29	36.50	8.86	34.46	9.78
Length of Psychiatric Hospitalization	104.00	9.54	104.00	9.54	148.17	62.46	150.00	81.70
Number of Psychiatric Hospitalizations	4.00	1.73	4.00	1.73	3.67	2.15	3.27	1.16
Medication								
Antipsychotic	21.33	8.33	21.33	8.33	48.67	53.99	77.27	53.64
Antidepressant	3.33	5.77	3.33	5.77	10.50	11.18	.91	2.94
Anti-Parkinsonian	2.67	4.62	2.67	4.62	7.67	2.54	4.00	5.52

volving characteristics which were not present in underresponders). Employing the MANOVA program (Appelbaum, 1974) and manipulating the order of the analysis, the following tests were performed: interaction effects (examining response pattern, change status, and mean), effects of change status (examining response pattern, interaction effects, and mean), and effects of response pattern (examining change status, interaction effects, and mean). The interaction effects were examined first, and where they proved to be significant, an analysis of simple effects was performed (also utilizing the MANOVA program). The analysis of response pattern effects (i.e., effects of the overresponder/underresponder differentiation) are not included in the present study as they virtually recapitulate the analyses already performed on the effects of this differentiation. Identical analyses of variance were performed for the outpatient and inpatient samples.

Interaction effects in the outpatient sample. F-tests of the significance of the interaction effects for the outpatient sample are presented in Table 25. The interaction effects were significant with regard to four variables: skin conductance level, Taylor Scale of Manifest Anxiety, Perceptual Dysfunction (Scale 7 of the Structured Clinical Interview), and Physical Complaints (Scale 8 of the Structured Clinical Interview).

Table 25

Analysis of Variance for Change Status and Response Level --Interaction Effects for Outpatient Sample

Variable	F(1,36)	Mean Square	p less than
Skin Conductance Level	18.707	109.802	.001
Spontaneous Fluctuations	3.286	201.153	.075
Beck Scale	1.099	26.009	.302
Taylor Scale	7.302	178.279	.010
Bellak Scale	2.465	2.688	.121
SCI Scales			
Anger-Hostility	.179	.269	.678
Conceptual Dysfunction	1.020	1.896	.320
Fear-Worry	.241	.288	.632
Incongruous Behavior	.398	.691	.539
Incongruous Ideation	1.922	2.644	.171
Lethargy-Dejection	.835	.818	.630
Perceptual Dysfunction	5.834	9.739	.020
Physical Complaints	5.644	14.060	.022
Self Depreciation	1.011	.510	.323
Sexual Problems	.009	.020	.920
Age	.062	5.238	.800
Length of Hospitalization	.157	2.797	.696
Number of Hospitalizations	.061	.048	.802
Antipsychotic Medication	.021	8.957	.879
Antidepressant Medication	.889	3.099	.646
Anti-Parkinsonian Medication	.081	3.099	.775

The simple effects of change status in the outpatient sample on skin conductance level, Taylor Scale, SCI Scale 7, and SCI Scale 8. The simple effects of change status on skin conductance level (for the two response patterns) are represented graphically in Figure 3. There was no significant effect of change status on skin conductance level for underresponders, F (1, 36) = 3.10, ns; but there was a highly significant effect for overresponders, F (1, 36) = 19.80, $p < .001$. Response pattern had a significant effect on the skin conductance level of both changers and nonchangers, however: F (1, 36) = 10.37, $p < .003$; F (1, 36) = 113.86, $p < .001$; respectively. It is of considerable interest to note that the skin conductance levels associated with the response patterns of the changers were less extreme than were those of the nonchangers.

The simple effects of change status on the Taylor Scale of Manifest Anxiety are represented graphically in Figure 4. Change status was responsible for a significant effect on the Taylor scores of underresponders in the direction of higher scores for changers, F (1, 36) = 11.72, $p < .002$, but not on those of overresponders, F (1, 36) = .09, ns. The Taylor scores of changers were not effected by response pattern, F (1, 36) = .58, ns, while the scores of nonchangers were affected significantly, F (1, 36) = 26.55, $p < .001$.

A graphic representation of the simple effects of change status on the Perceptual Dysfunction Scale of the

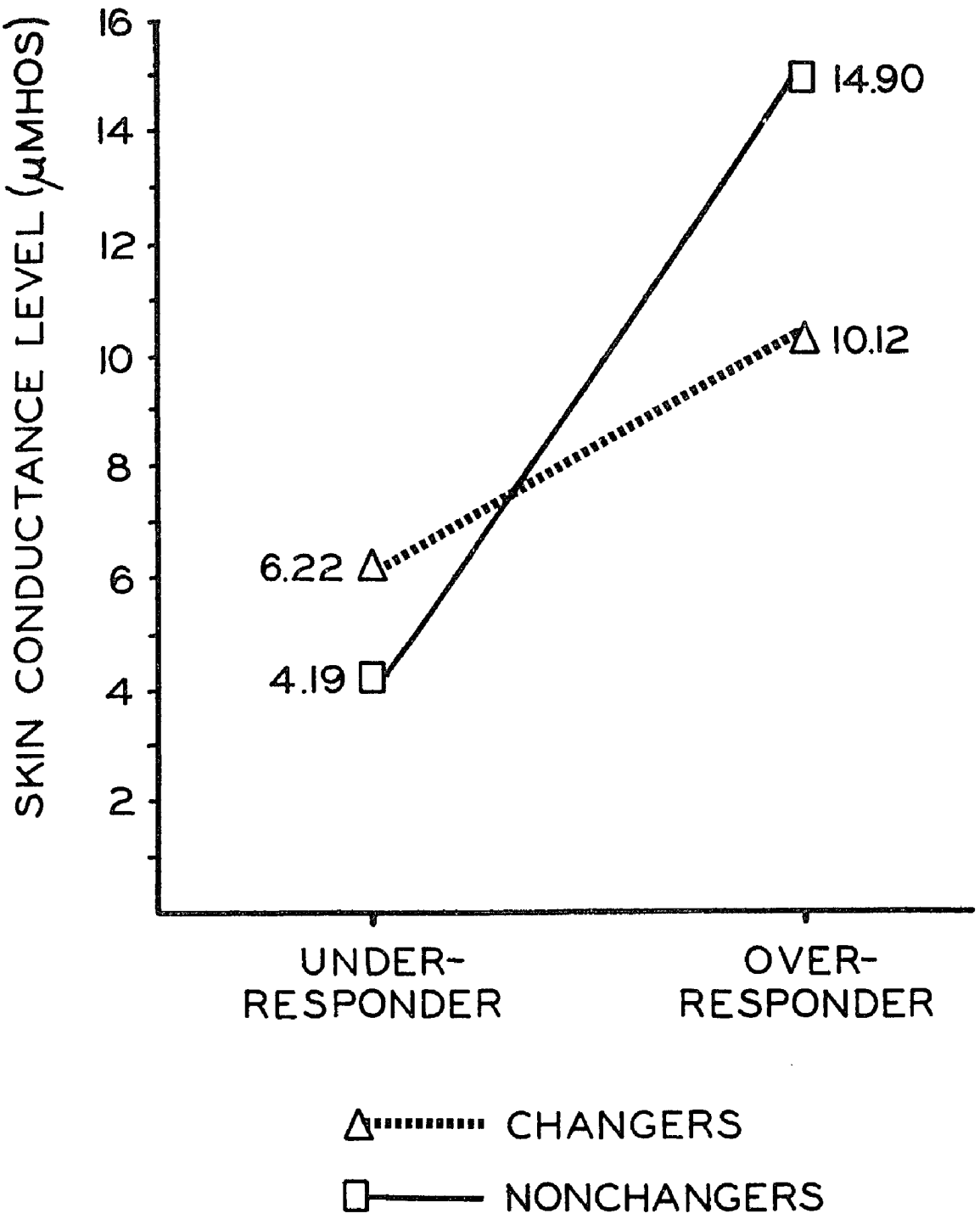

Figure 3. The simple effects of change status on skin conductance level for the two response patterns. Outpatient sample.

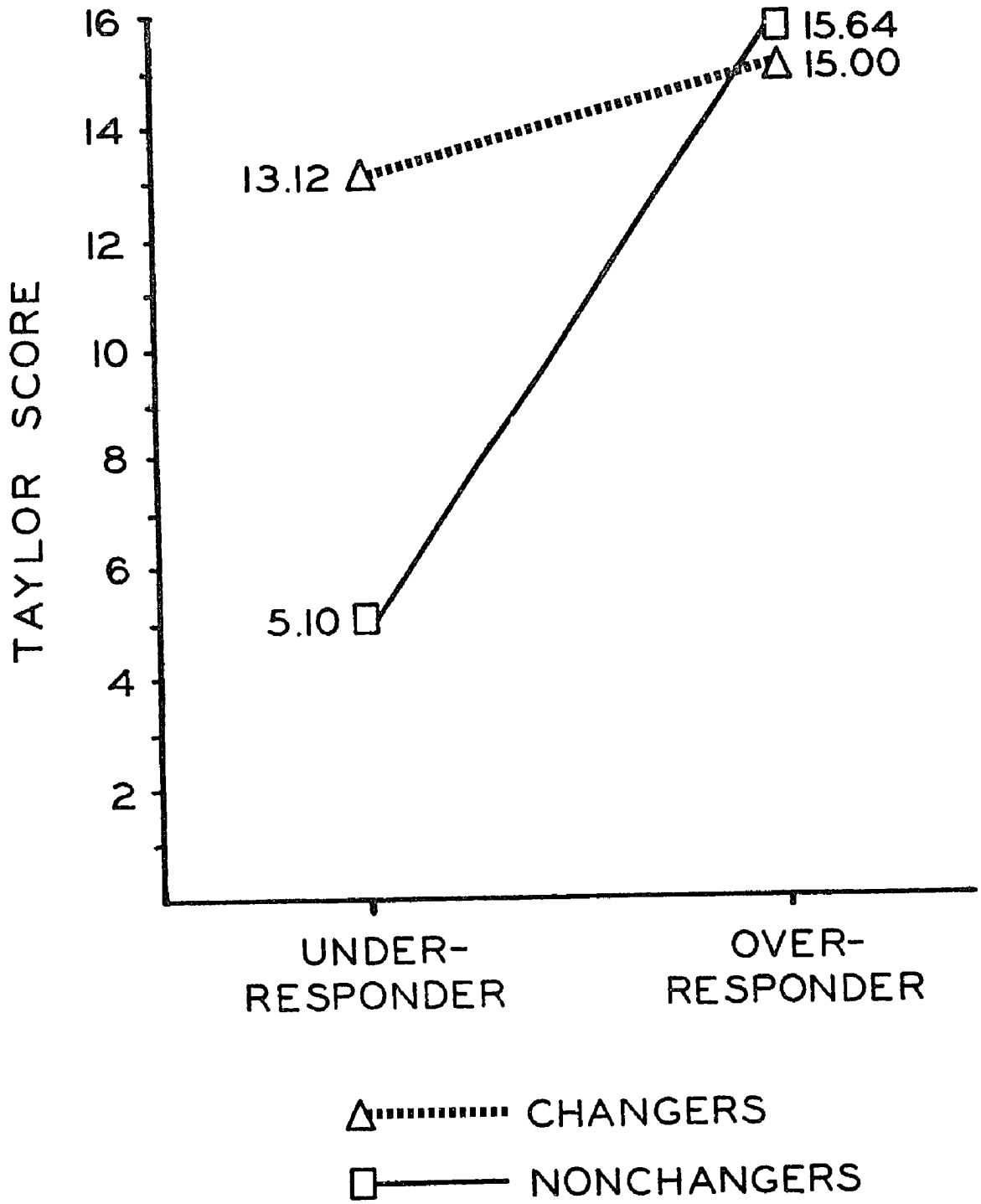

Figure 4. The simple effects of change status on the Taylor Scale of Manifest Anxiety for the two response patterns. Outpatient sample.

