NONVERBAL COMMUNICATION, RESPONSE TO PERFORMANCE FEEDBACK,
AND PSYCHOPHYSIOLOGICAL ACTIVITY IN DEPRESSION

by

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Abstract

The present study evaluated selected aspects of recent behavioural, cognitive, and psychophysiological theories of depression. Of major concern was Lewinsohn's suggestion that a deficit in social-skill may be a critical determinant of depressive behaviour. An analysis of the concept of social skill suggests that it may involve two component processes: 1) the ability to emit situationally appropriate behaviours that others can identify and respond to discriminatively, and 2) the ability to identify and respond discriminatively to the situationally-appropriate behaviour of others. It follows from Lewinsohn's position that depressed subjects should exhibit deficits in either or both of these processes. In order to evaluate this hypothesis, three groups of subjects -- depressed, nondepressed psychiatric controls, and normal controls -- participated in a two-part experiment. In the first part, which was designed to elicit various forms of facial expressive behaviour, subjects were exposed to a differential classical conditioning procedure, in which one CS was followed by presentation of a "pleasant" pictorial UCS, another CS was followed by an aversive auditory UCS, and a third CS was presented with no consequating event. Videotapes were made of subjects' facial expressions during CS presentation and continuous recordings of subjects' skin conductance and heart rate were taken. In the second part of the experiment, subjects observed the videotapes from the first session of 3 other subjects, one from each group of subjects. In this session, subjects were required to guess, on the basis of changes in the facial expressions of the subjects observed, which of the 3 types of conditioning trial the observed subject was undergoing. During this session, subjects also estimated their anticipated performance
immediately prior to undergoing each of the three sets of judgements. Results of the judgemental task indicated that depressed subjects were the most difficult of all subjects to accurately judge and that this deficit did not seem to be due to response predispositions on the part of depressed subjects. This finding was interpreted as being consistent with Lewinsohn's social-skill hypothesis, but inconsistent with Ferster's notion that the depressive is a poor observer of the environment. The three groups' estimations of their anticipated performance did not differ systematically, thus failing to support Beck's speculation that the depressive is characterized by a generalized set of negative expectations regarding the outcome of future events. More importantly, changes in subjects' verbal ratings of their anticipated performance were highly correlated with the discrepancy between anticipated and actual performance on previous trials for all groups. This finding was inconsistent with predictions from Seligman's "learned-helplessness" model of depression. Finally, psychophysiological data indicated that depressives were electrodermally hyperresponsive in comparison with other subjects, and also exhibited an elevated tonic heart-rate. These findings were interpreted as being inconsistent with speculation that the depressive is refractory to stimulation.
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Chapter I
RATIONALE

The present study evaluated selected aspects of recent behavioural theories concerning the origin and maintenance of depressive behaviour. Although the phenomena of depression have provoked a wide proliferation of speculative accounts as to their origin, (cf. Beck, 1967; Becker, 1974) and a significant quantity of empirical research on the biology of depression (Mendels and Stinnett, 1973; Perris, 1973), only in recent years have behavioural researchers approached the area.

The conceptual background derives from Lewinsohn's (1974a, 1974b; Lewinsohn, Weinstein and Shaw, 1969) multifactorial account of the origins and maintenance of depression. According to this formulation, a number of situational and personal factors are causally related to the production of a low rate of response-contingent positive reinforcement. This latter state of affairs represents the critical eliciting condition for depressive behaviour. Depressive behaviour is thereafter maintained through a number of processes such as increasing social isolation and aversive feedback which produce a "vicious circle" effect maintaining and deepening the depression.

Of the personal factors which Lewinsohn has identified as critical antecedents of depression, perhaps the greatest emphasis has been placed on what has been termed a deficit in "social skill", defined as the ability to emit behaviours that are reinforced by others, and not to emit behaviours that are punished by others. According to this formulation, certain types of behaviour which are essential for effective social interaction may be diminished or entirely absent in the repertoire of depressed
persons. Several different lines of research have provided evidence consistent with this social skill deficit hypothesis, including studies involving the direct observation of the behaviour of depressed and non-depressed subjects in small-group interaction (Lewinsohn, Weinstein, and Alper, 1970; Libet and Lewinsohn, 1973; Libet, Javorek and Lewinsohn, 1973), and studies involving observation of the non-verbal behaviour of depressed subjects in interview situations (Ekman and Friesen, 1974; Shannon, 1970).

An analysis of this concept suggests that what has been termed "social skill" may involve important component processes: (1) the ability to emit behaviours which others can identify and respond to discriminatively, and (2) the ability to accurately identify and respond discriminatively and appropriately to behaviour emitted by others. The former process may be termed "expressive" social communication and the latter "receptive" social communication. Both processes are conceived as involving the phenomenon of stimulus control. In the case of "expressive" social communications, the hypothetical depressive deficit involves an inability to emit behaviours which control the reinforcing and punitive behaviour of others. In the case of "receptive" social communications, it is suggested that the depressive's behaviour may be under relatively weak social stimulus control. More specifically, it may be that the depression-prone individual is relatively unable to respond appropriately to behaviours emitted by others which serve as discriminative stimuli for the reinforcing and punitive behaviour of the individual toward whom they are directed. If the depressive is characterized by a deficit in social skill and if the concept of social skill is meaningfully related to the processes of
expressive and receptive social communication, then depressed individuals might be expected to exhibit deficits in either or both of these areas. These hypotheses were tested in the present study by employing two tasks. In the first task, facial expressive behaviour from depressed and nondepressed subjects was elicited by aversive, neutral and "pleasant" forms of stimulation presented within a tripartite differential classical conditioning procedure. Subjects' facial expressions during this task were videotaped. During the second (judgemental) task, depressed and non-depressed subjects were shown the videotapes of other subjects undergoing the first task and were required to identify, on the basis of facial expression behaviour emitted by the subjects they were observing, which of three different phases of the conditioning procedure the observed subject was undergoing. Three groups of subjects -- depressed, psychiatric control and normal control -- were employed. It was predicted that, relative to nondepressed subjects, depressed subjects would be both difficult for others to judge accurately and inaccurate in making judgements of others.

A second aspect was concerned with the evaluation of predictions from Beck's (1967) and Seligman's (1972, 1975) theories of depression. Beck's theory states that depression occurs among people who exhibit a constellation of disorders of thought, characterized as abnormalities in a primary triad of cognitive schemata. The components of this primary triad include: (1) a negative construction of experience, (2) a negative self-concept, and (3) negative expectations with regard to the outcome of future events. It would seem to follow from Beck's conceptualization that at least the second and third of the above components might be reflected in a tendency for depressed subjects to be relatively pessimistic in com-
parison with nondepressed subjects when required to estimate the adequacy of their performance on a given task. In the present study, subjects participating in the second part of the experiment made judgements on the videotapes of other subjects participating in the first part of the experiment on three separate trials. In order to evaluate Beck's formulation, subjects were required to estimate how well they would perform prior to each of these occasions. On the basis of Beck's theory, it was predicted that, relative to nondepressed subjects, depressed subjects would provide lower estimates when requested to anticipate their performance.

Seligman's theory posits that depression occurs when the individual has acquired a cognitive set which is characterized as "learned helplessness". The central characteristic of this set is said to be the individual's "belief" or "expectation" that his behaviour and its outcome are independent of one another. It would seem to follow from this formulation that an individual who believes that his responding and its outcome are independent events would be relatively unlikely to change his estimations of his future performance in the light of feedback regarding previous performance.

The use of a methodology which required subjects to predict their performance immediately prior to undergoing each of three separate trials on a task allowed for investigation of the extent to which discrepancies between predictions of performance and actual performance were related to the subsequent changes in predictions of performance among depressed and non-depressed subjects. On the basis of Seligman's theory, it was predicted that changes in depressed subjects' estimates of their performance on subsequent trials would be unrelated to discrepancies between estimates of performance and actual performance on prior trials.
A final aspect of the present study dealt with the question of differences between depressed and nondepressed subjects in autonomic activity. Clinical descriptions of depressed patients and certain theoretical positions characterize depression as a state of decreased "arousal" and decreased responsivity to stimulation (cf. Lazarus, 1968, 1972). The results of empirical studies in this regard have been contradictory and inconclusive (e.g., Ban, Choi, Lehman and Adamo, 1966; Lewinsohn, Lobitz and Wilson, 1973). The use of classical conditioning methodology in the present study presented an opportunity to acquire data pertaining to these issues. Thus, measures of electrodermal and cardiovascular activity were taken from all subjects participating in the study.
Chapter II
LITERATURE REVIEW

Depression: Epidemiology and Classification

The authors of several basic reviews of the experimental, theoretical and clinical literature on the topic of depression have been unanimous in agreeing that scientific sophistication in the area is remarkably poor both in terms of an adequate quantity of well-controlled experimental data and in terms of the development of a coherent approach to the organization of such data (cf. Beck, 1967; Grinker, Miller, Sabshin, Nunn & Nunnaly, 1961; Lewinsohn, 1974a, b). Friedman (1964, p. 244) notes that "It seems as if the psychiatric profession has taken for granted that all that can be known about depression has already been discovered and thoroughly described. As a matter of fact, one finds ... that clinically, relatively little new has been added to the description of depressions in general since antiquity. Textbook descriptions of this entity are stereotyped accounts which have been copied from book to book and repeated from generation to generation". This state of affairs is particularly disconcerting when one considers the ubiquity of the problem of depression. McLean and Ledwidge (1974) note that "reactive depression is the primary diagnosis of 19% of Canadian and 25% of American psychiatric facility inpatients." In surveys of the general population, Mayer-Gross, Slater and Roth (1960) found that approximately 3 out of every 1000 people could be characterized as being depressed to a severe enough degree to require treatment. Roth (1959) suggested that the incidence of depression may be as high as 4% in men and 8% in women. Canadian statistics for the year 1970 reveal that 40.46%
of all psychiatric hospital inpatients fall within one of the International Classification of Diseases' categories of depression (McLean & Ledwidge, 1973). Various other epidemiological researches attest to the wide-ranging presence of depression (cf. Silverman, 1968) and further reinforce the belief that the present state of disarray with respect to the scientific understanding of the origins and treatment of depression is a major shortcoming of contemporary research in psychopathology.

One possible source of this confused and confusing state of affairs stems from the lack of an adequate definition of depression. Lewinsohn (1974a, p. 63) notes that

"The term "depression" ... is sometimes used to refer to a normal mood state, an abnormal mood state, a symptom, a symptom syndrome, as well as to a disease process and possibly to a series of disease processes."

The American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders - Second Edition* (American Psychiatric Association, 1968) lists at least 7 major disorders characterized as depressive in nature (e.g., Psychotic Depressive Reaction, Manic-Depressive Illness), and at least 6 other disorders which presumably have depressive elements (e.g., Adjustment Reaction of Adult Life, Asthenic Personality) yet nowhere is it specified what the exact criteria are for describing a particular individual as depressed. Although such a lack of definitional guidelines may be considered trivial by some, it is probable that this shortcoming is responsible for the relatively low reliability of inter-psychiatrist agreement on the diagnosis of depression (Beck, Ward, Mendelson, Mock & Erbaugh, 1962). (This, however, is not an uncommon characteristic of psychiatric descriptive systems (Ullmann & Krasner, 1968) and other general psychological
taxonomies (Mischel, 1968)).