Structured Clinical Interview is presented in Figure 5. There was no effect of change status on the level of perceptual dysfunction of underresponders, F (1, 36) = .13, ns; but there was a significant effect for overresponders in the direction of greater perceptual dysfunction for changers F (1, 36) = 9.97, $p < .003$. Conversely, response pattern had a significant effect on the level of perceptual dysfunction of changers, F (1, 36) = 8.70, $p < .006$, but not of nonchangers, F (1, 36) = .05, ns.

The simple effects of change status on the Physical Complaints Scale of the Structured Clinical Interview are presented graphically in Figure 6. Changers were not significantly different from nonchangers in the underresponder pattern, F (1, 36) = 1.17, ns; but they were different in the overresponder pattern, F (1,36) = 4.80, $p < .033$. Whereas response pattern did not effect the level of physical complaints of nonchangers, F (1,36) = 1.25, ns, it did have a significant effect on the changers in the direction of a higher level of complaints among overresponders F (1, 36) = 5.57, $p < .023$.

The effects of change status in the outpatient sample. F-tests of the significance of the effect of change status on the variables under consideration are presented in Table 26.

The effect of change status on the Taylor Scale of Manifest Anxiety was significant, F (1, 36) = 5.30, $p < .026$, even after taking the interaction effects into

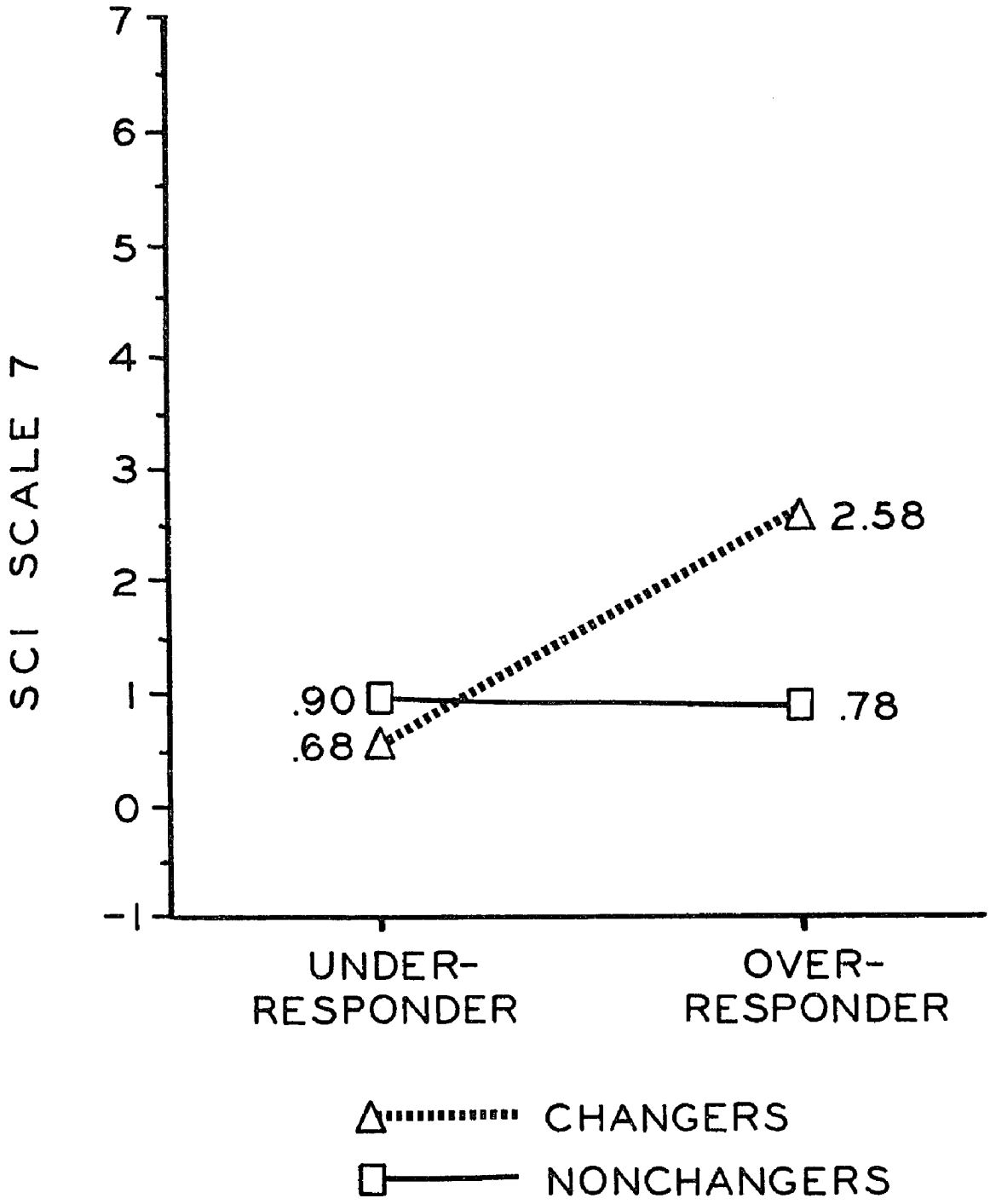

Figure 5. The simple effects of change status on the Perceptual Dysfunction Scale of the Structured Clinical Interview for the two response patterns. Outpatient sample.

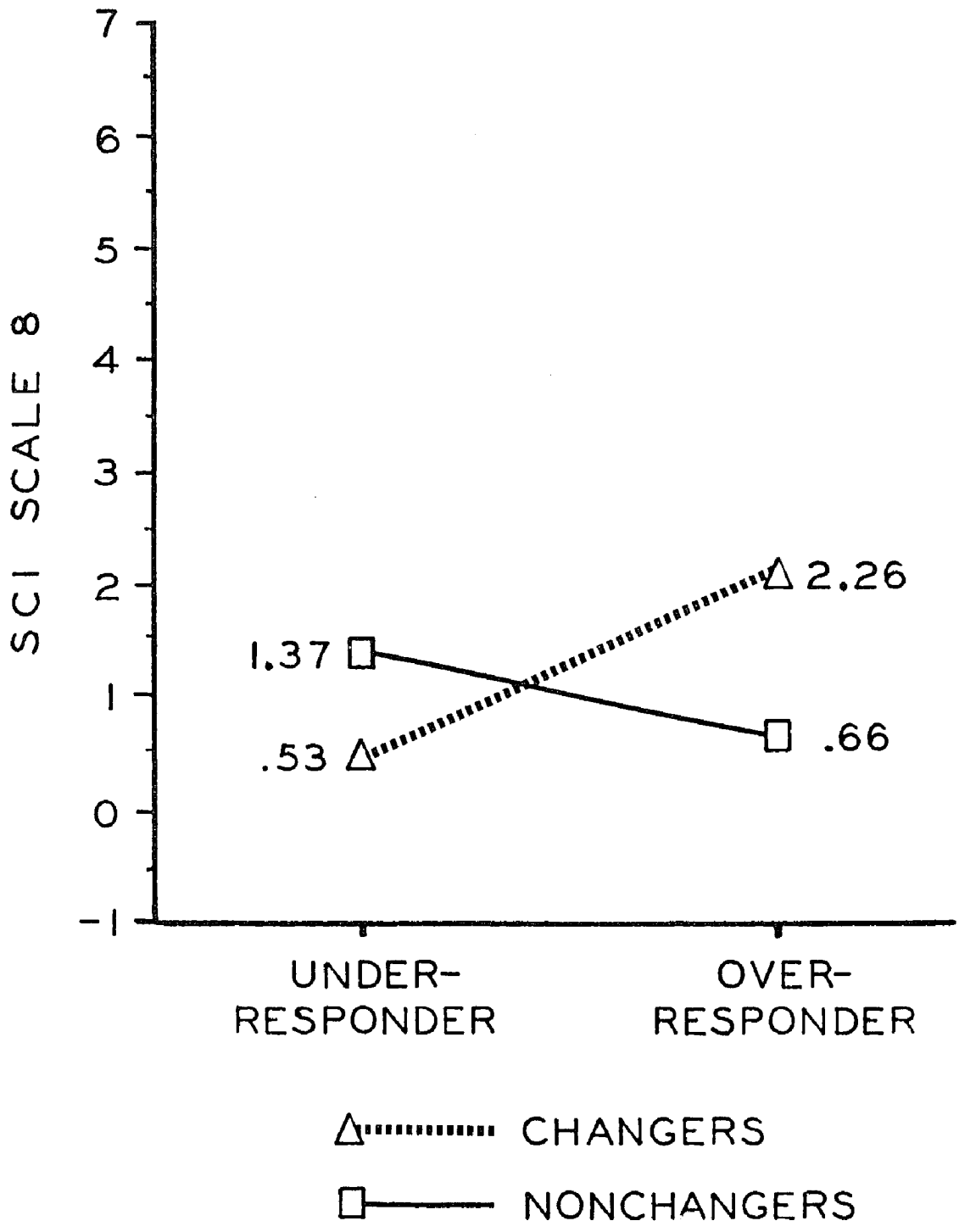

Figure 6. The simple effects of change status on the Physical Complaints Scale of the Structured Clinical Interview. Outpatient sample.

Table 26

Analysis of Variance for Change Status and Response Level --Effect of Change Status, Outpatient Sample

Variable	F(1,36)	Mean Square	P less than
Skin Conductance Level	3.066	17.997	.085
Spontaneous Fluctuations	1.903	116.508	.173
Beck Scale	.303	7.178	.592
Taylor Scale	5.296	129.313	.026
Bellak Scale	.040	.044	.837
SCI Scales			
Anger-Hostility	4.047	6.086	.049
Conceptual Dysfunction	.049	.092	.820
Fear-Worry	3.368	4.025	.071
Incongruous Behavior	6.779	11.765	.013
Incongruous Ideation	5.866	8.069	.019
Lethargy-Dejection	.857	.839	.636
Perceptual Dysfunction	3.593	5.998	.063
Physical Complaints	.546	1.361	.529
Self Depreciation	4.913	2.479	.031
Sexual Problems	2.628	5.563	.110
Age	1.651	139.386	.205
Length of Hospitalization	4.056	72.153	.049
Number of Hospitalizations	1.229	.981	.274
Antipsychotic Medication	2.966	1238.160	.090
Antidepressant Medication	.105	.366	.746
Anti-Parkinsonian Medication	1.334	51.150	.255

account. In contrast to the low underresponder and high overresponder Taylor scores exhibited by the nonchangers, the changers exhibited Taylor scores that were nearly equal for overresponders and underresponders; and the scores for both response patterns of the changers were approximately equal to the overresponder scores of the nonchangers.

Changers scored higher on the Anger-Hostility Scale of the Structured Clinical Interview (Scale 1) than did nonchangers, F (1, 36) = 4.05, $p < .049$. On the Incongruous Behavior Scale (Scale 4), nonchangers were rated more pathological (i.e., they scored higher) than were changers, F (1, 36) = 6.78, $p < .013$. On Scale 5, Incongruous Ideation, changers were rated as being more pathological than were nonchangers, F (1, 36) = 5.87, $p < .019$. Being a changer was also associated with exhibiting a higher degree of Self Depreciation (Scale 9), F (1, 36) = 4.91, $p < .031$.

Length of hospitalization was also significantly related to change status in the direction of less accumulated psychiatric hospitalization for changers, F (1, 36) = 4.06, $p < .049$.

Although failing to reach significant levels, the following trends ($.05 < p < .1$) were noted for changers in comparison with nonchangers: skin conductance levels were generally lower (the above sections on interaction effects have already noted the fact that the skin conductance

levels associated with the response patterns of the changers were less extreme); Fear-Worry scores (SCI Scale 3) were higher; Lethargy-Dejection scores were indicative of more severe pathology (although the analysis of simple effects above has shown also that the changers' scores are more extreme in both directions of this measure); and milligram-equivalent dosages of antipsychotic medication were lower.

Interaction effects in the inpatient sample. F-tests of the significance of the interaction effects are presented in Table 27. The only significant interaction effect was in the case of skin conductance level, F (1, 36) = 6.98, $p < .012$.

The simple effects of change status in the inpatient sample on skin conductance level. The simple effects of change status on skin conductance level is graphically represented in Figure 7. Changers exhibited a significantly higher level of skin conductance than did nonchangers in the underresponder pattern, F (1, 36) = 8.64, $p < .006$, while there was no significant difference in the overresponder pattern, F (1,36) = .72, ns. Whereas there was a highly significant difference in skin conductance level among the nonchangers according to their response pattern, F (1, 36) = 111.82, $p < .001$, there was no significant difference among the changers, F (1, 36) = 3.10, ns. As was the case with skin conductance for the outpatient sample, the inpatient changers exhibited a less extreme range of

Table 27

Analysis of Variance for Change Status and Response Level --Interaction Effects for Inpatient Sample

Variable	F(1,36)	Mean Square	p less than
Skin Conductance Level	6.979	42.251	.012
Spontaneous Fluctuations	1.525	22.546	.223
Beck Scale	.011	.417	.915
Taylor Scale	.070	2.915	.788
Bellak Scale	1.446	3.335	.235
SCI Scales			
Anger-Hostility	.171	.283	.684
Conceptual Dysfunction	.008	.005	.929
Fear-Worry	.290	.551	.600
Incongruous Behavior	.122	.257	.728
Incongruous Ideation	.647	1.729	.568
Lethargy-Dejection	.060	.112	.804
Perceptual Dysfunction	.841	1.658	.632
Physical Complaints	1.275	1.825	.265
Self Depreciation	.044	.030	.830
Sexual Problems	1.750	3.014	.191
Age	.063	5.260	.798
Length of Hospitalization	.001	4.225	.976
Number of Hospitalizations	.077	.195	.779
Antipsychotic Medication	.399	1028.728	.538
Antidepressant Medication	2.463	115.639	.122
Anti-Parkinsonian Medication	.764	16.902	.608

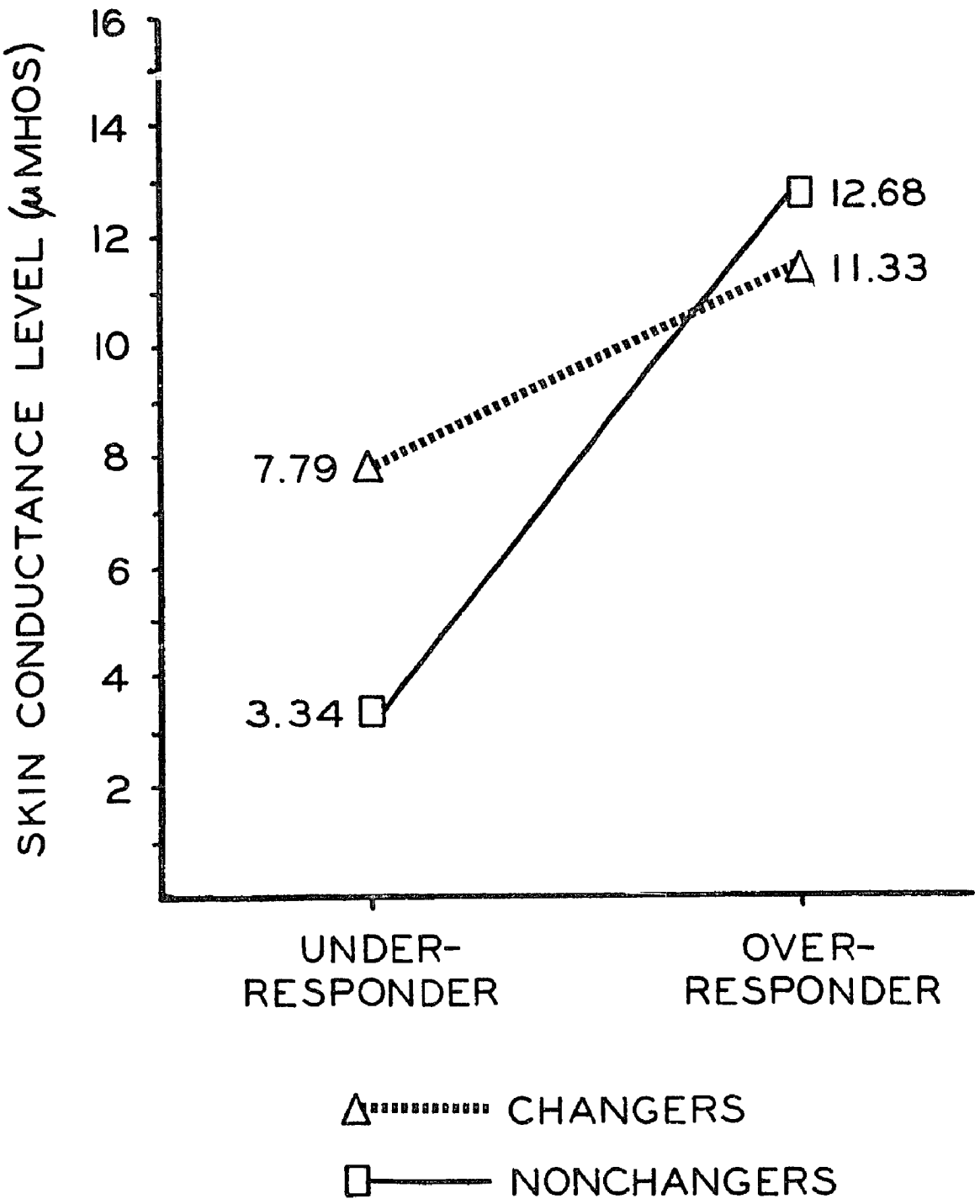

Figure 7. The simple effects of change status on skin conductance level for the two response levels. Inpatient sample.

skin conductance level than did their nonchanger counterparts.

The effects of change status in the inpatient sample. F-tests of the significance of the effects of change status are presented in Table 28. Changers had a significantly higher number of Physical Complaints (Scale 8 of the Structured Clinical Interview) than did nonchangers, F (1, 36) = 12.83, $p < .001$. They also exhibited higher Anger-Hostility ratings (SCI Scale 1) that were on the verge of significance, F (1, 36) = 3.96, $p < .051$.

The only other effect that even approached significance concerned milligram-equivalent levels of antipsychotic medication. As in the outpatient sample, there was a nonsignificant trend towards lower levels of such drugs among the changers, F (1, 36) = 3.38, $p < .071$.

Further observations concerning changers. In every instance, changers, while in their underresponding phase, exhibited isolated trial-one responses. While there were nonchanger underresponders who also exhibited this particular phenomenon (six in all, equally distributed between the outpatient and inpatient samples), it was striking that each of the 11 changers invariably manifested this special subpattern of underresponsivity.

Comparisons of the Outpatient and Inpatient Samples

Differences in GSR characteristics. Group means and standard deviations have been presented earlier in Tables 3 and 5. The t-tests of outpatient/inpatient differences

Table 28

Analysis of Variance for Change Status and Response Level --Effect of Change Status, Inpatient Sample

Variable	F(1,36)	Mean Square	p less than
Skin Conductance Level	2.005	12.138	.162
Spontaneous Fluctuations	.030	.450	.857
Beck Scale	.117	4.582	.734
Taylor Scale	1.642	68.026	.206
Bellak Scale	.345	.795	.567
SCI Scales			
Anger-Hostility	3.961	6.549	.051
Conceptual Dysfunction	1.258	.891	.269
Fear-Worry	1.547	2.941	.219
Incongruous Behavior	.314	.659	.585
Incongruous Ideation	2.547	6.800	.116
Lethargy-Dejection	.137	.258	.714
Perceptual Dysfunction	.072	.141	.787
Physical Complaints	12.827	18.359	.001
Self Depreciation	1.557	1.076	.218
Sexual Problems	.033	.056	.852
Age	2.545	210.974	.116
Length of Hospitalization	2.006	10220.599	.162
Number of Hospitalizations	.559	1.414	.534
Antipsychotic Medication	3.383	8717.460	.071
Antidepressant Medication	.602	28.274	.551
Anti-Parkinsonian Medication	2.281	50.425	.136

are given in Table 29.