In attempting to come to some sort of definition of what is meant by the term, the question arises as to whether we should speak of "depression" or "depressions". The history of speculation about depression is highlighted by issues and controversies regarding the differentiation and distinction of various "types" of depression (Beck, 1967; Becker, 1974). Thus attempts have been made to formalize the difference between grief and melancholia (Freud, 1917), endogenous and reactive depression (cf. Becker, 1974), psychotic and neurotic depression (cf. Beck, 1967), in addition to the various forms of depression outlined in the currently-used psychiatric classification manual (American Psychiatric Association, 1968). These numerous distinctions, however, are more concerned with an implicit or explicit differentiation of presumed etiologies than with a simple description of observable phenomena which lead to the inference of "depression". McKinney and Bunney (1969), following from Lehman (1959), distinguish between the primary and secondary "symptoms" of depression, the former considered to be invariant in all depressions, while the presence of the latter tends to be more variable. The primary symptoms refer to the presence of a "despairing emotional state", and "the depressive mood". Secondary symptoms refer to "such things as social withdrawal, psychomotor retardation, anorexia, weight loss and sleep disturbances" (McKinney and Bunney, 1969, p. 240). The difficulties involved in this ambiguous distinction can be illustrated by considering the attempt to formulate research analogues of depression based on behavioural phenomena observed in animals subjected to particular experimental manipulations (McKinney and Bunney, 1969; Seligman, 1972, 1974; Suomi and Harlow, 1972). McKinney and Bunney cite as one of the
main factors limiting the generalizability of such models the fact that it is difficult to assess the primary emotional state in animals. As Skinner (1963) has pointed out, however, it is no easier to objectively assess the "primary emotional state" in humans. The labeling process which results in the application of the diagnosis of depression to an individual represents a series of inferences drawn from the observation of overt behaviours such as verbalization of guilt, dysphoria, fatigue, presence of a "sad" facial expression (depressive facies) (Beck, 1967), certain characteristics of voice quality (Hargreaves, Starkweather & Blacker, 1965), decreased verbal productivity (Aronson & Weintraub, 1967; Hinchliffe, Lancashire & Roberts, 1971a) decreased general activity level (Williams, Barlow & Agras, 1972), and various nonverbal cues such as stooped posture and slow gait (Waxer, 1974). Other diagnostic factors include situational and historical factors such as recent loss or separation or the occurrence of an unusually large number of recent life-stresses (Paykel, Myers, Dienelt, Klerman, Lindenthal & Pepper, 1969). Furthermore, Wittenborn (1965) emphasizes that the diagnosis of depression also involves the exclusion of behaviours such as bizarre verbalizations and apparent delusions and hallucinations thought to typify persons labelled as schizophrenic. Yet other authors allow that such things as somatic delusions, and hallucinations characterize the phenomenon termed "psychotic depression". Beck (1967) notes that in certain instances, patients presenting with a variety of somatic complaints but none of the other "classical" signs of depression may actually be suffering from a "masked" form of depression.

Thus, it is apparent that a wide range of phenomena have been subsumed under the single linguistic label "depression". It must be pointed out
however, that many of the traditionally noted signs of depression are
not invariant across all people so described. This point can be illustrated
by considering the interrelationships between clinical ratings of the severity
of depression and many of the somatic changes taken to be signs confirming
the diagnosis of depression. Beck (1967) emphasizes that the presence of
these signs is highly variable, resulting in their low intercorrelations
with each other, and their low (.27-.31) correlation with ratings of the
severity of depression.

The issue of classification is central to, and has occupied the
attention of most authors of treatises on the topic of depression. So
pervasive has been the tendency to dichotomize and classify various types
and levels of depression that Stengel (1964) in a review of such classi-
fication schemes, reported having found 38. The assumption underlying the
various approaches to classification is that differences in patterns of
depressive phenomena reflect the operation of different etiological processes.
Thus, the formulation of a taxonomic scheme represents an early stage of
theory-building.

Of the many distinctions typically made, the two most frequently
made are between endogenous and exogenous (or reactive) depression and
between psychotic and neurotic depression.

The endogenous-reactive distinction is based on the observation that
among psychiatric patients whose problems seem of a depressive nature,
two extreme groups can be roughly discriminated: a group whose depressive
pattern seems clearly to be a reaction to a recent loss or life-stress,
and a group for whom no apparent precipitating factor can be identified and
for whom depressive behaviour seems to have persisted over a relatively
long period of time. Because life circumstances appear to be so intimately linked up with the reactive depressive pattern, its causation has commonly been ascribed to environmental or intrapsychic factors. The endogenous pattern, due to its apparent independence from environmental circumstances, has been attributed more often than not to genetic and biochemical influences (although intrapsychic processes, at times, have been grouped in this class). It is highly possible, however, that the latter could be a case of causal inference by default, since there is no obvious reason why in this case a genetic-biochemical explanation should be either more parsimonious or more valid per se than a psychological explanation. Furthermore, controversy still centres around the validity of the endogenous-reactive distinction. For example, Stenbach (1965), in a retrospective analysis of 86 patients diagnosed as depressed, concluded that in all cases at least one disturbing event could be related to the onset of the depressive episode. The format of this study, however, could not be considered scientifically acceptable. This issue becomes increasingly complex when considering the possible etiological role of precipitating events. Mendels notes that "... the fact that a patient reports an association between a stressful life experience and the onset of an illness ... does not ... constitute proof that the reported stress caused the illness." (Mendels, 1971, p. 28). A variety of explanations of the relationship between stressful events and the onset of depressions is available: (1) the stressful event may temporally coincide with, but be causally unrelated to the onset of depression, (2) the stressful event may be an effect of the depression, rather than vice versa, (3) the life stress may interact with an underlying predispositional factor, (4) the stressful event may have been
a critical eliciting factor.

While factor analytic studies of depression have derived factors reflecting the endogenous-reactive patterns, these factors are typically bipolar, with the terms *endogenous* and *reactive* describing the opposite poles (Carney, Roth & Garside, 1965; Kiloh & Garside, 1963). While the authors argue from these findings that endogenous and reactive depression are independent clinical entities, the fact that the endogenous and reactive patterns represented the extreme points along a single bipolar factor supports a unidimensional continuum interpretation. Controversy regarding the interpretation of these findings still rages (Becker, 1974).

Beck (1967) in summarizing the endogenous-reactive debates concluded that while some differences in symptomatology between the two groups are apparent, little evidence, whether physiological, psychological or genetic supports the proposition that endogenous depression results from some sort of biochemical disturbance, while reactive depression is somehow environmentally determined.

The neurotic-psychotic distinction is another dichotomy about which there is considerable disagreement. The distinction was formulated in order to distinguish those depressions which evidence "gross misinterpretation of reality ... delusions and hallucinations" (Beck, 1967, p. 82) from those which seem less bizarre. As with the endogenous-reactive distinction, the psychotic-neurotic distinction is taken to represent a pair of independent "types" of depression; however, even less research is available here to support the validity of the distinction. What evidence there is would seem to support the view that the neurotic-psychotic distinction primarily
reflects a dimension of severity of depression.

In addition to the endogenous-reactive, and the psychotic-neurotic distinctions, a wide variety of other terminologies has been used to further subdivide depressive phenomena. Yet it is apparent that methodological, conceptual and empirical inconsistencies have frustrated most attempts to bring order to the area. Possibly because of this, the clinical use of these terminologies reflects this inconsistency. The terms endogenous and psychotic, and reactive and neurotic are typically used interchangeably in the clinical setting. Further, Mendels (1971) reported that the term endogenous can also be substituted for the classification of manic-depressive psychosis. With such a degree of confusion both in the literature and in clinical practice, it is little wonder that theories of depression based on such nosologies have rarely been subjected to critical tests.

Theories of depression

The proliferation of theoretical explanations of depressive phenomena has been so widespread that the present discussion cannot hope to provide a representative sampling of them. For this reason, the present discussion will be restricted to those positions which can be deemed to have had a significant impact on the professional community.

Biophysical theories. Probably the oldest tradition with respect to the provision of explanatory models for depression has been to ascribe the origins of depressive behaviour to genetic or biochemical influences. Although the humoural theories of the Greeks can be seen to have relevance here, it was probably Kraepelin (1913) who provided the greatest impetus for the hypothesis that depressive phenomena, particularly the pattern he
termed "manic-depressive psychosis", derive from aberrant physiology.

Kallman (1952) presented impressive evidence to the effect that manic-depressive psychosis is the result of a hereditary predisposition. Utilizing the technique of studying pairs of patients varying in their hereditary similarity to one another, he found a 100% concordance rate for manic-depressive psychosis among identical twins. His studies, however, were subject to a number of methodological difficulties, including problems of diagnosis, sampling bias and determination of zygosity. Later studies attempting to correct for these difficulties reported decidedly more humble figures (Beck, 1967). Most recent research on genetic transmission of affective disorders has been concerned with establishing morbidity rate percentages among relatives of probands diagnosed as suffering from illnesses of psychotic proportions. Much of this research is consistent with a hypothetical genetic contribution within restricted diagnostic groups (Perris, 1973).

Most of the more recent physiological theories of depression derive from observation of the apparent effects on depressed individuals of physical and pharmacological therapies. Kraines' (1965) theory lays particular emphasis on pathophysiology of the hypothalamus as the central etiological variable in depression. Beck (1967) criticises this theory on the grounds that the evidence on which it is based is fragmentary, questionable and inconsistent. A more influential position in recent years has been that of Schildkraut (1965) and his colleagues, and has come to be known as the catecholamine hypothesis. Briefly, the substance of this position has derived from the apparent effectiveness of the monoamine oxidase inhibitors and tricyclic antidepressants in the treatment of depression. The argument
runs somewhat as follows: since these substances (monoamine oxidase inhibitors and tricyclics) seem effective in the treatment of depression, and since their effects seem to involve the provision of an increase in the availability of norepinephrine at receptor sites in the brain, depression might be due to a relative deficit in the availability of norepinephrine at those receptor sites. In addition to the criticism that few, if any, tests of the catecholamine hypothesis have come from the study of depressed humans, it can also be objected that the logic of this argument runs up against the correlation-causality issue and would hold only in a system in which possible alternative modes of causality are extremely restricted. A critical test of this hypothesis would involve experimentally producing a decrease in brain norepinephrine at the appropriate sites and observing whether depressive behaviour resulted. Even so, this would not preclude the possibility that such a norepinephrine depletion could be the result of behavioural processes. Empirical evidence has increasingly supported the proposition that environmental events have numerous effects upon physiological structure and function (Altman, Wallace, Anderson and Das, 1968; Greenough, 1975; Miller, 1972).

A further area of biological research into depression which has provoked some etiological hypotheses has been concerned with sodium and potassium electrolyte balance in depression. Interest in the role of these substances derives from the importance of electrolyte distribution and movement between axons and extracellular fluid in the maintenance of neuronal resting potential and the propagation of action potentials. While a considerable quantity of research has been conducted along these lines, the area is fraught with methodological difficulties and inconsistent findings.
In general, the only consistent finding seems to be that of sodium retention among depressed patients, diminishing with recovery from depression. With regard to changes in potassium metabolism, no consistent patterns have been found (Baer, 1973).

A recent attempt to integrate physiological theories of depression with psychodynamic, object-loss and reinforcement models appears in Akiskal and McKinney (1975).

Intrapsychic and cognitive theories of depression. This section deals with those explanatory models of depression in which the causal process is largely attributed to the operation of hypothetical (and often complex) mental apparatuses.

The classical psychoanalytic position on depression originated in the writings of Abraham (1911, 1916) and was later elaborated by Freud (1917). According to Abraham, the "melancholic" is characterised by a hereditary predisposition toward oral eroticism such that his psychosexual development fails to progress beyond the oral stage. The re-enactment of childhood frustrations in later life produces a libidinal regression to this stage and concomitant melancholia. Freud contended that melancholia is produced by a loss in fact or fantasy that threatens the ego. As a result of this threat, the ego introjects the lost object, and the person's hostility toward that object emerges in the form of masochistic self-derogation. Unfortunately little, if any empirical data have been brought to bear on these hypotheses.

A number of authors have proposed, and attempted to verify the hypothesis that depression is produced by achievement-related attitudes
inculcated in the depressed person during childhood by his or her parents. Cohen, Baker, Cohen, Fromm-Reichmann, and Weigert (1954) argued that depressed patients were raised in families that had been singled out as "different" by the community. Within this context, the parents attempt to achieve higher status by pressuring one person to achieve. The person singled out in this manner thus acquires a predisposition to depression in later life. Becker (1960) reformulated this hypothesis, emphasizing that the child later to become a depressive, reacts to parental pressure to conform and achieve by adopting the values of his parents and other authorities as a means of placating them. In comparing a group of manic-depressive patients with a group of normal controls, equated on age, education and reading ability, he found the manic-depressive subjects to score higher on scales purporting to measure the extent to which achievement is invested with positive value (value achievement), authoritarianism, and conventional attitudes. Curiously, no differences were found on a scale of need for achievement. Katkin, Sasmor, and Tan (1966) contrasted conformity and achievement-related characteristics of depressed and paranoid schizophrenic subjects. In an Asch-paradigm test of conformity, depressed subjects showed a greater tendency to conform. However, in contrast with Becker's (1960) findings, no differences were observed between the groups on a number of questionnaire measures of achievement orientation. The inconsistency between these studies with respect to achievement orientation may reflect differences in the control groups utilized, and suggest that differences in achievement orientation may be more a function of psychiatric status than being specific to depressed patients.