There were no significant outpatient/inpatient differences that were stable across both sessions. In the first session, the recovery time of inpatients was significantly shorter than that of the outpatients, but this difference was not significant during the second session. In the second session, inpatients exhibited significantly fewer spontaneous fluctuations, while outpatient overresponders showed a significantly higher response amplitude than did inpatient overresponders. There were no other significant differences in GSR characteristics.

Differences in psychological ratings. Group means and standard deviations were reported earlier in Tables 6, 8, 9, and 10. The *t*-tests of outpatient/inpatient differences are presented in Table 30.

For every comparison except the first session overresponders, the inpatients were rated as significantly more pathological on the Conceptual Dysfunction Scale (Scale 2 of the Structured Clinical Interview). The inpatient underresponders were also seen as being more pathological with regard to Incongruous Behavior (SCI Scale 4). Although this trend extended to the overresponders as well, it did not approach a significant level of difference. In the first session, the outpatient overresponders were rated significantly higher on the Fear-Worry Scale (SCI Scale 3), and there was a mild trend in the same direction during the second session. The only other significant difference was

Table 29

t-Tests for Differences in GSR Characteristics of Outpatient and Inpatient Samples

Variable	*t* Value for Outpatient/Inpatient Differences	
	Overresponders	Underresponders
	First session	
Skin Conductance Level	.18(12.79)	1.09(19.99)
Amplitude	1.81(12.50)	--
Latency	1.71(12.98)	--
Recovery Time	4.32(13.31)**	--
Spontaneous Fluctuations	.72(15.75)	.09(20.00)
	Second session	
Skin Conductance Level	.70(15.58)	2.07(14.99)
Amplitude	2.35(11.47)*	--
Latency	.35(14.77)	--
Recovery Time	2.05(16.93)	--
Spontaneous Fluctuations	2.15(16.10)*	1.42(17.96)

Note. *t*-tests were computed utilizing separate variance estimates. Numbers in parentheses represent degrees of freedom.

*$p < .05$

**$p < .001$

Table 30

t-Tests for Differences in Bellak Stimulus Barrier Ratings, Beck Depression Inventory, Taylor Scale of Manifest Anxiety, and Structured Clinical Interview Scales for Outpatient and Inpatient Samples

Scale	*t* Values for Outpatient/Inpatient Differences			
	Overresponders		Underresponders	
	First Session	Second Session	First Session	Second Session
Bellak	1.33(9.32)	1.09(13.93)	1.08(20.00)	.39(18.05)
Beck	.30(8.52)	.11(13.30)	.86(19.72)	.86(17.55)
Taylor	.84(7.98)	.32(14.50)	.47(19.23)	.48(16.88)
SCI Scales				
1	.94(10.82)	.01(11.15)	1.25(16.59)	.02(14.09)
2	.67(14.14)	2.26(12.93)*	2.16(15.01)*	2.19(11.00)*
3	2.38(10.17)*	1.39(12.92)	.35(19.15)	.31(18.40)
4	1.23(15.28)	1.32(16.92)	2.92(19.99)**	2.17(18.22)*
5	.63(9.72)	.21(15.83)	.39(19.78)	1.15(12.71)
6	.40(10.42)	.89(13.01)	.36(15.10)	.90(17.31)
7	.69(11.61)	.81(14.70)	.12(15.50)	.39(16.35)
8	1.05(13.98)	.76(15.59)	.62(16.26)	1.43(12.75)
9	.41(15.18)	.35(14.16)	.79(18.12)	1.13(15.56)
10	2.16(15.44)*	.67(16.63)	.28(17.15)	1.72(18.36)

Note. *t* values were computed utilizing separate variance estimates. The numbers in parentheses represent degrees of freedom.

*$p < .05$

**$p < .01$

the isolated fact that during the first session inpatient overresponders exhibited more Sexual Problems (SCI Scale 10) than did their outpatient counterparts.

A graphic representation of the Structured Clinical Interview profiles of the average scores (first and second sessions) of overresponders and underresponders in the outpatient and inpatient samples is shown in Figure 8. It is evident that, for most of the scales, there is less difference between inpatients in each response pattern than there is between outpatients. Also, the scores of inpatients of both response patterns tend to fall between the scores of the overresponder and underresponder outpatients. It would appear that there is less effect of response pattern on these psychological measures for the inpatient sample.

Differences in age, psychiatric hospitalization, and psychotropic medication. Group means and standard deviations have been previously presented (see Tables 12 and 14). The *t*-tests of outpatient/inpatient differences are presented in Table 31.

There were no significant differences in the ages of the various groups.

As degree of institutionalization was the criterion for the formation of the two samples, there were predictable differences with regard to length and number of psychiatric hospitalizations.

The inpatient underresponders were receiving a signif-

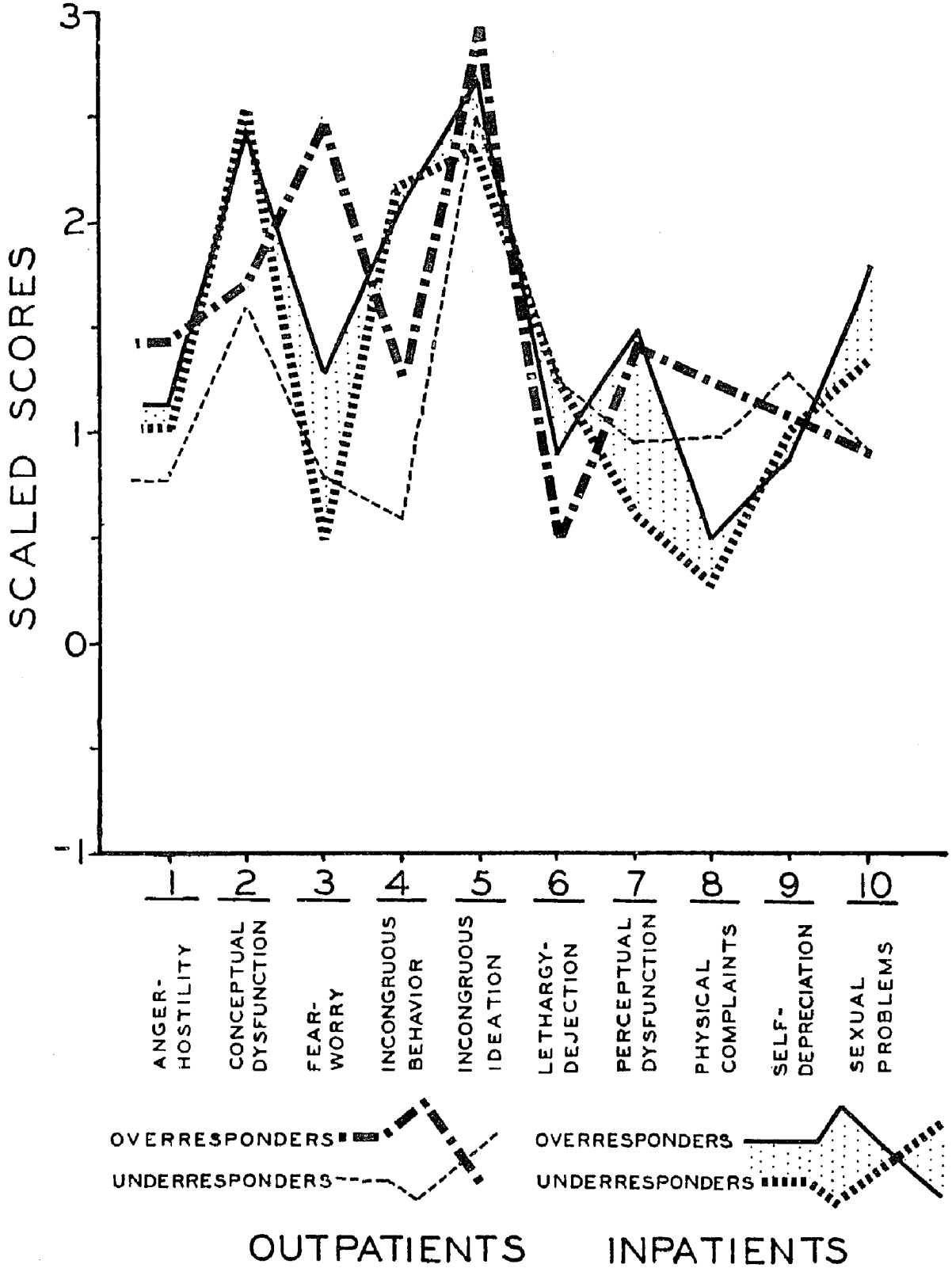

Figure 8. Structured Clinical Interview profiles for inpatients and outpatients, by response pattern.

Table 31

t-Tests for Differences in Age, Length and Number of Psychiatric Hospitalizations, and Daily Mg-Equivalent Dosages of Antipsychotic, Antidepressant, and Anti-Parkinsonian Medications for Outpatient and Inpatient Samples

Variable	*t* Values for Outpatient/Inpatient Differences	
	Overresponders	Underresponders
First session		
Age	1.03(13.41)	.05(18.32)
Length of Hospitalizations	5.68(6.04)**	6.45(12.09)**
Number of Hospitalizations	2.77(8.02)*	6.55(18.19)**
Medication		
Antipsychotic	1.27(7.74)	3.22(14.71)*
Antidepressant	1.83(6.37)	1.48(12.00)
Anti-Parkinsonian	1.38(13.36)	.03(17.76)
Second session		
Age	.52(15.48)	.71(17.48)
Length of Hospitalizations	6.54(7.05)**	5.97(11.10)**
Number of Hospitalizations	3.90(9.56)*	5.27(18.75)**
Medication		
Antipsychotic	1.18(9.48)	3.40(13.49)*
Antidepressant	2.20(7.53)	.56(14.30)
Anti-Parkinsonian	.81(16.49)	.24(17.86)

Note. *t* values were computed utilizing separate variance estimates. The numbers in parentheses represent degrees of freedom.