Beck (1967) proposed an entirely cognitive theory of depression,
according to which depression is the result of the activation of a triad of cognitive disturbances that predispose an individual to the various forms of affect, thought and behaviour characteristic of depression. The components of this triad include a tendency to construe experiences in a negative manner, a negative view of the self, and negative expectations regarding the future. This cognitive triad results in a tendency for the individual to misinterpret experience and thus produces the affective, motivational, physical and behavioural disturbances of depression.

According to Beck (1970), the operation of this primary triad can be observed in a number of cognitive errors including those of arbitrary inference, overgeneralization, selective abstracting, etc.

A number of studies by Beck and his colleagues have provided support for this position. The basic paradigm employed in these studies has involved exposing depressed and nondepressed subjects to success and failure experiences while concurrently assessing various measures of performance, expectancy and mood. Loeb, Beck, Diggory, and Tuthill (1967) found that depressed subjects gave lower probability of success estimates than did nondepressed subjects when asked to evaluate their likely future performance on a card-sorting task. Depressed subjects gave lower ratings of their actual performance than did nondepressed subjects, in spite of the fact that their actual performance did not differ. A later study by Loeb, Beck and Diggory (1971) found essentially the same results. These findings are consistent with the notion that depressives evidence negative expectations with regard to the outcome of future events, and a negative construction of experience.

Although Beck's position is reasonably well-elaborated, its value
as an heuristic model of the origin of depression is questionable. It
neglects to explain on the basis of theoretical or empirical considera-
tions how cognitions become disordered, or how disordered cognitions can be
assumed to exert their effects on physiology, behavior, affect or motivation,
and runs afoul of the logical requirement that once one postulates an in-
ternal mechanism as an explanatory device, one must them explain the opera-
tion of the internal device (Skinner, 1953).

Behavioural theories. Concurrent with the recent rapid development of
techniques for the modification of psychopathological problems has been the
willingness of clinicians and researchers to extend the general principles
and conceptual orientation of behavioural psychology to phenomena which have
traditionally been the province of other schools of thought and to problems
which seem, on an intuitive level, to contraindicate a behavioural approach.
Thus, Skinner (1957) initiated the extension of basic principles of operant
behaviour to an analysis of language and thinking, Lindsley (1960) has studied
hallucinations within the context of free-operant responding, various
aspects of schizophrenia have been subjected to intense behavioural analysis
(Salzinger, 1973), and pain and pain-related behaviours have come within the
purview of behavioural approaches (Craig & Coren, 1975; Fordyce, Fowler,
Lehman, DeLateur, Sand and Trieschmann, 1973). In recent years, theorists
working from a behavioural orientation have begun the attempt to account for
the origins and maintenance of the phenomena of depression.

The origins of a behavioural account of depression can be traced
to Skinner's (1953) Science and human behaviour wherein he highlighted a
"weakening (or reduced probability of occurrence) of most forms of behaviour"
as a central feature of depressions and speculated that such a state of affairs could result from a loss of control over the social environment, or a preponderance of aversive control exerted upon the individual by the environment. Although Skinner's remarks were clearly speculative, his major contribution toward the theory of depression was in pointing out the feasibility of accounting for a presumably intrapsychic phenomenon within the context of a functional analysis of behaviour.

Ferster (1965, 1966) provided the first elaboration of Skinnerian concepts as they relate to depression. According to this perspective, the central feature of most depressions is a reduced frequency of emission of positively reinforced behaviour. The depressed person is assumed to be deriving relatively little payoff for engaging in any variety of behaviours and, hence, these behaviours either drop out of the individual's repertoire, or fail to develop over time. Coincident with the reduction in positively reinforced behaviour is an increased frequency of occurrence of aversively motivated behaviour such as verbal complaints, crying, etc. These patterns of behaviour thought to be the major data characterizing a depressive repertoire can be related to several variables. Such variables include: (1) an increase in the quantity of behaviour required to produce a significant effect upon the environment (schedule of reinforcement); (2) the presence of aversive and conditioned aversive stimuli; (3) sudden changes in the environment such as the loss of a discriminative stimulus/conditioned reinforcer (as in the death of a spouse or loved one); (4) a disproportionate maintenance of the individual's repertoire by negative rather than positive reinforcement; (5) punishment; (6) physical organismic changes (such as those involved in ageing) which prevent the individual from engaging in previously
reinforced behaviour; and (7) a deficit in the learning of behaviours required in order to obtain positive reinforcement in important life-contexts (Ferster, 1966).

Whereas Ferster stressed the importance of a wide variety of behavioural processes in the etiology of depressive behaviours, Lazarus (1968, 1972) focused his attention particularly upon a generalized loss of reinforcers. Lazarus (1968) restricted his argument to cases in which verbal and life-history data suggest that the individual has undergone "stressful or other provoking emotional experiences" (i.e., the so-called "reactive" depression as opposed to the "endogenous" pattern which Lazarus accepts as physiologically determined). It is held that depression is best conceived as a state of "emotional inhibition" resulting from the presence of inadequate or insufficient reinforcers. Covert ideational phenomena such as the anticipation of a nonreinforcing state of affairs may have a similar effect to that of the material loss of reinforcers. The state of behavioural deprivation which results from an acute or chronic nonreinforcing state of affairs is assumed to produce a depressive behavioural pattern characterized as one of refractoriness to a wide variety of stimuli. The operant processes which are assumed to result in this state of refractoriness are hypothesized to produce this effect through an elevation of sensory thresholds (Lazarus, 1972).

While Lazarus' speculations regarding elevated sensory thresholds and behavioural refractoriness to stimulation have some intuitive appeal, empirical data relevant to his position are meagre and contradictory. Research on psychophysiological reactivity in depression, which might be taken as an index of the depressive's responsiveness to stimulation, has
resulted in inconsistent findings (Ban, Choi, Lehman, and Adamo, 1966; Protopopov, 1948 (reviewed in Astrup, 1962); Lewinsohn, Lobitz and Wilson, 1973). (This research will be reviewed in greater detail in a later section).

Further data from different kinds of research are also inconsistent with the hypothesis that the depressed person is characterized by having elevated sensory thresholds. If this were the case, it would seem reasonable to assume that such a change in sensory functioning would be expressed in impaired performance on certain cognitive-intellectual and concept formation tasks. Yet Friedman (1964) found that patients rated as being severely depressed showed little or no impairment on a variety of psychological tests. Nutter, Cruise, Spreng, Weckowicz and Yonge (1973) found no differences in performance between patients rated as highly and slightly depressed on the basis of the Beck Depression Inventory and a group of nondepressed normal control subjects on a concept formation task. A recent paper by Miller (1975) has taken issue with these and similar findings and has argued that a variety of "psychological deficits" have been observed in empirical research with depressed subjects. It is not clear, however, how most of the deficit studies reviewed by Miller relate to Lazarus' proposals.

Thus, it must be concluded that support for Lazarus' hypothesis relative to elevated sensory thresholds in depression remains, at best, equivocal.

Costello (1972a, 1972b) argued that while the processes emphasized by Ferster (1965, 1966) and Lazarus (1968, 1972) may produce depressive behaviour, these conditions can be related to a single, unitary process; a decrement in the effectiveness or reinforcers brought about through endogenous physiological factors (e.g., a decrease in brain norepinephrine)
or the disruption of a chain of behaviours. Pointing to evidence from factor analytic studies regarding the large loading of "loss of interest" items on factors of depression, Costello argues that the conditions identified by Ferster and Lazarus have insufficient heuristic power to explain the reported loss of interest in activities that have no clear relationship to the specific etiological factor under consideration. Thus, so the argument goes, "even in the case where a (depressed) person's behaviour was narrowly under the control of the person who is now dead, and where none of the learned instrumental behaviours are being elicited, there is no obvious reason why, for instance, the grieved person should have no appetite for food when it is freely presented" (Costello, 1972a, p. 242).

One mechanism through which this loss in reinforcer effectiveness may come about involves a decrease in the "attractiveness" of a reinforcer as a result of the removal of an antecedent CS or $S^D$. The absence of a well-established $S^D$ from a behavioural chain reduces the reinforcement effectiveness of all components of that chain. In this way, a variety of performances which may be components of an extended chain of behaviours, but which may not appear to have any obvious relationship to the specific loss suffered by the depressed individual, may be weakened.

In spite of the fairly elaborate theoretical background Costello used in formulating his position, certain aspects of his argument are not clear. The first question relates to whether or not the hypothesis that reinforcers lose their effectiveness for the depressed person is specific to those events and stimuli which have been major reinforcers for the depressed individual in the past. In other words, is this change a decrease in the effectiveness of reinforcers per se, or is it simply a weakening in
the control of the person's behaviour by stimuli which in the past have been salient reinforcers? There are some empirical data which bear upon this question. The studies reported by Loeb, Beck and their colleagues (Loeb, Beck, Feshbach and Wolf, 1964; Loeb, Beck, Diggory, and Tuthill, 1967; Loeb, Beck, and Diggory, 1971), discussed earlier in relation to Beck's theory, are of relevance here. These studies contrasted the effects of artificially controlled success and failure experiences on depressed and nondepressed subjects. Across all three studies, it was found that apparently successful performance produced higher self-ratings of optimism and level of aspiration, and greater increments in actual performance among depressed subjects than among nondepressed subjects. These findings suggest that, contrary to Costello's proposal, depressed individuals are more powerfully affected by positive reinforcement than are nondepressed subjects. One must be cautious about over-interpreting these findings, however, since the contrast groups in these studies were all drawn from psychiatric populations, and it may have been that these groups were simply less affected by positive reinforcement than were the subjects in the depressed groups.

Seligman (1972, 1973; cf. also Wolpe, 1971) has recently proposed a highly influential model for the etiology of depression derived from the study of "learned helplessness" in animals. In a series of aversive conditioning studies, various animal species (including man) have been subjected to experimental procedures involving the presentation of uncontrollable aversive stimuli (Maier, Seligman and Solomon, 1969; Seligman and Groves, 1970; Thornton and Jacobs, 1971), usually severe electrical shock. The typical behavioural effects of this type of treatment include:
diminished response initiation, a diminished response repertoire, an inability to learn adaptive responding when escape, avoidance or positive reinforcement become available (Overmier and Seligman, 1967; Seligman and Maier, 1967) and a virtual absence of pain-elicited aggression (Powell and Creer, 1969). Other effects include: anorexia and concomitant weight loss, whole brain norepinephrine depletion and an increased frequency of ulcer formation (Miller and Weiss, 1969; Weiss, 1968; Weiss, Stone and Harrell, 1970). Animals which had been provided with a prior history of mastery over aversive events, in the form of avoidance training, remained relatively immune to the effects of learned helplessness training (Seligman and Maier, 1967; Maier, 1970). The diverse phenomena produced by learned helplessness training are, at the same time, phenomena which are frequently noted to be associated with clinical reactive depression, and this similarity lends credence to the suggestions of Seligman (1972, 1973), Wolpe (1971) and others that similar processes may be operating, notwithstanding traditional criticisms of such "subject analogues" (Maher, 1966).

In one of the earliest papers outlining his position on the relationship between learned helplessness and depression, Seligman (1972, p. 241) interjected a word of caution, noting that "most of the evidence for (the learned helplessness model of) depression is largely anecdotal and selected experimental tests in man on the helplessness theory of depression are needed." Since that time, some research relating to the theory has become available. One such line of research has taken as its basis, the close conceptual similarity between learned helplessness and Rotter's (1966) concept of locus of control. According to Rotter, individuals vary in the extent to which they perceive reinforcement as being
under their own control (internal locus of control), or under the control of such factors as luck and circumstance (external locus of control). Similarly, the "helpless" individual has learned that responding and reinforcement are independent of each other. Thus, if learned helplessness and external control are empirically similar, and the learned helplessness model of depression is valid, depressed individuals should tend to score toward the external end of the internal-external locus of control dimension. A number of studies have attempted an empirical test of this hypothesis by investigating the correlation between locus of control scores and scores on various indices of depression. Miller (1971) reported a rank order correlation of .24 (p < .001) between the Beck Depression Inventory and Rotter's (1966) Internal-External scale, in a group of 201 undergraduates. Abramowitz (1969) reported a Spearman's rho of similar magnitude (ρ = .28, p < .05) between external locus of control and depression as measured by the Guilford D (depression) scale. Calhoun, Chaney and Dawes (1975) investigated the correlation between Rotter's Internal-External scale and Zung's (1965) Self-Rating Depression Scale, and Lubin's (1965) Depression Adjective Checklist, analysed separately for sex. Respondents were 81 undergraduates. Results revealed significant correlations between external locus of control and depression (as measured by the Zung scale) for both sexes (r = .58 for males, .38 for females), but only one significant correlation between locus of control and depression as measured by the Lubin scale (.50 for males, .09 for females).

Although significant, the magnitude of these correlations leave much to be desired, and must be considered to give rather weak support to
the learned helplessness model of depression. One source of this weak relationship between locus of control and depression could relate to the possibility that whereas the depressed individual may on the whole perceive an independence between what one does and its effect on the environment, there is also that component of the phenomenology of depression termed "guilt" which implies that the depressed individual perceives that at some time in his life, his behaviour has had adverse consequences, probably for some member of the social environment. Thus, while behaviour and its positive effects may seem independent, the individual may be overly sensitized to the adverse effects of his actions. To the extent that this is an accurate characterization, the notion that the depressed person has acquired a form of helplessness may be lacking in specificity. A further potential source of the relationships found in these studies relates to the wording of items on the various locus of control scales. The content of items scored in the "external" direction tends to be negatively-toned and socially undesirable. It would seem likely, therefore that depressed subjects would be relatively more likely to endorse "external" items than would nondepressed subjects.

In a more direct test of the learned helplessness model of depression, Miller and Seligman (1973) examined a series of predictions from the theory among four groups of college students differentiated into depressed and nondepressed groups according to their Beck Depression Inventory scores and internal and external locus of control groups according to their Rotter Internal-External scale scores. All subjects were exposed to two series of tasks. In the first (or chance) task, subjects were seated in front of a projection screen and asked to predict whether the
slide they were about to see would have an "X" or an "O" marked on it. The experimenter surreptitiously controlled which of the slides would actually be presented, thus controlling the subject's performance on the task. In the second (or skill) task, subjects were required to operate an apparatus consisting of a tilted wooden platform connected, through a pulley, to a string. Balanced on the platform was a steel bearing which was actually held in place on the platform by a concealed electromagnet, thus allowing the experimenter to control whether or not the bearing would fall off the platform. Subjects' task was to raise the platform, by pulling the string, to a predetermined level without letting the bearing fall. Ten trials on each task were given. Prior to each trial on both tasks, subjects were required to estimate on a 0-10 scale the probability that they would be successful in achieving the experimental goal. In order to control for the effects of reinforcement the same 50% monetary reinforcement schedule was used on both tasks.

A series of predictions from the learned helplessness model were tested. The following results were interpreted as being consistent with the model. Three measures of expectancy change revealed that nondepressed subjects showed greater expectancy changes than did depressed subjects on the skill task. Nondepressed subjects showed more change in expectancies for success on the skill task than on the chance task, whereas depressed subjects did not. In the skill task, expectancy changes were significantly and negatively correlated with Beck Depression Inventory scores, while in the chance task they were not. Thus, the learned helplessness model of depression received considerable support. At least two weaknesses which restrict the generalizability of this study are apparent. The first is
the failure to include a psychiatric control group against which to evaluate whether or not the differences obtained were specific to depressed individuals or whether they were more closely related to psychiatric status in general. The second drawback relates to the fact that the range of depression scores obtained from subjects in the study (0-21) was relatively small compared to the total possible range of scores on the Beck Depression Inventory (0-63). Thus, the amount of information gathered on the magnitude of effects as a function of severity of depression is restricted.

Lewinsohn and his colleagues at the University of Oregon have engaged in what is certainly the most prolific attempt to account for depression within a social learning context. Drawing from the earlier theoretical accounts of Ferster (1965, 1966) and the more recent proposals of Seligman (1972, 1973), Lewinsohn (1974a) suggests that the numerous behavioural patterns which highlight the depressive constellation are functionally related to a low rate of response-contingent positive reinforcement. The suggestion is that the particular reinforcement regimen that the individual is experiencing serves as an eliciting stimulus for various respondents which in turn have operant consequences by setting the occasion for depressive behaviours" (Lewinsohn, Weinstein and Shaw, 1969, p. 232). In addition to being elicited by situational events, depressive behaviours are often strengthened as a result of social reinforcement in the form of sympathy, concern and other more subtle payoffs. At the same time, the depressed person himself becomes an aversive stimulus to others, prompting avoidance on their part, thus further increasing his isolation and reducing the reinforcement available to him. A
non-reinforcing environment can occur through a number of processes such as a specific loss through the death of a significant other or accumulating misfortunes. Lewinsohn further suggests that another highly important antecedent of a low rate of positive reinforcement is a lack of social skill on the part of the depressed person. Lewinsohn's theoretical system is represented schematically in Figure 1.

It is apparent that, while social learning theorists have only recently begun to be concerned with developing a formulation of depression, there has been no dearth of speculation since the beginnings of this attempt. To what extent these formulations can be supported on empirical grounds is, of course, a major concern. Some data which bear on a selected aspect of the positions of Lazarus (1968, 1972) and Seligman (1972, 1973) have already been reviewed (cf. pp. 22 & 26). Yet, for the most part, support for behavioural theories of depression has been forthcoming on purely logical, rather than empirical grounds. If the depressed person is best characterized by a given behavioural constellation (e.g., a low rate of emission of behaviour), then it would seem to follow that the variables identified by Ferster, Lewinsohn and others are critical because of the fact that basic research dealing with similar response classes has shown them to be important in a laboratory setting. Perhaps the most important of these relates to the definition of behaviour and reinforcement. The key tenet of most of the behavioural approaches to depression is that the depressed person is characterized by a general reduction in the amount of behaviour he or she engages in. Yet even if it were possible to measure
Fig. 1. Schematic representations of Lewinsohn's model of the origins and maintenance of depressive behaviour (adapted from Lewinsohn, 1974a, p. 67).
the amount of behaviour engaged in by a person both prior to and during an episode of depression, it could still be pointed out that any living organism is continuously engaging in behaviour of some sort. A second difficulty in most behavioural theories of depression derives from the fact that most such theories propose that depression originates from a reduction in the amount of positive reinforcement impinging upon the individual. The difficulties with this proposal are both conceptual and practical. In order to determine whether a given individual has experienced a decrement in reinforcement, it is first necessary to identify what were previously the reinforcers for his behaviour. Yet the identification of reinforcers cannot be made independently of their effects on behaviour (Skinner, 1938). In addition, applied behaviour analysts typically note the subtlety of reinforcers and concomitant difficulty in identifying them. One partial solution to this problem has been to simply identify on an intuitive basis a sample of events and stimuli which may reasonably be thought to have reinforcing value, and assume that these items are a reasonably representative sample of a universe of reinforcers (Cautela and Kastenbaum, 1967; MacPhillamy and Lewinsohn, 1971). Even so, however, these samples may miss identifying important reinforcers on an idio- graphic basis. In the end, however, even granted that it were possible to identify all reinforcers for a particular individual, the possibility of observing the occurrence of all reinforcing events for that person at all times remains remote.

Fortunately, however, enough evidence consistent with the behavioural position on depression is available that the aforementioned issues do not loom large. The first such line of evidence comes from
factor-analytic studies of depressive symptomatology. Among the most powerful of factors obtained in this research are factors which load highly on items reflecting a decreased general activity level among depressed patients. Grinker et al. (1961) reported among their analysis of a "Current Behaviours" checklist, a factor reflecting isolation, withdrawal and apathy, and a factor reflecting a general retardation of behaviour and gait. A factor highly similar to the apathy factor of Grinker et al. was reported by Friedman, Cowitz, Cohen and Granick (1963) and loaded heavily on items reflecting retardation, apathy, loss of energy, withdrawal and isolation. Lorr, Sonn and Karz (1967) report the identification of a similar factor.

Other studies have focussed on noncontent aspects of verbal behaviour. Aronson and Weintraub (1967) compared the verbal productivity of depressed psychiatric patients with that of a group of normal army inductees and found that in 10 minute speech samples, depressed patients produced significantly less speech. Using the MMPI depression scale as an index of severity of depression it was also found that as depression decreased, verbal productivity approached normal, while as it increased, productivity diverged from that of normal subjects. Hinchcliffe, Lancashire and Roberts (1971a) and Truax (1971) report replications of these findings, the former using as subjects psychiatric patients with a primary diagnosis of depression, and the latter using schizophrenic patients varying in depression. One significant caution that must be pointed out in the interpretation of these findings is that none of these studies utilized a psychiatric control group in order to evaluate the extent to which verbal productivity differences are specific to depressive status. Williams,
Barlow and Agras (1972) developed a behavioural observation scale for the rating of severe depression. Items on the scale included such behaviours as talking, taking a shower, smiling, etc. Correlations between ratings of depression from this scale and from more traditional measures of depression such as Hamilton's (1960) Rating Scale for Depression, and Beck's Depression Inventory (Beck, Ward, Mendelson, Mock and Erbaugh, 1962) were .71 and .67 respectively. Analysis of ratings of depression as assessed by these measures and distributed over time revealed that as patients improved in depressive status, the number of discrete behaviours engaged in, increased. It was further noted that patients who were discharged while their behavioural ratings revealed a declining trend in the amount of behaviours engaged in were more likely to be re-admitted to hospital within a period of one year than were patients whose ratings revealed the opposite trend. The latter finding, however, is based on a small sample and cannot be regarded as anything more than suggestive.

Lewinsohn and Libet (1972) and Lewinsohn and Graf (1973) investigated the relationship between the extent to which a person engages in behaviour that is rewarding and depression as assessed by Lubin's (1965) Depression Adjective Checklist. Since a major assumption of Lewinsohn's approach to depression is that a low rate of positive reinforcement is a critical eliciting condition for depression, it was predicted that self-ratings of depression would be negatively correlated with the number of pleasant activities engaged in. In the first study (Lewinsohn and Libet, 1972), 30 college students were differentiated into a depressed group, a psychiatric control group and a normal control group according to strict psychometric and clinical criteria (MMPI profiles, scores on the
Grinker et al. (1961) Feelings and Concerns Checklist and ratings by interviewers). All subjects were administered the Pleasant Events Schedule (MacPhillamy and Lewinsohn, 1971) which consists of 320 events and activities that the subject rates according to how pleasant these activities are and how often they are engaged in. From the initial testing, an individual pleasant activities schedule was generated for each subject and consisted of the 160 items deemed by him or her to be the most pleasant of the entire schedule. For the next 30 days, subjects rated the extent to which they engaged in such pleasant activities and filled out the Lubin checklist. For the two dependent variables, five correlations, computed over days, were performed. The first was a measure of the relationship between pleasant activities and mood on the same day (0 displacement). In order to obtain evidence regarding the direction of causality, the daily ratings were displaced one and two days in either direction so that in one case the depression scale score preceded the pleasant activities score, while in the other case, the activities score preceded the depression score. In this manner it was found that same day ratings resulted in a relatively strong mean correlation between few pleasant activities and depressive mood (mean r was in the neighbourhood of -.40). The displaced correlations did not differ significantly from one another, thus making it impossible to infer the direction of causality. The results of this study, therefore, support one of the basic tenets of behavioural theories of depression — that there is an association between the extent to which a person engages in pleasant activities and mood — but fail to confirm a further implication of such theories — that changes in activity level temporally precede, and are causally related to changes in mood. In a later study, Lewinsohn and Graf (1974) attempted to
replicate these findings using a broader sample of subjects (including psychiatric patients) divided into three age ranges (18-29, 30-49 and 50 and over). Groups were differentiated according to the same criteria as used in the previous study. The previous findings of a strong negative correlation between pleasant activities and depressed mood was replicated. In addition, the magnitude of this correlation for depressed subjects was significantly greater than it was for psychiatric and normal control subjects. An analysis of the mean number of pleasant activities engaged in by the various groups revealed that depressed subjects reported engaging in significantly fewer pleasant activities than did the other groups. Assuming an identity between pleasant activities and reinforcement value, these results support the hypothesis that depression is accompanied by a low rate of positive reinforcement. Further analysis revealed that this difference was due to both a restricted range of pleasant activities on the part of the depressed patients, and the fact that depressed subjects engaged in fewer replications of pleasant activities than did subjects in the other groups.