*$p < .01$

**$p < .001$

icantly higher daily milligram-equivalent dosage of antipsychotic medication than were their outpatient counterparts. There were no other significant medication differences.

The difference between the proportions of changers in each sample. It was shown in the tests of Hypothesis 3 that a higher proportion of both the outpatient and inpatient schizophrenic samples changed GSR pattern than did the controls. It was further predicted that a higher proportion of outpatients would change patterns than would inpatients. A test of the difference between the proportions of changers in the two schizophrenic samples proved to be significant in the predicted direction, $z = 1.77$, $p < .04$, one-tailed.

V--DISCUSSION

The results of the present study clearly support the hypotheses it set out to examine: Schizophrenics do exhibit one of two specific maladaptive patterns of arousal; these patterns are highly correlated with patterns of stimulus barrier functioning; and certain schizophrenics change from one pattern to the other. In the following section, the nature and implications of these patterns are discussed, and the clinical picture found to be associated with each pattern is explored. The existence of schizophrenics who change patterns is discussed, and the nature of this phenomenon and the clinical findings associated with it are elucidated. The implications of these findings for the general theory of schizophrenia is then explored. Finally, suggestions for further research are enumerated and the clinical implications are discussed.

The Two Patterns of Arousal and Stimulus Barrier in Schizophrenia

Arousal is a highly complex and multidimensional facet of human functioning. The present study chose to examine three specific aspects of arousal, as reflected in changes of skin conductance: orienting activity, spontaneous fluctuations of skin conductance, and baseline level of activation.

The orienting response, as described by Pavlov (1941) and Sokolov (1960), is a momentary increase in arousal occasioned by the presentation of a novel stimulus. Reflected in several physiological systems, an orienting response represents the organism's recognition that it has been presented with some input that potentially requires a reaction--classically, either fight or flight. The increase in arousal is preparatory to just such a reaction. The amplitude of an orienting response represents a measure of its intensity. The latency of an orienting response is a measure of the speed of the individual's reaction, while the recovery time is indicative of the amount of time required to return to the individual's normal level of activation. Habituation to a particular stimulus (i.e., the eventual cessation of orienting response following the repeated presentation of a stimulus) represents a process through which the individual becomes satisfied that a particular stimulus does not represent any danger or necessitate any specific action in response to it.

Spontaneous fluctuations of skin conductance are orienting response-like reactions without any observable external stimuli. While fluctuations of this variety can be caused by certain physical activities (e.g., a sudden movement, scratching, a sudden exhalation of breath), such fluctuations were excluded from the current data. The remaining spontaneous fluctuations must be viewed as reactions to the thoughts and affects arising from the internal life

of the individual. Spontaneous fluctuations thus represent the body's reaction to "stimuli" produced internally--namely, the internal impulse life of the individual. It would appear that as with external stimuli, impulses from within are evaluated by the organism before they are admitted to awareness, and that this process involves a heightening of certain facets of physiological functioning.

The characteristics of an individual's orienting activity and of his spontaneous fluctuations reflect an extremely basic level of his responsivity to stimulation. They represent the process by which stimuli--of both internal and external origins--are recognized as either requiring or not requiring further reaction.

The third aspect of arousal examined in the present study was baseline level of activation. This measure is inextricably bound up with orienting activity--both being affected by it and playing a role in its extent. Together with the characteristics of an individual's orienting activity, this measure of general activation state gives a more complete picture of the overall arousal of that individual.

As predicted by the various two-factor arousal theories (e.g., Gruzelier, 1973; Lapidus & Schmolling, 1975), schizophrenics were found to exhibit at all times one of two dichotomous patterns of arousal--neither of which was exhibited by any of the controls. These two patterns, termed the overresponder and underresponder modes, were found to be closely related to two distinguishable levels

of maladaptive stimulus barrier functioning (as measured by the Stimulus Barrier Rating of Bellak et al., 1973).

As reflected in the psychophysiological measures of skin conductance characteristics, the overresponders were slow to habituate, exhibiting a high number of orienting responses to a repeated stimulus. They also exhibited a very high incidence of spontaneous fluctuations in skin conductance level. The baseline skin conductance level of the overresponders was abnormally high. Finally, the outpatient overresponders exhibited greater response amplitudes than did controls.

In sharp contradistinction to this pattern, the underresponders exhibited either a complete absence of orienting response activity, or the total extinguishing of orienting responsivity after an isolated initial response. This group showed remarkably few spontaneous fluctuations, and their baseline level of skin conductance was abnormally low.

Sophisticated recent understandings of stimulus barrier (e.g., Gediman, 1971; Bellak et al, 1973) maintain that this phenomenon is a complex area of active ego functioning. Stimulus barrier is seen as concerned with the regulation and modulation of the individual's fundamental relationship to stimulation--of internal as well as of external origin--relating to whether or not the organism permits itself to recognize and react to a specific stimulus. As in the case of sophisticated understandings of arousal, the need to differentiate the issues of characteristics of responsivity

from baseline levels is also recognized.

In the present study, the striking relationship found between patterns of psychophysiological arousal (as reflected in GSR patterns) and distinct levels of stimulus barrier functioning (as measured by the Stimulus Barrier Ratings of Bellak et al., 1973) supports the assertion, suggested by the literature, that these are two quite different approaches which describe an identical aspect of human function: the individual's most basic pattern of responsivity to both the external world and his own internal impulse life.

The results of the present study showed that schizophrenics of both patterns exhibited pathologically maladaptive levels of stimulus barrier functioning. The overresponders, however, were rated as significantly more pathological in this regard than were the underresponders.

The normally functioning individual is able to screen and modulate the amount and type of stimulation that is admitted into his consciousness. Probably employing both constitutionally determined and subsequently learned functioning of the autonomic nervous system, he is capable of maintaining an adaptive level of contact with stimuli from the environment and with impulses from his own internal psychic life. In this way the individual is able to be responsive without being traumatically overwhelmed by stimulation. This balance is reflected physiologically in his ability to react to novel stimuli on the one hand, while

being able to habituate to them in a reasonable amount of time on the other. This degree of responsivity allows for a productive level of stimulation, reflected in moderate levels of baseline arousal. The presence of a moderate amount of spontaneous fluctuations is suggestive of a similarly adaptive level of responsivity to internal stimulation.

It would appear that the schizophrenic is particularly deficient with respect to dealing with stimulation--be it internal or external in origin. The selective filtering of stimulation, which is accomplished by the individual without this sort of pathology, is absent in the schizophrenic: Stimuli either elicit an unusually intense level of reaction to which the schizophrenic is unable to become acclimated, or they are virtually excluded from awareness, producing no reaction at all. Similarly, schizophrenics exhibit two extremes of responsivity to internal stimulation, exhibiting an extremely abnormal excess or paucity of spontaneous fluctuations. The schizophrenic therefore shows unproductive baseline levels of arousal: He is either hyperaroused or hypoaroused, depending upon his overall pattern.

These findings of hyperarousal and hypoarousal in schizophrenics (as compared with normal controls) are consistent with the inverted-U relationship between arousal and performance efficiency discussed by Schmolling and Lapidus (1972), in which schizophrenics would be expected to fall at the extreme ends of arousal functioning, while the most

efficiently functioning normals would be expected to show moderate levels of arousal.

The overresponder is in a continuously high state of arousal. He is bombarded by stimuli to which he is continually having to react. Moreover, his reactions are of an intensity that seems to indicate that these stimuli are being interpreted as potentially dangerous.

The physiological correlates of orienting response--increased cardiac activity, heightened muscle tension, palmar sweating, etc.--have long been recognized as fight or flight reactions. When stimuli are of an intensity great enough to produce an actual threat to the well-being of the individual, extreme reaction is appropriate and necessary. On the other hand, a well functioning individual will naturally respond to a novel stimulus of moderate intensity with a moderate version of these reactions, until he interprets that the stimulus does not pose any real threat. At this point habituation takes place. In the case of mild, non-threatening stimuli, no orienting response need occur.

The schizophrenic overresponder, however, reacts to moderate stimuli as though they present a real and present danger: His reactions are of high intensity, and they persist. His threshold sensitivity is low, and his integrating and coping functions are similarly deficient.

The painful extent to which this explanation is true is best demonstrated anecdotally in the case of a 24 year old schizophrenic who was part of the inpatient sample in this

study. During his first testing session, this patient, W.T., was administered the tone habituation sequence with the audio signal generator mistakenly set at 15 dB instead of the appropriate setting of 75 dB. This lower level of intensity, used in the study to screen prospective subjects for acceptably acute auditory functioning, is approximately the equivalent of a telephone busy signal when the receiver is held one foot from the listener's ear. Despite the mild nature of the stimulus, W.T. exhibited a full-scale orienting response 14 out of the 15 times it was presented! (It should be mentioned that this session was not included as part of the data in the study, due to this irregularity.)

Stated phenomenologically, these findings suggest that the overresponder reacts to the normal range of environmental stimuli (and, by extrapolation from the findings concerning spontaneous fluctuations, he similarly reacts to internal impulses) as though they were all potential danger signals. He is more sensitive, more reactive, and less able to integrate his experience in such a way as to enable him to modulate adaptively his reactions. He is thus in a constant state of extreme expectation and arousal, resembling the activated psychotic described by Lapidus & Schmolling (1975).

The underresponder, on the other hand, is in an unusually low state of baseline arousal. He has reduced his contact with the stimulus-laden world, and has ceased to exhibit the type of responsivity that is essential for adap-

tive functioning. Even his responsivity to his internal, impulse life has virtually been extinguished, as reflected by the virtual absence of spontaneous fluctuations. Schizophrenics of this pattern resemble the retarded psychotics described by Lapidus & Schmolling (1975).

In considering this pattern, one is reminded of the clinical observation by Engel (1962) that certain individuals with constitutionally low sensory thresholds exhibit a "heightening" of their stimulus barrier, associated with a defensive withdrawal and reduction of incoming stimuli. The underresponders do, in fact, achieve less pathological stimulus barrier ratings on the Bellak structured interview than do overresponders.

This finding must be interpreted, however, with respect to what is meant by "less pathological." Underresponders, by becoming unreactive to stimulation, achieve lowered levels of baseline arousal. The resultant low level of general activation most certainly occasions a decreased amount of clinically observable active pathology (with underresponders being rated as more pathological only with respect to the Lethargy-Dejection Scale of the Structured Clinical Interview). Nevertheless, the inverted-U relationship that has been observed to hold between arousal and performance efficiency suggests that this extreme of the arousal continuum is just as dysfunctional as the opposite. It is noted that the maladaptive nature of the stimulus barrier functioning of underresponders is different in kind

from that of the overresponders, but not necessarily in degree of adaptation. A limitation of the Bellak Stimulus Barrier Scale is that it is unidimensional, and that it responds to this difference in pattern solely as one of degree of adaptation.