Social skill and social communication deficits in depression.

One can conclude from the evidence reviewed thus far that a significant quantity of empirical data is consistent with many of the expectations and hypotheses of behavioural accounts of depression. Yet, if the depressed individual is so because he emits a low frequency of positively reinforced behaviours, certain questions still remain unanswered, viz: what variables may be expected to induce such a nonreinforcing state of affairs, and can certain organismic characteristics be identified which predispose an
individual to experiencing such circumstances? One manner of attempting to resolve these questions derives from the social-skill hypothesis originally outlined by Lewinsohn, Weinstein and Shaw (1969, cf. also Lewinsohn, 1974a, 1974b). This hypothesis states that the depressed person is characterized by a lack of social skill, defined as the inability to emit behaviours that are positively reinforced, and not to emit behaviours that are punished by others. Similarly, Ferster (1973) has suggested that the "basic behavioural process" underlying depressions may be a limited "repertoire of observation" which is to say that the depression-prone individual exhibits a relative or absolute low frequency of emission of behaviours which may be described as coming under the control of environmental discriminative stimuli. (Apparently Ferster saw no reason to distinguish between diminished responsiveness to social as opposed to non-social stimuli.) An analysis of these formulations suggests that the individual predisposed toward becoming depressed may exhibit deficits in two potentially distinguishable patterns of discriminative behaviors: 1) the ability to emit situationally appropriate behaviour which may be unambiguously identified and responded to by others, and 2) the ability to perceive or respond discriminatively to the situationally appropriate behaviour of others. Cast in a slightly different terminology, it might be suggested that the depressed person exhibits deficits in both expressive and receptive aspects of social communication.

A significant proportion of most people's daily lives is spent within a context of interpersonal interaction. Skinner (1953) has noted that a large bulk of the reinforcement that one obtains occurs in such situations in the form of social reinforcement. Given the importance of such everyday interpersonal interactions for the acquisition of reinforcement, it can be
assumed that most people acquire repertoires of social behaviour which facilitate these performances. One component of such a repertoire would include those behaviours which set the occasion for the reinforcing and punitive behaviours of others. Stated somewhat differently, it is suggested that the types of behaviour an individual emits in a given interpersonal situation cue the types of behaviour which are directed back toward that person. According to the formulation presented herein, the person who is predisposed toward becoming depressed may either be characterized by a low frequency of emission of such "social cueing" behaviours, or a relatively higher frequency of emission of behaviours which less adequately cue the modes of behaviour emitted by other people. As a result of this, the individual who exhibits such a deficit would frequently fail to acquire reinforcement. Further, he or she would run the risk of experiencing a relatively greater frequency of aversive behaviour directed toward him by other persons. This behavioural deficit on the part of the individual predisposed toward becoming depressed may have further untoward consequences in that he or she may serve as an ambiguous social stimulus prompting avoidance on the part of others. Aside from the obvious negative sequelae of being avoided by others, such a state of affairs may be further destructive in that the individual is then faced with a greater reduction of social reinforcement in addition to the loss of models for the emission of appropriate behaviour.

The second form of behavioural deficit which is proposed to characterize the depressive -- a relative inability to respond appropriately to the social communication of others -- represents a pattern complementary to that described above. Just as effective social communication requires
the emission of responses which control the social behaviour of others, it can also be assumed that a second component of this process includes a sensitivity to meaningful nuances of the behaviour of others. A significant proportion of the behaviour of others in interpersonal contexts consists of the emission of behaviours of various degrees of subtlety which serve as discriminative stimuli for the individual toward whom the behaviour is directed. An individual who is relatively unable to discern and effectively utilize such cues in the behaviour of others is unlikely to emit responses which adequately conform to the requirements of the situation. Such a person would fail to adequately reinforce the other person's behaviour and this, in all probability, would result in the emission of aversive, or at best nonreinforcing behaviour on the part of the other. It is herein hypothesized that such a deficit may underlie and be causally related to the occurrence of depression.

While empirical data directly relevant to these hypotheses are scanty, a number of converging lines of evidence attest to their plausibility. Studies reviewed in previous sections relating to patterns of verbal productivity provide findings consistent with, and predictable from the present formulation (Aronson and Winetraub, 1967; Hinchliffe, Lancashire, and Roberts, 1971; Truax, 1971). These studies reported that depressed patients could be significantly differentiated from normal controls in terms of low levels of total speech output generated in standardized situations. To the extent that depression is related to a social communication deficit of the type suggested by the present formulation it would be expected that depressed patients would show deviant patterns of verbal behaviour.

Other studies of the verbal interaction patterns of depressed
subjects provide data consistent with the theoretical position outlined above. Rosenberry, Weiss and Lewinsohn (1969) reported that depressed subjects responded less predictably and homogeneously than nondepressed subjects when asked to indicate, while listening to tape recorded speeches, when they would behave in such a manner as to maintain rapport with the speaker. Lewinsohn, Golding, Johannson, and Stewart (1968) required pairs of depressed and normal subjects to communicate with each other via typewriters. While normal-normal subject pairs tended to increase the number of words typed per message, pairs of depressed subjects showed this tendency to a lesser extent. This difference, however, was only marginally significant (p < .10). Utilizing a highly complex system for the recording of verbal interaction (Lewinsohn, Alper, Johannson, Libet, Schaeffer, Rosenberry, Sterin, Stewart and Weinstein, 1971), a number of differences have been found between depressed and nondepressed subjects with respect to the kind and quantity of verbal behaviours observed in small group settings. Within such a context, depressed individuals tend to elicit fewer verbal behaviours from other people than do subjects belonging to psychometrically-defined normal and psychiatric-control groups (Lewinsohn, Weinstein, and Alper, 1970; Schaeffer and Lewinsohn, 1971; Libet and Lewinsohn, 1973; Libet, Javorek, and Lewinsohn, 1973). Other deficits observed among depressed subjects in these studies include engaging in fewer behaviours, emitting fewer positive reactions toward others, and evidencing a longer latency of reaction to others' behaviour than do nondepressed persons (Libet and Lewinsohn, 1973). Taken together, these findings strengthen the position outlined above in that they report patterns of behavioural deficits representing both receptive and expressive aspects of the social communication process among depressed
subjects. The generalizability of these findings remains questionable, however, due to the fact that all subjects in these studies were college students.

Another area of research pertaining to the present formulation consists of the study of various forms of "expressive" behaviour, commonly subsumed under the label "nonverbal communications". Such overt expressive behaviours as gestures, changes in facial expression, eye contact, etc., have in recent years been the subject of considerable inquiry (cf. Ekman and Friesen, 1968) although only a few investigations in this general area have employed depressed subjects. It is evident that these various forms of behaviour may be salient discriminative stimuli for other people in the social environment and, to a large extent, may be important in controlling the dispensation of reinforcement on the part of other people. Waxer (1974) showed sound-edited videotapes of the standardized intake interviews of 5 depressed and 5 nondepressed psychiatric patients to 67 psychology faculty, graduates and undergraduate students, asking them to identify, on the basis of nonverbal cues alone, the diagnostic group membership of each of the people observed. Results of this study revealed that judges were able to correctly discriminate depressed subjects from nondepressed subjects at a level beyond the .0005 level of significance. Thus, it is apparent that

1. The use of this terminology introduces some potential confusion with respect to definitions and differentiations of various components of the social communication process. This can be highlighted by considering Skinner's (1957) definitions of verbal behaviour, viz.: any behaviour reinforced through the mediation of other people (p. 2). In terms of this definition, most of what is commonly referred to as "nonverbal communication" may, in all probability, be "verbal" but "nonvocal". For the purposes of the present discussion, however, the more common distinction -- nonverbal -- will be retained.
there is something about the nonverbal behaviour of depressed people which is salient enough to allow other people to discriminate them from other people having different diagnostic labels. According to questionnaire data acquired from raters, some specific behaviours which differentiated depressed subjects from other subjects included decreased eye contact, and postural cues. Hinchcliffe, Lancashire and Roberts (1971b) compared depressed subjects with subjects whose depressive episodes had undergone remission on total duration of eye contact, eye contact while speaking, and total number of eye contacts with an interviewer during a 5 minute interview. Analyses revealed significantly lower scores on all three variables for the presently depressed group. While it is unclear how these behaviours may be functionally related to the manner in which other people behave toward the depressed person, it is possible that they may affect others' reactions in a manner consistent with the above hypothesis. Ekman and Friesen (1974) contrasted the nonverbal behaviour in interview situations of 31 female depressives 9 of whom were diagnosed as psychotic depressives, 7 of whom were neurotic depressives and 15 of whom carried the diagnosis of schizophrenia. The activity of subjects' hands during the interviews was classified into two groups of movements: illustrators (which "seem to illustrate what is being said verbally" (p. 211)) and adaptors (which seem to be less directly related to a process of embellishing the flow of speech and are characterized by such activities as picking, rubbing, or squeezing). It was found that psychotic depressives engage in significantly less "illustration" than do schizophrenics. A significant trend in the same direction was found in the neurotic depressive group. Although an increase in illustrator activity was found between admission and discharge among the depressed groups, it is not reported
whether this increase was of a large enough magnitude to eliminate differences between these subjects and those in the schizophrenic group. These subjects were also given ratings on the Brief Psychiatric Rating Scale (BPRS, Overall and Gorham, 1962) and correlational analyses were carried out between illustrator activity and the various scales of the BPRS. The most important findings for the purposes of the present discussion were significant negative correlations between illustrator activity and the scales depressive mood (r = -.51 at admission, -.39 at discharge) and motor retardation (r = -.66 at admission, -.36 at discharge).

The studies reviewed this far provide some evidence for the existence, among depressed subjects, of deviant patterns in expressive aspects of social communication. Clearly, both the quantity and the quality of this research can be brought to bear on questions regarding the presence, extent and nature of any deficits in receptive aspects of the social communication process among depressives. While the notion that the depressed patient is characterized by a relative insensitivity to the interpersonal communications of others is not unheard of in the literature (Cohen et al., 1954; Stuart, 1967) no studies have attempted to test this hypothesis directly, and what evidence there is relative to this issue is only suggestive. Shannon (1970) compared the performance of a depressed, a schizophrenic and a medical patient control group on the Brief Affect Recognition Test (BART; Ekman and Friesen, 1974). This test uses the tachistoscopic presentation of still pictures of people portraying the facial expressions of 6 different emotions (happiness, sadness, anger, etc.). Subjects are simply required to indicate which emotion is being portrayed. Performance of the depressed group was significantly impaired relative to the medical
control group, but not to the schizophrenic group in the recognition of "anger"; and was significantly impaired relative to both groups in the recognition of "fear".

A methodology for more adequately assessing the hypothesis that depression may be related to deficits in the emission of and response to social communication cues is suggested by research on social communication among nonhuman primates which has recently been extended to the study of "affective recognition" in man. The basic paradigm under consideration here was originally developed to investigate factors thought to be related to the grossly deviant social behaviour seen in adult rhesus monkeys who had been subjected to social isolation treatments in earlier life. Brief reviews of the many deficiencies in social interaction observed in such animals are available in Harlow (1962) and Mason (1960). In attempting to account for these phenomena, Mason (1960) proposed a theory strikingly similar to the formulation of depression advocated here. Briefly, Mason hypothesized that the pervasive social deficits observed in rhesus monkeys subjected to early isolation experiences result from a deficit in the ability to emit cues controlling reinforcement from the social environment and a consequent inability to learn appropriate behaviours. A test of this hypothesis was conducted by Miller, Caul and Mirsky (1967; cf. also Miller, 1967; Mirsky, 1968). A group of rhesus monkeys which had been raised in total social isolation for their first year of life, and a group of feral rhesus monkeys of the same age were initially tested and compared on an avoidance task. No differences in ability to acquire the avoidance response were found between the two groups. Subsequent to this initial testing, the same subjects were tested on a co-operative avoidance task
in which a "sender" monkey was administered electric shock on a Sidman schedule while a "receiver" monkey controlled the manipulandum which could be used in order to avoid shock for the other monkey. The sender monkey's face was broadcast to the receiver monkey via a closed circuit television screen. A 6 second CS was presented to the sender monkey immediately prior to the administration of each shock. If, during this period, the receiver monkey operated the manipulandum, shock could be avoided. All possible pairs of feral and isolate monkeys were tested as both senders and receivers. The results of this study showed that while feral-feral pairs were able to acquire adequate avoidance responding, neither isolate-isolate nor feral-isolate pairs were effective. In the latter case, avoidance responding was poor regardless of whether isolate or feral monkeys performed as senders of receivers. The authors note that these findings indicate an inability on the part of the isolate monkeys to emit facial cues facilitating the acquisition of an avoidance response by the feral monkeys as well as a further inability to make use of the facial cues of feral monkeys in order to acquire an avoidance response themselves. The relationship between these patterns of behaviour and those hypothesized to characterize the depressive in the present formulation should be apparent.

The basic paradigm outlined by Miller, Caul and Mirsky (1967) has been adapted to research on human subjects by several authors. The basic concept in studies of this nature has been one of determining whether it is possible for human subjects to recognize facial cues which differentiate between varying experimental conditions. Gubar (1966) assigned university students to groups of 3 subjects each. One subject in each group was designated the "experienced" observer. Upon his arrival at the laboratory,
the "experienced" observer participated in a discrimination task. This task required the acquisition of two operant responses — one negatively reinforced by shock avoidance, the other positively reinforced by a click on a counter. A buzzer and a bell presented 6 seconds prior to either shock or reward served as discriminative stimuli. After this subject had reached criterion performance on the task, a second subject was then placed in the same experimental situation. Once this subject had attained criterion performance, the experienced observer was taken to an adjoining room from which he could observe the other subject performing the experimental task. Only the subject's neck and head were visible to the observer. The observer was then instructed that the subject he was viewing was performing the same experimental task. A shock electrode was then attached to the observer and he performed the same task as before (i.e., operant button pressing responses) relying on the subject's facial expressions as discriminative stimuli for his responses. The same contingencies were operative for the observer and the subject. After 25 trials, the experienced observer was replaced by a naive observer who had the task explained to him and who performed it for 25 further trials. Analyses of correct and incorrect responses revealed that, while naive observers were unable to discriminate between shock and reward trials, experienced observers were successful at correctly discriminating shock and reward trials (p < .01).

Lanzetta and Kleck (1970) conducted a somewhat different adaptation of the Miller et al. paradigm. Subjects participated in 3 experimental sessions, during the first of which they were exposed to a discriminative classical conditioning procedure. A red light served as CS+ and was sequenced 12 seconds later by administration of electric shock while a
green light served as CS-. Thirty conditioning trials were administered during which videotapes were made of each subject's facial area. In sessions 2 and 3, subjects returned and viewed the videotapes of six subjects participating in the first session of the experiment. In these sessions, subjects were required to determine by observing changes in facial expression whether the subject that they were observing was viewing a red or a green light. The first and last 10 trials of the 30 that each subject experienced were shown. Incorrect guesses during the second and third sessions were consequated with electric shock. Results revealed that the subjects' accuracy of judgement was well beyond that which would be expected by chance ($p < .001$). Some further interesting relationships noted in this study were a strong negative correlation between an individual subject's judgemental accuracy and the accuracy with which others were able to judge that subject ($r = -.80$), and significant positive correlations between the degree of electrodermal arousal for each subject in the first session and the number of errors made in judging that subject in the following sessions. The former finding indicated that subjects who made few mistakes in judging others were themselves difficult to judge, while the latter finding indicated that subjects who displayed greatest electrodermal responsiveness in the first session were those who were most difficult to judge for others in the following sessions.

Yet a third variation on the basic paradigm of Miller et al. has been explored by Buck, Savin, Miller, and Caul (Buck, Savin, Miller, and Caul, 1972; Buck, Miller, and Caul, 1974). In the 1972 study, subjects were run in pairs in a single session. Subjects arrived independently at the laboratory, the first one to arrive being designated as the "sender"
subject, and the second to arrive being designated as the "receiver". Sender subjects were led to an experimental room, and seated, while receiver subjects were taken to a second room. Both subjects had physiological recording electrodes for heart rate and skin resistance attached. Sender subjects were shown a series of 25 slide photographs depicting 5 content categories (sexual, scenic, maternal, disgusting and unusual). After observing each slide for 10 seconds, these subjects were given 20 seconds to verbalize their emotional response to the slide. At the end of this verbalization period, sender subjects then rated the strength and pleasantness of their emotional reactions to each slide on 9-point scales. At the same time, receiver subjects observed a television monitor over which was broadcast a picture of the sender subject's facial area. Receiver subjects were required, at the end of each of the sender subject's 30 seconds observation-verbalization intervals, to make judgements regarding which category of slide the sender subject was viewing and to rate the slides on the same 9-point scale. It was found that accuracy of communication attained levels significantly above that which would be expected by chance among female pairs, but not among male pairs. Significant positive correlations were found between sender subjects' reactions for both male and female pairs and a significant positive correlation on ratings of the strength of sender subject's reactions was found for female pairs. Further, significant negative correlations were found between sender subjects' pleasantness index ratings and change in sender subjects' number of skin conductance responses from the preslide to the slide period ($r = -0.91$); median size of largest skin conductance response during the slide period ($r = -0.65$) and heart-rate change from the preslide period to
the first description period ($r = -.60$), thus in part replicating the physiological findings of Lanzetta and Kleck (1970). A more recent study (Buck, Miller, and Caul, 1974) utilizing the same methodology demonstrated statistically significant communication for both males and females, although communication accuracy was greater for the latter. The previous physiological findings were replicated as well.

The present study employed a modification of the procedure devised by Lanzetta and Kleck (1970) in order to test the hypothesis that depressed subjects exhibit deficits in the emission of nonverbal social communication cues and in the perception of such cues emitted by others. Three groups of subjects -- depressed, nondepressed psychiatric controls, and normal control -- participated in a two-phase experimental procedure. In session I subjects' facial expressions were videotaped while they underwent a tri-partite differential classical conditioning procedure. In session II, subjects returned and were shown the videotapes of subjects from all three diagnostic groups recorded in session I. The task in session II involved identifying which of 3 types of conditioning trial the subject they were viewing was undergoing.

**Psychophysiological activity in depression**

A secondary purpose of the present study was to collect information regarding patterns of autonomic activity in depression. Over the years, a number of investigators have concerned themselves with studying various psychophysiological parameters in an attempt to characterize the psycho-physiological "state" of depression. Among the physiological parameters whose functioning has been related to depression are: skin
resistance/conductance (Dawson, Schell and Catania, 1974; Ban, Choi, Lehman and Adano, 1966; Goldstein, 1965; Greenfield, Katz, Alexander and Roessler, 1963; Lader and Wing, 1966; Lewinsohn, Lobitz and Wilson, 1973; McCarron, 1973; Riazansky, 1965; Zuckerman, Persky and Curtis, 1963), electromyographic activity (Goldstein, 1965; Martin, 1956; Noble and Lader, 1971; Rimon, Stenback and Hahmer, 1966; Shipman, Oken, Goldstein, Grinker, and Heath, 1964; Whatmore and Ellis, 1959, 1962), cardiovascular activity (Dawson et al., 1974; Kelly and Walter, 1968; McCarron, 1973), electroencephalographic activity (Shagass, 1955a, 1955b; McCarron, 1973), gastrointestinal activity (Henry, 1930; Kehoe and Ironside, 1963) and salivation (Busfield and Wechsler, 1961). Methodologies employed in this research have been diverse and include studies of tonic activity, phasic response to various forms of stimulation, and classical conditioning. Although a considerable quantity of research has been conducted from this orientation, few generalizations are possible from the data, largely as a result of the "one shot" nature of many such studies, in addition to the presence of empirical inconsistencies and methodological limitations.

Probably the most consistent findings in such studies have been acquired in studies of musculoskeletal parameters. Martin (1956) reported that electromyographic (EMG) recordings from the frontalis and forearm extensors revealed heightened muscle tension among a group of dysthyms (anxious, depressed or obsessional patients) than among a group of hysterics. Whatmore and Ellis (1959) reported that resting EMG's were significantly higher among a group of depressives than among a group of controls, and that the degree of this heightened activity was correlated with the extent of psychomotor retardation among the depressives. A
later study (Whatmore and Ellis, 1962) reported a significant decrease in EMG activity among extremely retarded depressive subsequent to a course of psychotherapy. Later evaluations during a period of "good health" prior to relapse showed that EMG levels increased significantly. This finding prompted the conclusion on the authors' part that heightened EMG activity is a "physiological trait" of depression-prone individuals. In a study comparing a group of depressives with a group of nondepressed psychiatric patients and a group of normals, Goldstein (1965) recorded a number of physiological variables, including EMG activity taken from 7 different sites. The experimental paradigm involved one period of exposure to white noise interpolated between periods of rest. In comparison to the nondepressed and normal groups, depressives manifested heightened EMG activity as recorded from trapezius and frontalis sites. Noble and Lader (1971) studied changes in resting EMG (recorded from the left forearm extensor muscle) in a group of depressed patients prior to and following a course of electroconvulsive therapy (ECT). Pre-post comparisons revealed heightened activity prior to ECT. In sum, these findings suggest increased EMG activity among depressed patients, a finding inconsistent with the notion that depression is characterized as a state of physiological unresponsiveness. Proponents of the latter position have tended to look toward studies utilizing electrodermal measures for support.

Studies utilizing electrodermal indices of arousal have tended to support the position that depression is characterized by decreased physiological responsiveness, although the evidence here is contradictory. Greenfield, Katz, Alexander and Roessler (1963) exposed 40 subjects to a series of auditory stimuli ranging from subthreshold to 120 db while
recording skin conductance (SC). (In keeping with the present terminological convention, all studies of electrodermal phenomena reviewed in this section will be described in terms of skin conductance activity, regardless of whether the original data were expressed in terms of skin resistance as was the case with the Greenfield et al. study.) The bottom and top quartile of responders were then taken, and the scores of these subjects on the MMPI D scale were compared. It was found that low SC responders scored significantly higher on this index of depression that did high responders. It seems curious that while these investigators were presumably interested in acquiring correlational data they chose the methodologically questionable tactic of choosing comparison groups from the extremes of the distribution of scores and performing a t-test rather than utilizing all subjects' scores and simply computing a correlational coefficient. McCarron (1973) compared the SC activity of a group of depressed subjects and a group of normals. Groups were differentiated according to MMPI profiles. Subjects were given an initial task of reading and delivering the contents of an envelope. After this, they were attached to the recording equipment and, after a rest period, required to answer yes or no to 12 questions related to the contents of the envelope. Results indicated reduced lability of skin conductance responses for the depressed group, particularly during the interrogation procedure. Riazansky (1965) has also reported similar findings.