Bellak et al. (1973) recognize the existence of two distinct aspects of stimulus barrier functioning. They differentiate sensory threshold levels, which they see as responsible for the baseline "state" of the organism, from integrative and coping abilities, which they see as being more implicated in patterns of responsivity. Nevertheless, in their Stimulus Barrier Scale, they combine these two facets into a single, one-dimensional judgement. They fail adequately to provide for the existence of precisely that divergence in stimulus barrier patterns that the present study has demonstrated to exist in schizophrenia.

Factors Relating to the Overresponder and Underresponder Patterns

The response patterns of schizophrenics in this study were unrelated to a number of variables concerning demography or clinical history. Age, education, occupation, and marital status did not play a significant role; and the effects of psychopharmacological intervention were of relevance only for the inpatient schizophrenics. The traditional subdiagnoses of schizophrenia were unrelated to the response pattern distinction, with the exception of a trend

indicating that schizophrenics classified as chronic, undifferentiated type were likely to be underresponders.

In the outpatient sample there were two striking psychological correlates of response pattern: Overresponders were more anxious--both by clinical observation (the Fear-Worry Scale of the Structured Clinical Interview) and by self-report (the Taylor Scale of Manifest Anxiety); and underresponders were observed to be more depressed (the Lethargy-Dejection Scale of the Structured Clinical Interview), although they did not subjectively acknowledge feelings of depression (the Beck Depression Inventory).

The effects of institutionalization on response pattern are important to the interpretation of the findings of this study. While there was an insignificantly higher incidence of the overresponder pattern in the outpatient sample and of the underresponder pattern in the inpatient sample of the present study, there was still an approximately equal distribution of response patterns in both samples. Apparently, even long, chronic institutionalization (11.9 years being the average length of accumulated psychiatric hospitalization for inpatients in this study) does not influence response pattern. Nevertheless, chronic institutionalization significantly affected the psychological correlates of the two patterns. Of the psychological variables employed in the present study (viz., the Beck Depression Inventory, the Taylor Scale of Manifest Anxiety, and the Structured Clinical Interview) none of the variables which were found

clearly to be related to response pattern in the outpatient sample held for the inpatients. It must be concluded that the effects of chronic institutionalization minimize and obscure the differences in clinical picture between overresponders and underresponders. Thus it is in the outpatient sample that the effects of response pattern on clinical picture can be more accurately observed.

It may be generally concluded, therefore, that the result of the overresponder's sensitivity is a pathologically high level of anxiety. External stimuli and internal impulses are experienced by him as dangerous impingements against which he finds he lacks an adequate defense. He is left in a state of anxious and fearful anticipation: feeling unsure of when the next possibly dangerous impingement will occur, uncertain that he will be able to cope with it, and unconvinced that there can ever be any form of input to him that will not aggravate this painful situation.

The clinical picture that is associated with the underresponder pattern is one of loss of interest and enjoyment, pessimism, and generalized lack of energy and motivation. This picture is reminiscent of the neurasthenic depression that many clinicians (Roth, 1970; Zaslow & Semrad, 1964; et al.) have observed to routinely follow periods of psychotic excitement in schizophrenics. It is as though the underresponder, too, is potentially oversensitive to stimulation and as ill-equipped adaptively to cope with it as is the overresponder. In this case, however, his reac-

tion is to withdraw from the sources of potential stimulation in order to fend off the feared possibility of vulnerability to anxiety-producing impingements upon his being. The underresponder cuts himself off not only from external stimuli, but also from internal impulses and affectual experience. He does not experience himself as being depressed, for he has withdrawn from just such affects. Nevertheless, this massive withdrawal from the world of stimulation results in a general loss of activation which renders the underresponder bereft of motivation and energy.

It has been noted by Gruzelier (1973) that this picture is particularly descriptive of the "burned out," chronic schizophrenic. It may well be that chronic schizophrenics tend toward the underresponder pattern almost exclusively in the later stages of their deterioration. This prediction is consonant with that of Lapidus and Schmolling (1975) that such a pattern would lead to increased deterioration and chronicity rather than recovery. These predictions would account for the disproportionately high number of underresponders among schizophrenics diagnosed as chronic, undifferentiated type.

Schizophrenics Who Change Patterns: An Attempt at Adaptation

Perhaps the most interesting result of the present study was the finding that certain schizophrenics change their response pattern over the course of time. As these

changes took place in both directions, it would appear that these particular schizophrenics alternate between the two response patterns, being at times in the overresponder mode and at other times in the underresponder mode. To test the extent of this alternation, a longitudinal study of changers would be required--with more than the present study's two sessions included. Nevertheless, the current findings indicate that such an alternation of patterns does take place, and its course and stability invite further research.

In no instance in the present study did any schizophrenic exhibit a normal habituation pattern. Even the changers, who swung from one extreme of responsivity to the other, were unable to achieve a normal balance at any time. It must therefore be concluded that the stimulus barrier functioning of schizophrenics is impaired on an extremely fundamental level, resulting in a virtually complete inability to achieve a normal pattern of modulation of contact with the world of stimulation. This dysfunction represents a deficit for which adequate remediation is not readily achieved.

Nevertheless, it would appear that the very existence of alternation between response patterns represents an attempt on the part of the schizophrenic to modulate his responsivity to stimulation. If it is in fact the case that the schizophrenic is unable to achieve a normally modulated pattern of responsivity, he has but three options: He can

remain in an overresponder mode, relating to his environment, but always at the expense of being in a state of distressingly high arousal and anxiety; he can remain in an underresponder mode, at a substantially reduced level of arousal and anxiety, but at the expense of a near total withdrawal from his environment and general enervation; or he can alternate between these extremes, at times opening himself to stimulation, while at other times withdrawing from it.

The analysis of the comparison of changers and nonchangers yielded several results that support the conclusion that a changing response pattern represents an adaptive attempt to compensate for otherwise impaired stimulus barrier functioning. In both the outpatient and inpatient samples, changers exhibited skin conductance levels which were indicative of more nearly normal baseline levels of arousal. Changers in their overresponder phases had levels that were less severely elevated than were those of nonchanger overresponders, while changers in their underresponder phase exhibited levels that were less depressed than were nonchanger underresponders. Thus changers are able to approximate normal levels of arousal.

A most intriguing finding was that changers, when in their underresponder phase, in every case exhibited isolated trial-one responses. This extremely fast habituation appears to represent a somewhat less complete withdrawal from the world of stimulation than does the total absence of any

response characteristic of most nonchanger underresponders. In this way, also, the alternation of response patterns seems to represent more of an attempt to approximate the stimulus barrier functioning of nonschizophrenics.

Further indication of the adaptive features of alternating response patterns was found in the hospitalization history and medication levels of changers. Outpatient changers had a significantly shorter length of accumulated psychiatric hospitalization, and their inpatient counterparts showed a trend in the same direction. There was also a trend in both samples toward lower levels of antipsychotic medication among changers. Schizophrenics who alternate between patterns are therefore less dependent upon institutionalization and psychotropic medication for maintaining an adequate level of functioning. It would also seem that such schizophrenics have a better prognosis than do nonchangers. This assertion is reinforced by the fact that there were significantly fewer changers found among chronically institutionalized patients of the inpatient sample than among the outpatient sample: Changers are capable of more adaptive functioning than are nonchangers.

Since there were so few changers in the inpatient sample, and because of the previously mentioned tendency for the effects of chronic institutionalization to overshadow more subtle psychological differences, the remaining conclusions concerning the nature of changers must be based on those that were found in the outpatient sample of the

present study.

In general, changers exhibit a higher level of active and affective responsivity than do nonchangers. They are more anxious, both in terms of their own report (the Taylor Scale of Manifest Anxiety) and in terms of clinically observed pathology (the Fear-Worry Scale of the Structured Clinical Interview). They are more belligerent and irritable, and they are also more prone to feelings of guilt, inferiority, and worthlessness (the Anger-Hostility and the Self Depreciation Scales of the Structured Clinical Interview). In one sense, these findings indicate a greater degree of pathology in those schizophrenics who alternate patterns; but in a more real sense they indicate that these individuals are more fully alive than are their nonchanger counterparts. There is no reason to assume that nonchangers are any less angry than changers or that they have a higher level of self-esteem. It is far more likely that the changers are simply more in touch with these feelings and more prone to give expression to them. This conclusion is supported by the finding that changers exhibit less incongruous behavior than do nonchangers. The changers have more direct means for expressing what nonchangers express through bizarre acting out.

The adaptive nature of the changer's clinical picture is consistent with Sullivan's conception of the role of anxiety (1953 & 1964). He repeatedly emphasized the importance of anxiety in interpersonal growth:

> To say that a person is able to stand some anxiety is another way of saying that he is able to observe previously ignored and misinterpreted experience in such a fashion that his formulation of himself and of living can change in a favorable direction. (1953, p. 301)

Nevertheless, Sullivan recognized that such changes could take place only when a moderate amount of anxiety was experienced: There is no growth where there is a complete avoidance of anxiety, nor where anxiety reaches a severe level of intensity. Modulated experience, with resulting gradients of anxiety, is necessary for adaptive functioning, and the alternation of response pattern seems to be the schizophrenic's means of attempting to achieve this end.

Whether all schizophrenics potentially have the capacity to alternate response patterns, or whether this ability is constitutionally determined, requires further research. Nevertheless, the results of the present study suggest reasons why a schizophrenic might choose either to be fixated in one of the two patterns or to alternate between them--if, in fact, such a "choice" is possible.

Among nonchangers, there is a clear trade off between the elevated anxiety levels of the overresponder mode and the withdrawal and enervation of the underresponder mode. A striking finding of the present study was that changers in <u>both</u> modes exhibit high levels of experienced anxiety. The Taylor Scores of the changers in both their overresponder and underresponder modes closely approximated those of nonchanger overresponders. Thus the price of being a

changer is having to endure higher levels of anxiety.

This conclusion is consonant with the observations concerning changers: Changers remain more affectively alive than do nonchangers. They are more directly reactive to their environment and exhibit a broader range of active and emotional responses. By alternating between the two extreme patterns of schizophrenic stimulus barrier functioning, they accomplish some degree of modulation that would otherwise not be open to them. In so doing, they achieve a more adaptive relationship with their environment and with their inner psychic life. Nevertheless, this adaptation opens them not only to the possibility of a more productive existence, but also to the potential for fuller affective experience.