Other studies of electrodermal activity in depression have compared groups of depressives with various control groups in classical conditioning paradigms. Ban, Choi, Lehman and Adamo (1966) compared orienting responses (OR), unconditional responses (UCR), simple conditioning and
differential conditioning across groups of normal controls, "neurotic" depressives, "endogenous" depressives and schizophrenics with depressive symptomatology. Conditional stimuli consisted of yellow or green lights while the unconditional stimulus consisted of a 900 Hz tone presented at "maximal intensity". OR amplitude was significantly greater for the normal group than for the patient groups as was UCR amplitude. Analysis of conditioning data revealed that the normal group exhibited a greater number of conditional anticipatory skin conductance responses than did the patient groups. The normal group also evidenced greater differentiation of skin conductance responding during the differential conditioning stage of the experiment, exhibiting fewer responses to the unreinforced conditional stimuli (CS) and more responses to the reinforced CS than did the depressive groups. Unfortunately, a number of deficiencies in methodology and reporting militate against uncritical acceptance of these results. The authors failed to provide any data supporting the breakdown of groups into different diagnostic categories independent of clinical judgement. It is also unclear whether the differences obtained are specific to depression or a more general correlate of psychiatric status. This criticism can be applied equally to all studies which fail to include a group of psychiatric patients who are not depressed. Furthermore, actual levels of electrodermal activity summarized for the different groups and stages of the conditioning process were not reported, thus making it impossible to evaluate the actual magnitude of the differences obtained. Dawson, Schell and Catania (1974) compared the skin conductance values of a group of depressed patients and a group of normal controls across 5 experimental tasks: 1) rest, 2) free association, 3) adaptation, 4) conditioning and
5) reaction time. Evaluations of depressed patients were made at two points in time: 1) prior to undergoing ECT, and 2) subsequent to ECT. Analyses for pre-ECT measures indicated that depressives exhibited lower UCR magnitudes and longer UCR latency. Additionally, control subjects exhibited significant OR and anticipatory response (AR) conditioning while depressives did not. These results were consistent with those reported by Ban et al. (1966). A study conducted by Lewinsohn, Lobitz, and Wilson (1973) however, reported somewhat contradictory findings. In two studies, Lewinsohn et al. compared the SC response amplitude of three groups of college students differentiated according to strict psychometric criteria into depressed, psychiatric control and normal control categories. The experimental design followed what was basically a nondifferential classical conditioning procedure with electric shock as the UCS. Results revealed that the three groups did not differ significantly with respect to conditional AR activity, or with respect to post shock magnitudes of electrodermal activity. It was found, however, that during the period corresponding to that of shock administration, the depressed group showed a larger increase in SC than did the other two groups; a finding contradicting those of Ban et al. (1966) and of Dawson et al. (1974). The authors suggested that depressed people tend to be more sensitive to aversive stimulation than normals and individuals exhibiting psychiatric difficulties other than depression. It is difficult to account for these discrepant findings, although it is possible that sample and stimulus differences between the various studies may have been contributory factors.

Research on cardiovascular response as it relates to depression is sparse. Three studies show close agreement, however, in finding that
depressed patients show elevated tonic heart rate (HR) levels in comparison with normal controls (Kelly and Walter, 1968; McCarron, 1973; Dawson et al., 1974). Furthermore, Dawson et al. (1974) found that depressed patients show less HR change to different experimental procedures and in a classical conditioning paradigm than do normals.

Owing to the nature of the experimental paradigm used in order to test the major hypotheses of this study, it was possible to collect concurrent information pertinent to the issue of autonomic activity in depression. The physiological parameters of interest in this study were skin conductance (SC) and heart rate (HR). A tri-partite differential conditioning procedure similar to that employed by Hare and Quinn (1971) in a study of physiological response among psychopaths was employed. The use of HR and SC was dictated by a number of considerations. SC data are widely used in psychophysiological research and provide an index of autonomic activity which is highly sensitive to experimental treatments (although their exact meaning remains obscure (Edelberg, 1972)). Furthermore, SC data have been widely used as dependent variables in the study of classical conditioning processes in general (Prokasy, 1965) and as they relate to depression. Unfortunately, the use of electrodermal measures in psychophysiological research has been so widespread and interpretations of such activity have been made so glibly that their use as a sole criterion of such concepts as arousal, responsiveness and conditionability is open to serious question. In the area of depression specifically, several studies using electrodermal data have used their findings to argue that depressives tend to be either under-responsive or over-responsive to external stimulation, or that depressives may be characterized as being under-
aroused or over-aroused, etc. The use of a single poorly understood physiological index as the basis for making inferences regarding overall "arousal" is clearly questionable especially in the light of the low intercorrelations between alternative indices of arousal (Lacey, 1967). The use of HR data was dictated by the desire to include a second index of physiological activity as well as a number of other considerations. Cardiovascular activity is being increasingly assessed in contemporary psychophysiological studies, especially within classical conditioning paradigms. Furthermore, some research is available on cardiovascular activity in depression and the present design allowed for an attempt at replication of these findings. Most importantly, however, it has become clear in recent years that cardiovascular activity shows some interesting differential patterns in response to various experimental manipulations and that such changes as have been found to occur may be interpreted meaningfully in a psychological sense (Gunn, Wolf, Block and Person, 1972; Lacey, 1967).

Accordingly, in the present study, HR and SC were recorded within the context of a tri-partite classical conditioning procedure in the first session of the experiment.
Chapter III

METHOD

Overview

Three groups of subjects (depressed, psychiatric control and normal control) participated in the two sessions of the experiment. In session 1, which was designed to elicit various types of discriminable facial expressive behaviour, subjects were exposed to a tripartite differential classical conditioning procedure. The three aspects of this procedure included the presentation of an aversive CS-UCS sequence, the presentation of a CS-UCS sequence designed to elicit "pleasant" responses, and a neutral trial. Subjects' facial expressions were recorded on videotape and their HR and SC activity was recorded on a polygraph during the initial session. During the second session of the experiment subjects were shown the videotape taken in session 1 of three other subjects, one from each diagnostic category. In this second session, subjects were required to view each videotape and guess on the basis of changes in the facial expressions of the subjects observed, which of the 3 types of conditioning trial the observed subject was experiencing. In addition, subjects were required to estimate their anticipated performance immediately prior to observing each videotaped set of trials from each subject.

Subjects

Subjects were 30 females classified into depressed (D), psychiatric control (PC), and normal control (NC) groups. Prior to participating in the first session of the experiment, all subjects completed a battery of 3 psychological tests, including: 1) the Minnesota Multiphasic Personality Inventory (MMPI), 2) the Beck Depression Inventory (BDI; Beck et al., 1961),
and 3) a scale developed in order to assess the self-monitoring of expressive
behaviour (SM; Snyder, 1975). All psychiatric subjects were patients re-
gistered at the Health Sciences Centre Hospital at the University of Bri-
tish Columbia. Subjects included in the D group met the following criteria:
1) clinical diagnosis belonging to one of the DSM-II categories of depres-
sion, 2) no history of ECT in the year prior to participation in the study,
3) BDI scores of 20 or greater. BDI cut-off criteria were decided upon in
relation to the values reported by Beck et al. (1961) in their validation
studies. According to this report, a BDI score of 21 reliably discrimina-
ted between nondepressed and severely depressed subjects whereas a score of
17 reliably discriminated nondepressed from moderately depressed subjects.
Eight D group subjects were inpatients, 2 were outpatients. Subjects in-
cluded in the PC group were also registered patients at Health Sciences
Centre, and met the following criteria: 1) presently receiving treatment
for a psychiatric disorder other than depression, 2) no history of ECT in the
year prior to participation in the study, 3) BDI scores of less than 20. Seven
of these patients were outpatients, while 3 were inpatients. Subjects in-
cluded in the NC group met the following criteria: 1) no prior history of
hospitalization for any psychiatric problem, 2) not presently taking any
psychoactive medication, 3) BDI scores of 12 or less, 4) no MMPI scales
elevated above a T-score of 70. None of these subjects was receiving psy-
chiatric treatment at the time of participation in the study. Subjects in-
cluded in this group were acquired through two means: 1) hospital staff and
2) subjects recruited by acquaintances of the experimenter. A final condi-
tion for participation in the experiment was willingness to take part in a
second session.

Three subjects (2 in group D, 1 in group PC) indicated their un-
willingness to continue during session 1 and were replaced.

Inpatient subjects were all obtained from the same 25-bed acute care ward of the hospital. Potential subjects were sequential admissions to the ward who met the above criteria for inclusion in the D or PC group. In addition to the consent of the patient, it was necessary to acquire consent from the patient's primary therapist(s). The ultimate effect of this was to eliminate those potential subjects who were undergoing acute "crisis" situations, or who were deemed significant suicide risks.

Outpatient subjects were patients attending the hospital's Behaviour Therapy Clinic, and were recruited through their primary therapists.

No attempt was made to control patients' medication regimens. Descriptive data on all subjects are presented in Table 1.

The NC, PC and D groups had mean ages of 38.4 (S.D. = 12.05), 27.9 (S.D. = 5.40) and 38.6 (S.D. = 7.20) years respectively. Comparison of the three groups by analysis of variance showed a significant age difference (F(2, 27) = 4.97, p < .05). This significant F value is primarily accounted for by the lower mean age of group PC in comparison with groups NC and D.

Mean scores on the BDI were as follows: NC = 2.4 (S.D. = 3.84), PC = 10.2 (S.D. = 6.01), D = 32.1 (S.D. = 9.23). Analysis of variance performed on these data revealed, not surprisingly, a significant difference (F(2, 27) = 52.26, p < .01). Multiple comparisons by the Neuman-Keuls technique showed that all 3 groups differed from one another at the p < .05 level.

Group profiles on the nine regular clinical scales of the MMPI were compared using a technique described by Greenhouse and Geisser (1959). This procedure, similar to multivariate analysis of variance, separates
Table 1. Age, marital status, hospitalization status, primary diagnoses and medication regimens of all subjects in the study.

<table>
<thead>
<tr>
<th>Group</th>
<th>Subject Number</th>
<th>Age</th>
<th>Marital Status</th>
<th>Inpatient/Outpatient</th>
<th>Primary Diagnosis</th>
<th>Medication Regimen</th>
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<tbody>
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<td>Anxiety neurosis</td>
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</tr>
<tr>
<td></td>
<td>2</td>
<td>34</td>
<td>M</td>
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<tr>
<td>Group</td>
<td>Subject Number</td>
<td>Age</td>
<td>Marital Status</td>
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<td>Primary diagnosis</td>
<td>Medication</td>
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<td>45</td>
<td>M</td>
<td>Inpatient</td>
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<td>40</td>
<td>M</td>
<td>Inpatient</td>
<td>Depressive neurosis</td>
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<td>S</td>
<td>Outpatient</td>
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<td>S</td>
<td>Inpatient</td>
<td>Depressive neurosis</td>
<td>Valium (5 mg. q.i.d.) Chloral Hydrate (1 g, h.s.)</td>
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</table>
Table 1. (continued)

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<th>Age</th>
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<th>Inpatient/Outpatient</th>
<th>Primary diagnosis</th>
<th>Medication regimen</th>
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<td>29</td>
<td>35</td>
<td>M</td>
<td>Inpatient</td>
<td>Endogenous depression</td>
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<td>30</td>
<td>39</td>
<td>M</td>
<td>Inpatient</td>
<td>Bipolar manic depressive illness</td>
<td>Lithium carbonate (600 mg., b.i.d., 300 mg., h.s.)</td>
</tr>
</tbody>
</table>

a NC = normal control, PC = psychiatric control, D = depressive

b M = married, S = single, D/S = divorced or separated

c Primary diagnoses were taken from clinical files and in some cases may not correspond to specific DSM-II categories.

d Medications expressed in terms of quantity and schedule of administration on the date of each subject's participation in session 1; h.s. = at bedtime, b.i.d. = twice a day, t.i.d. = three times a day, q.i.d. = four times a day.
components of variance into that attributable to differences between tests, groups, and the interaction between tests and groups. The latter term, if significant, is interpretable as reflecting differences in test profiles between groups. The results of this analysis (Table 2) revealed no reliable differences attributable to tests or the tests X groups interaction. Thus, the profiles of test differences were similar for all three groups. A tendency toward significance was found for the groups factor \((F (27,2) = 12.40, p < .10)\) although conventional standards of statistical reliability were not obtained. This occurred despite the fact that one-way analyses of variance on each of the MMPI scales revealed several significant differences. In particular, these analyses suggested that on scales Hy, Pd, Pt, and Sc, subjects in groups PC and D exhibited significantly elevated scores relative to subjects in group NC. On the MMPI D-scale, the univariate analyses further suggested that subjects in group D exhibited significantly elevated scores relative to subjects in group PC, who in turn exhibited elevated scores relative to subjects in group NC.

Average MMPI profiles for the three groups are presented in Fig. 2.

Apparatus

The experiment was conducted in a room within the Health Sciences Centre complex. Subjects sat in a comfortable, reclining chair. Located approximately 2 meters in front of the subjects was a table, to the front of which was attached a 55 cm x 31 cm sheet of black cardboard on which were mounted 4 jewel lights coloured blue, red, amber and green. Each of these lights was approximately 7 mm in diameter. The spatial
Table 2. Results of the multivariate analysis of MMPI profiles (for technique, see Greenhouse and Geisser, 1959).