Being more in touch with one's experience and feelings is essential to the realization of human potential. Guntrip (1961) describes the process of therapy as an attempt to help the patient tolerate those feelings and needs which the patient has spent his life trying to repress. Nevertheless, the experience of these affects is perceived--even by healthier patients--as dangerous, and great resistance is brought to bear to avoid this outcome. Freud (1915, 1937, & 1940) refers to this as "psychical inertia." As Searles (1961b) points out, the experience of the whole range of human emotions is fraught with intense anxiety for the schizophrenic, who struggles

> with loneliness, unfulfilled dependency, and feelings of abandonment; with fear and guilt, helplessness and

despair; disillusionment and grief. (p. 485)

For the schizophrenic, his past experience with being in touch with his feelings

> consists too little in a flowering of personal capacities and enrichment of life-experience, and too much, rather, in successive personal losses, increasing anxiety and loneliness and, very often, mounting tragedy in the family as a whole. (Searles, 1961a, p. 447)

Thus the path taken by changers, while it provides the best prognosis for recovery and further growth, is one not easily taken, as it is trod only at the cost of considerable risk and pain.

The Nature of Schizophrenia: Some Stimulus Barrier Considerations

Schizophrenic Development

Although no specific data are available concerning the stimulus barrier functioning of preschizophrenics, it may be inferred that such individuals must have markedly impaired stimulus barrier functioning. The magnitude and extent of the deficit is so great in schizophrenics, that it is hard to imagine that their preschizophrenic functioning in this area was not already severely deficient. Moreover, most theories of the genesis of schizophrenia posit for the preschizophrenic just such ego weaknesses and vulnerabilities.

As Lapidus and Schmolling (1975) describe, the preschizophrenic suffers both from lower sensory thresholds and from a relative inability to integrate and cope with sensory input. These deficits are likely to be at least

in part constitutional, particularly with regard to sensory thresholds (Brody & Axelrad, 1966; Greenacre, 1941; A. Freud, 1967). The deficits are certain also to relate to the early experience of the infant with the world of stimulation.

Etiological and experimental factors stressed by many theorists and researchers of schizophrenia (e.g., Benjamin, 1965; Winnicott, 1958 & 1963; Guntrip, 1961 & 1968) point to the importance of pathology in the mother-child relationship in the development of schizophrenic functioning.

Mothering that is so inadequate as either to fail to ameliorate the painful impingement of powerful external stimuli on the infant, or to be unable to satisfy the internal needs of the infant to the point that these impulses reach a level of intensity that is experienced as dangerous, cannot foster effective autonomous stimulus barrier functioning of the infant. The traumatic stimulus overload that is engendered when the mother seriously fails to augment the as yet inadequate stimulus barrier functioning of the infant sets the stage for an all-or-nothing stance toward stimulation. Its results are individuals who are either stimulus hungry, and need a high level of stimulation to feel satiated (Bellak, 1963), or stimulus avoidant, and employ withdrawal and other defensive manoeuvres to minimize stimulation (Engel, 1962). What is not produced in such circumstances is an adaptive capacity for modulating stimulation and the ability to maintain a moderate overall

level of arousal.

These deficits in stimulus barrier functioning greatly increase the preschizophrenic's vulnerability to later damaging impingements which precipitate the actual psychosis. It is clear from the clinical histories of schizophrenics that these precipitating impingements may be of the traumatic variety (Fenichel, 1945) or of the cumulative type (Khan, 1963). In either event, the result is the fracturing of the already precarious stimulus barrier, decompensation, and the emergence of an overtly psychotic condition.

It may be, as is generally suggested by the anxiety-reduction theories of schizophrenia (Freud, 1894; Arieti, 1967), that all schizophrenic psychoses first emerge as overresponder conditions, and that the underresponder mode represents a maladaptive secondary response to the anxiety generated by such states. It is also possible (as suggested by Lapidus & Schmolling, 1975) that the overresponder mode is typical of reactive psychoses, while the underresponder mode represents the stimulus barrier style of process schizophrenia.

In any event, it would appear that a more adaptive outcome than the permanent adopting of either response pattern is an alternation between patterns. Schizophrenics who change patterns in such a way as to modulate their overall stimulus barrier functioning would appear to have the best prognosis in terms of achieving adequate levels of function and recompensation.

Nevertheless, one must conclude that even those schizophrenics who successfully recompensate (i.e., regain an adequate level of functioning) are deficient with respect to stimulus barrier functioning. This conclusion is drawn from the fact that all of the outpatient schizophrenics in the present study showed one of two maladaptive stimulus barrier patterns, despite the fact that many of them had recovered sufficiently from the acute phase of their illness as to be able to hold jobs and maintain at least a minimal level of social interaction.

The Loss of Ego Boundaries in Schizophrenia

Freud, even in his most rudimentary formulation of stimulus barrier (1895), placed it spatially at the outer surface of the organism, at the point of interaction with the external world. Later in his writings, he recognized that the stimulus barrier performed its function in an area analogous to that which is later the province of the mature ego. In the present view of stimulus barrier as an active ego function, it is important to recall that it is viewed as operating to mediate and modulate the individual's responsivity to stimulation. This assertion means that the stimulus barrier is involved in the most fundamental way in governing the interaction between the individual and the external world on the one hand, and between his conscious awareness and unconscious impulse life on the other.

The fact that the schizophrenic does not develop an adaptive level of modulation in such contacts appears

intimately related to his inability adequately to establish realistic boundaries between himself and the external world. As Searles (1959) has described the schizophrenic's boundary functioning:

> It is difficult or impossible for him to differentiate between himself and the external world: his ego boundaries are unstable and incomplete. He often cannot distinguish between memories and present perceptions.... He may be unable to distinguish between emotions and somatic sensations. (p. 317)

Had it been possible for the schizophrenic from infancy to function more comfortably--and therefore more freely--in these areas, he might have been able to achieve a more realistic and accurate sense of these boundaries. However, given the charged nature of such interactions, and the threat the schizophrenic experiences in opening himself to such contact, no such gradual differentiation is able to take place. In one sense, the schizophrenic prematurely differentiates himself from his environment by withdrawing from it in fear; in another sense, he never achieves a mature level of differentiation from it. Thus it is that defects in stimulus barrier functioning play a critical role in the severe blurring of ego boundaries that is characteristically part of schizophrenic pathology.

Hallucinations

The phenomenon of hallucinatory experience, particularly of an auditory nature, has long been recognized as a feature of schizophrenic pathology. Freud (1911) suggested that hallucinations represent an attempt on the part

of the schizophrenic to reestablish contact--albeit pathologically--with the external world. The findings of the present study would appear to support this contention.

The highest degree of perceptual dysfunction (i.e., hallucinatory experience, as measured by the Structured Clinical Interview) was found in the changers in their overresponder phase. It has been concluded that the changer pattern represents the most adaptive of the alternatives open to the schizophrenic, and that it is the overresponder phase of this pattern which is the one that is most open to contact with the external world. Conversely, the lowest levels of perceptual dysfunction were found to prevail in the underresponders in general, who, it has been seen, defensively reduce their contact with the external world and their internal impulse life. Thus it is reasonable to conclude that hallucinations are, in fact, associated with an attempt to relate to the external world. As Arlow and Brenner (1964) have noted, this attempt takes place by means of an alteration of the normal ego functioning, for the purpose of rendering the interaction subjectively less threatening.

This conclusion does not imply that withdrawn, underresponder schizophrenics have adequate reality testing or that they lack an internal phantasy life. On the contrary, the evidence (their high level of Conceptual Dysfunction and Incongruous Behavior as measured by the Structured Clinical Interview) indicates a severe lack of reality testing and a

high degree of psychotically disordered thinking. Such schizophrenics may be almost completely immersed in their own phantasy life.

A hallucination, however, is strictly defined (Hinsie & Campbell, 1970) as a response to a perceived stimulus where no such stimulus exists in reality. The specific nature of the stimulus barrier functioning of the underresponder reduces the likelihood of any response to stimulation--be it external or internal in origin. Thus, even though the underresponder may have phantasies that he lacks the ability to reality test, the phantasies do not elicit from him an active response. In the absence of such an evoked reaction, it is not technically possible to label their experience hallucinatory.

Thus, the reactivity of the overresponder to the world of stimulation--albeit often only to a psychotically distorted projection which is the product of his own phantasy life--is a facet of his functioning that represents an attempt to remain in contact with the external world. It is clearly more adaptive a stance than the withdrawal of the underresponder, although it is often a less socially accepted one.

Suggestions for Further Research

The present study presents information about a cross-section of schizophrenics who exhibit distinctly different patterns of arousal and stimulus barrier functioning. The

results are suggestive of ongoing processes that appear to be fundamental to the understanding of schizophrenic conditions.

The study most immediately suggested by the current findings is an inquiry into the day-by-day (or perhaps even hour-to-hour) variations in those schizophrenics who alternate between response patterns. The extent of this alternation, the longevity of each individual phase, and the factors which appear to precipitate such shifts would be most useful questions to explore.

A long-term longitudinal study examining the vicissitudes of response pattern from the time of the onset of schizophrenic pathology through the various stages of the illness and on into the phases of recompensation and subsequent recovery is also indicated.

Lapidus and Schmolling (1975) predicted a correlation between response pattern and the process/reactive distinction (Phillips, 1953) and distinctions of cognitive style (Witkin, 1965) and attentional style (Silverman, 1966). The experimental verification of these predictions merits further attention in view of the findings of the present study.

The utility of the multiple measures of psychopathology employed in the present study, combining the objectively observed indices of pathology of the Burdock and Hardesty (1969) Structured Clinical Interview and the subjectively reported measures of the Beck Depression Inventory (Beck et

al, 1961) and the Taylor Scale of Manifest Anxiety (1953) should be noted. In the present research this multiplicity of measures provided valuable insight into the clinical picture of particular subgroups of schizophrenia that otherwise might not have been gleaned.

Clinical Applications

In clinical practice, the importance of any diagnostic distinction is contingent upon its resulting in some practical difference in treatment strategy. The overresponder/underresponder differentiation delineated in the present study has obvious significance in differentially planning for the management and treatment of schizophrenics--both in terms of the short-term goals of facilitating the recompensation and deinstitutionalization of overtly psychotic patients, and in terms of more far reaching remediation.

An Indication of Prognosis

It is clearly suggested by this study that those schizophrenics who are capable of alternating back and forth between response patterns have the best prognosis for ultimate recovery. With the increasing degree of attention that is being given to the deinstitutionalization of psychiatric patients, such an indicator may be of considerable value in selecting likely candidates for deinstitutionalization. This is particularly true in the case of chronically hospitalized patients, where the enormous effects of institutionalization alone may be masking clinical signs

that might otherwise be indicative of superior prognosis.

Facilitating the Recompensation of Overtly Psychotic Patients

There is considerable advantage in recognizing the specific type of maladaptive stimulus barrier pattern of an individual schizophrenic. The findings of the present study indicate that the type of intervention that would be optimally utilized to aid a particular schizophrenic towards the alleviation of his symptoms and the reestablishment of a more adaptive level of functioning is dependent upon the nature of his arousal difficulties.

It is clear that the underresponder must be gently encouraged in the direction of increased interaction with his environment. As Gruzelier and Venables (1973) have noted, underresponders are capable of response when stimuli have signal value. Thus such schizophrenics might be induced into accepting higher levels of stimulation through the use of appropriate incentives. As it is their interaction with the human environment that is most in need of improvement, the use of interpersonal incentives such as approval and personal attention are probably of the most value.

Overresponders present quite a different problem. It is clear that, in their more acutely anxious moments, a general reduction in level of stimulation is in order. At other times what appears to be indicated are attempts at building a sense of security in the patient through sup-

portive and unintrusive human contacts (Searles, 1955). By decreasing the general level of apprehension in the overresponder, paths may be opened that will lead in the direction of a more successful focusing of his attention, and thereby to more adaptive functioning.

In an institution where there is a sufficient abundance of trained and responsive staff, and where emphasis is placed on understanding and responding to the specific interpersonal needs and dynamics of individual patients, such appropriately directed interventions are likely to already take place on an intuitive basis. Consistent with the observations of Searles (1959), the findings of the present study must be interpreted as underscoring the importance of such general ward treatment.

Psychopharmacological intervention also would appear to be of considerable importance on the level of facilitating the recompensation of schizophrenics to functional levels. Despite the fact that specific medication differences had little significant effect in determining the specific response pattern of individual schizophrenics, elsewhere (Grinspoon, Ewalt & Shader, 1967; NIMH, Psychopharmacology Research Branch Collaborative Study Group, 1964) the general effects of antipsychotic medication have clearly been shown to be of significant value in hastening the recompensation of schizophrenics. The often noted disappearance of the flaccid, catatonic schizophrenic from the wards of psychiatric hospitals combined with the increased

manageability of floridly psychotic, excited schizophrenics --both coinciding with the advent of the psychotropic utilization of phenothiazines--is suggestive of the conclusion that such medications ameliorate the maladaptive effects of both of the schizophrenic patterns of stimulus barrier dysfunction.

Finally, biofeedback techniques may prove to be of some advantage in providing a structured format for the direct teaching of more adaptive responsivity. Gilbert (1975) has found that the use of instruments which provide direct feedback concerning fluctuations in GSR and muscle tension can be beneficial in aiding schizophrenics to reduce elevated levels of arousal. This area of intervention is still in its infancy, and yet there is much to suggest that the differential application of such techniques to overresponder and underresponder schizophrenics may be useful in hastening their eventual recompensation.

Remediation of Deficits in Stimulus Barrier Functioning

One must conclude that even those schizophrenics who successfully recompensate are deficient with respect to stimulus barrier functioning. The remediation of the basic deficits in this area--to the extent that this is at all possible--is likely to require a very long-term intervention. Such an intervention would of necessity involve a form of corrective emotional experience, in such a way as to eventuate in an altered relationship between the schizophrenic and his environment.

It has been shown that the schizophrenic has come to view any stimulation--which is to say, any direct contact with either the external world or with his own impulse life --as potentially dangerous and experientially discomforting. The therapeutic alteration of this stance would involve the therapist's entering into an almost symbiotic relationship with his patient, so as to augment the patient's stimulus barrier functioning in a way that was not done in infancy. Once the patient had begun to feel more secure in relation to the stimulus-laden world--with the therapist functioning as an intermediary with that world--the process could enter into a final phase in which the therapist would encourage the ultimate gradual differentiation of the patient from him. Ideally such a process would result in an internalization of the auxiliary adaptive stimulus barrier functioning provided by the therapist, and thus in enhanced autonomous stimulus barrier functioning on the part of the patient. As Searles (1963) describes the general process of psychotherapy with schizophrenics:

> The therapist functions as an auxiliary ego to the patient in the patient's struggle with inner conflicts, until such time as, by identification with the therapist's strength, he becomes able to make this greater strength part of his own ego. (p. 698)

Such a therapeutic relationship is by no means easy to achieve or maintain. It involves the experience of intense emotions and extreme anxieties on the part of both the patient and the therapist (Searles, 1961b, 1961c, 1963).

Nevertheless, only such a relationship offers to the

schizophrenic the possibility of achieving the level of trust and integrated functioning essential for the development of a fuller and more adaptive interaction with both the external world and his own internal psychic life.

VI--SUMMARY

Recent research into arousal in schizophrenics points to the existence of two reciprocally functioning systems whose normal balance is disrupted--resulting in two different arousal patterns: one hyperaroused and the other hypoaroused.

The present research further examined these arousal patterns and their relationship to stimulus barrier--viewed as a complex, active ego function.

There were two experimental groups of schizophrenics--inpatients with a history of chronic psychiatric hospitalization (averaging 11.9 years), and outpatients with no more than one year accumulated psychiatric hospitalization; and a control group with no history of any psychiatric difficulties. There were 20 subjects in each group.

Each subject was tested twice, six weeks apart, with the following procedures being administered in order:

1. Beck Depression Inventory (Beck et al., 1961).
2. Stimulus Barrier Interview (Bellak et al., 1973).
3. GSR characteristics were measured in response to a series of 15 tones of moderate intensity.
4. Structured Clinical Interview (Burdock & Hardesty, 1969).
5. Taylor Scale of Manifest Anxiety (1953).

In addition, demographic, pharmacological, and institution-

alization data were gathered from the patients' records.

As predicted, all schizophrenics exhibited one of two patterns: an overresponder pattern, with an elevated skin conductance level, no habituation within the test period, and a high incidence of spontaneous fluctuations of conductance (and, in the case of outpatients, abnormally high response amplitudes); and an underresponder pattern, with a depressed skin conductance level, either no responses at all or an isolated trial-one response, and a low incidence of spontaneous fluctuations. These patterns were markedly different from the normal habituation pattern exhibited by all of the controls: moderate baseline levels, with 3-8 orienting responses, followed by habituation to criterion.

The predicted correlation between response pattern and Stimulus Barrier Ratings was found to be highly significant --with overresponders being rated more pathological.

An exploration of the factors relating to these response patterns found that outpatient overresponders were more anxious (both by clinical observation and their own report), while underresponders were rated higher on a scale of Lethargy-Dejection, but did not report more subjectively experienced depression. In the inpatient sample, these differences seemed to be obscured by the effects of institutionalization. Differences in medication, hospitalization, and clinical picture were insignificant, except for a trend towards chronic undifferentiated schizophrenics being underresponders.

As predicted, certain schizophrenics alternated between response patterns--although at no time did any schizophrenic exhibit a normal habituation pattern. Changers were more common among the outpatients.

The alternation between patterns seemed to serve an adaptive function for schizophrenics--suggesting an attempt at modulation of stimulation otherwise not open to them. Changers exhibited less extreme skin conductance levels, less accumulated institutionalization and lower levels of psychotropic medication. All changers exhibited an initial trial-one response in their underresponder phase --also suggestive of a more modulated approach. They also appeared to be more affectively alive--being rated more pathological with respect to anxiety (both objectively observed and subjectively reported), Anger-Hostility, and Self-Depreciation. Nevertheless, changers were less pathological with respect to Incongruous Behavior, suggesting a more adaptive capacity to express their conflicts.

The results of the present research support the view that stimulus barrier dysfunction and maladaptive patterns of arousal are two ways of viewing the same phenomenon, and that this defect is deeply implicated in the etiology of schizophrenia--particularly with regard to the schizophrenic's unstable ego boundaries and difficulty in maintaining a modulated relationship with his environment.

The results suggest that differential treatment according to response pattern is needed in helping schizo-

phrenics to recompensate.

Further research--particularly of a longitudinal type--into the vicissitudes of response patterns in schizophrenics is strongly indicated.

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Appendix A

CONSENT FORM

You are being asked to participate in a study that will compare how different people react to sounds. The study is going to compare people in hospitals to those who are not in hospitals.

You will be asked to listen through earphones for about fifteen minutes to a series of sounds. During this time your reactions will be recorded on a machine that measures sweating on your hands. You will also be asked a series of questions about yourself and how you have been feeling.

In a few weeks you will be asked to do all these things one more time.

So that I can know a little about your history and what medications you are taking, I will need your permission to look at your hospital/clinic records.

All of the information about you will be kept strictly confidential, and your name will in no way be used in the study.

Thank you very much for your help.

* * * *

I have read and understood the description of this

Appendix A (continued)

study, and I agree of my own free will to take part in it.

I give my permission for Richard Rubens to examine my hospital records to get information for this study.

_____
(Participant's Signature)

_____
(Witness)

_____
(Date)

Appendix B

TIMING OF TONES IN THE HABITUATION SEQUENCE

Tone Number	Number of Seconds Elapsed since Onset of Previous Tone
1	-
2	39
3	45
4	54
5	26
6	57
7	25
8	48
9	34
10	55
11	60
12	24
13	23
14	50
15	36

Appendix C

EQUIVALENT DOSAGES OF MEDICATIONS

Dosage Equivalents of Antipsychotic Agents in the Study[a]

	Estimated Milligram-Equivalent Dose
Chlorpromazine (Thorazine)	100.0
Fluphenazine (Prolixin)	2.5
Haloperidol (Haldol)	2.5
Mesoridazine (Serentil)	37.5
Molindone (Moban)	12.5
Thiorodazine (Mellaril)	80.0
Thiothixene (Navane)	6.0
Trifluoperazine (Stelazine)	7.5

Note. The figures in this table are arithmetic means computed from the ranges cited by Shader and Jackson (1975).

[a]Reference: Chlorpromazine=100.

Dosage Equivalents of Anti-Parkinsonian Agents in the Study

	Estimated Milligram-Equivalent Dose
Benzotropine (Cogentin)	.5
Trihexyphenidyl (Artane, Tremin)	1.0

Note. The figures in this table are those given by Prien (1973).