<table>
<thead>
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<th>F</th>
<th>p</th>
</tr>
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<td>n.s.</td>
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<td>12.40</td>
<td>&lt;.10</td>
</tr>
<tr>
<td>Individuals (within groups)</td>
<td>2,265.53</td>
<td>27</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Groups X Tests</td>
<td>1,864.98*</td>
<td>16</td>
<td>0.03</td>
<td>n.s.</td>
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<tr>
<td>Individuals (within groups) X Tests</td>
<td>792,295.30</td>
<td>216</td>
<td></td>
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</tr>
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</table>
Fig. 2. Average group profiles on the MMPI. NC = normal control, PC = psychiatric control, D = depressive.
arrangement of the lights was such that one light was located in the centre of the board, approximately 10 cm from the top. The other three lights were located 10 cm below the top light with one light being centered in the board, and the other lights placed 15 cm on either side. With this arrangement, the top light was located approximately at subjects' eye level. Located immediately above and behind the light board, and slightly to the right of the subject was a projection screen. Slightly behind and to the left of the light board was located a Sony AV 3400 videotape camera. The lens of the camera was located approximately 1 meter from the floor so that the angle from which subjects were videotaped was slightly below eye level, an angle which, from pilot testing, produced the highest quality recordings. Immediately in front of the camera's lens, was located an electronic shutter device which opened and closed with the warning stimulus — conditional stimulus — unconditional stimulus sequence. Opening and closing of the shutter was accompanied by an audible "whirring" sound as the internal motor was activated. Located immediately to the left of the subject, on a table, was a speaker (KLH Corp. Model-22 Acoustic Suspension) over which could be presented a loud (106 decibel) noise. The dimensions of the speaker were 45 cm high x 25 cm wide x 20 cm deep. The distance between the speaker and the subject's left ear when seated was approximately 15 cm.

Approximately 1.5 meters behind and to the right of the subject was located a stand on which the stimulus programming equipment was placed. Four Hunter decade interval timers controlled the presentation of stimuli, opening and closing of the shutter apparatus, and event marker pen on the polygraph.
Unconditional stimuli were of 2 types: 1) a loud, aversive noise (106 db), and 2) a series of 15 slide photographs. A different slide was used on each of the 15 trials involving pictorial UCSs. The order of slides presented as UCSs to all subjects was as follows: 1) "You are Cute" sign, 2) picture of a fried egg lying on the dessert, 3) "cute" children unwrapping Christmas presents, 4) a rose, 5) picture of shoulders and head of a woman projected upside down, 6) impossible figure, 7) newborn baby in hospital nursery, 8) mountain scenery, 9) half-naked man projected sideways, 10) coloured lights, 11) impression of smiling face in snow, 12) ornate painting of a cat done by a schizophrenic, 13) picture from Harlow studies of rhesus monkey clinging to cloth surrogate mother, 14) green-tinted telephone lying on rocky beach, 15) black-and-white photograph of female nude projected upside down.

The auditory UCS was recorded on an Ampex 800 tape recorder. The sound was originally produced by placing the recording microphone three inches in front of a speaker thus producing a "feedback" noise. This stimulus was recorded at a tape speed of 1 7/8 inches per second and played to subjects at a speed of 3 3/4 inches per second.

Continuous recordings were made of subjects' HR and SC in both sessions on a Beckman Type-R Dynograph, using Ag/AgCl electrodes and Beckman paste as the electrolyte medium. Skin conductance recordings were taken from the volar surface of the third phalange on the index and ring finger of subjects' right hands. Skin conductance was displayed directly in micromhos through a Lykken skin-conductance coupler (Beckman Type 9844). Heart-rate electrodes were placed on subjects' left and right wrists. Beat-to-beat changes in heart-rate were recorded through a Beckman Type 9857
cardiotachometer coupler, while the raw EKG was displayed simultaneously on a separate channel controlled by a Beckman Type 9806 A AC coupler.

Procedure

Session 1. In the first session of the study, subjects were taken to the experimental room and seated in a comfortable chair. The experiment was then described to them as a two-stage procedure involving "one's reactions to different forms of stimulation and to other people's experience of things which happen to them". After explaining the function of the various physiological recording electrodes, the following instructions were read to the subjects:

In front of you is a black board with a number of coloured lights on it. In addition, in front of you there is a screen and beside you there is a speaker. When the experiment begins, a number of things will happen. The first thing that will happen is that the light in the upper half of the board will come on. This is your signal that what we call a "trial" has begun. This light will remain on for several seconds. When this light goes off, one of three lights below it will come on and remain on for several seconds. A few seconds after this second light comes on, one of three things will happen. The first thing that may happen is that the second light in the series may simply turn off. The second thing that may happen is that a picture may be presented on the screen in front of you. The third thing that may happen is that you may hear a very loud, unpleasant but harmless noise. While all this is going on, a video-tape camera will occasionally be taking films of you.

After these instructions were read to the subjects, they were given a chance to ask any questions. All such questions were answered except insofar as they pertained to the research paradigm or hypothesis. Subjects then signed consent forms, subsequent to which the physiological recording electrodes were attached. After a 5 minute adaptation period, the first session began.
The first session employed a three part differential classical conditioning paradigm with three different CSs being conseuated by three different UCSs. The CSs were the coloured jewel lights mounted in the bottom half of the stimulus display board located in front of the subject's chair. The duration of CS presentation was 8 seconds, while the duration of presentation of auditory and pictorial UCS was 4 seconds. In order to ensure that subjects would be attending to the relevant stimulus cues, each trial began with the onset of a warning stimulus (WS) which preceded the CS by 6 seconds and terminated at CS onset. The WS was spatially separated from the CSs, and was located in the upper half of the stimulus board. Three different types of conditioning trial were thus employed. In the first type, one CS was consistently terminated by a 4 second presentation of a pictorial stimulus (CS-P), in the second type a different CS was consistently terminated by a 4 second presentation of an aversive auditory stimulus (CS-A), and in the third type, the CS was simply presented for 8 seconds after which it was turned off (CS-N). The conditioning procedure employed is depicted schematically in Fig. 3. Each type of trial was presented 15 times for a total of 45 trials in all. Three different orders of trial presentation were randomly generated with the sole restriction that in each block of 3 trials, all three types of conditioning trial would be represented. Subjects were randomly assigned to trial orders.

The arrangement of CS lights was altered randomly for each subject in order to control for any inherent eliciting properties of the lights themselves. Owing to the construction of the apparatus, however, it was impossible to randomize the spatial location of the CS lights, so that CS-P was always the light at the far right of the stimulus display, CS-A was
Fig. 3. Schematic representation of the differential conditioning procedure employed in this study. WS = warning stimulus, CS-A = aversive conditional stimulus, CS-P = pictorial conditional stimulus, CS-N = neutral conditional stimulus, UCS-A = aversive unconditional stimulus, UCS-P = pictorial unconditional stimulus, UCS-N = neutral unconditional stimulus. Solid line at bottom of figure indicates period of time during which subjects' facial expressions were videotaped.
always the light at the far left, and CS-N was always in the middle.

Inter-trial intervals ranged between 10 and 16 seconds. Prior to the beginning of conditioning trials, the video-tape camera was focused on the subjects' facial area. When trials began, the exposure of the videotape was controlled by the automatic shutter. The shutter apparatus was wired such that it opened 3 seconds prior to CS presentation and closed at UCS onset. Thus, the video recordings only occurred during an interval of 11 seconds prior to UCS administration, ensuring that unconditional responses were not recorded. In order to reduce the amount of time required for testing of subjects in the second session, only the first and last 15 trials were recorded on videotape (i.e., the first and last 5 presentations of each CS-UCS sequence).

Session 2. Inter-session intervals varied widely. In general, hospitalized subjects underwent the second session of the experiment much sooner than did nonhospitalized subjects. Mean inter-session intervals for the three groups were as follows: NC - 38.5 days (S.D. = 29.27), PC - 24.8 days (S.D. = 23.37), D - 20.7 days (S.D. = 11.62).

Prior to bringing subjects back to complete the second part of the experiment, subjects were assigned to squads comprised of 2 members of each of the 3 classes of subjects. This was done in order to make it possible to show each subject the videotape of one person from each class of subject as depicted in Fig. 4. In assigning subjects from each class to squads, it was attempted, as far as possible to choose subjects of similar ages.

When subjects returned to take part in the second session, they were seated in front of a television monitor and the procedure of the
Fig. 4. Manner in which videotapes were selected for showing to individual subject. Each octagon represents one subject. An arrow leading from one subject to another indicates that the videotape of the first subject was shown to the second, e.g., subject NC 1 observed the videotapes of subjects PC 1, D 1, and NC 2. NC = normal control, PC = psychiatric control, D = depressive.
The first session was reviewed. The following instructions were read:

In the first part of the experiment, which you have already completed, you went through a procedure wherein a number of different events happened. First, the blue light in the upper half of this black board went on, signalling to you that a "trial" had begun. Several seconds later, one of the three lights below the blue one went on. Depending on which light went on, a number of different things happened. If the amber (red, green) light went on, several seconds later it simply turned off and nothing else happened. If the green (amber, red) light went on, several seconds later, you were shown a slide photograph. If the third light went on, you heard a loud noise.

Thus, subjects who may have been unaware of the conditional relationships between stimuli in the first session had them explained at this point. The instructions then went on to say:

While all these things were going on, a video tape recording was made of your facial expressions at different points in time. These recordings began while the first light in the series, the blue light, was on. They ended at the exact same time as the second light in the series, the red, green or amber light, went off. No recordings were made while either the noise was on or the picture was on. Therefore, all that we have are recordings of people anticipating what is about to happen to them.

At this point, the experimenter stopped reading the instructions and queried the subject with regard to her understanding of the instructions up to that point. When it was clear that the subject understood the conditional relationships between stimuli in the first session, and the portion of the WS-CS-UCS sequence that was recorded, the nature of the experimental tasks was then explained. Subjects were informed that they would be viewing a series of 30 excerpts from each of three different people going through the first session of the experiment. They were then told that they were to view each of these excerpts for its full duration, and at the end of each, to guess which of the three possible events (pic-
ture presentation, sound presentation, or nothing) was about to occur to the subject they were viewing. Subjects indicated their judgement by calling out the words "sound", "picture" or "nothing". Prior to showing each set of 30 excerpts from each subject, subjects were further asked to estimate the number of trials they would judge correctly.

After instructions had been read to the subject, HR and SC electrodes were attached and a 5 minute adaptation period took place. After the adaptation period, the judgemental task then began. After each guess, the experimenter told the subject whether she was correct or incorrect, and, if incorrect, what the correct answer was. This was done for two reasons: 1) previous research using similar methodology had used complete informative feedback and 2) feedback allowed subjects to compare their actual performance with their prior prediction of their performance.

Each subject was shown the videotapes of three other subjects belonging to her squad; one subject from her own group and one subject from each of the other two groups. Order of presentation of the videotapes was randomly determined for each subject. After the subject had viewed the complete set of 30 excerpts from each subject, a brief pause occurred while the experimenter changed videotapes. During this pause, subjects were told how many excerpts they had judged correctly for the preceding subject, and were then asked to estimate the number they would get correct for the next subject. After the subject had viewed the full complement of videotapes from 3 other subjects, electrodes were removed and the experimenter attempted to answer any questions she might have had about the experiment.
Quantification of psychophysiological data.

SC data from the classical conditioning phase of the experiment were recorded over five scoring intervals corresponding to the following time periods in relation to each conditioning trial: (1) pre-stimulation -- the average of 3 SC levels, the first occurring four seconds prior to WS onset, the second at two seconds prior to WS onset, and the third exactly at WS onset. (2) WS -- the maximum SC response occurring in the seven second period commencing at WS onset and ending one second subsequent to WS offset. (3) CS -- the maximum SC value occurring in the eight-second interval commencing one second subsequent to CS onset and ending one second subsequent to CS offset. (4) UCS -- the maximum response occurring in the four-second interval commencing one second subsequent to UCS onset and ending one second subsequent to UCS offset. (5) post-stimulation -- the average of the three SC values beginning one second after UCS offset and at two and four seconds thereafter.

HR data were scored over thirteen two-second intervals. The first scoring interval began four seconds prior to WS onset, the second began two seconds prior to WS onset, and so on. Thus, the last scoring interval ended four seconds after UCS offset. The mean rate in beats per minute of all beats occurring wholly or partially within each scoring interval served as the HR value for that interval.
Chapter IV

RESULTS

Judgemental data.

Communication accuracy. The major dependent variable of this study consisted of the number of correct judgements by each subject observing videotapes in the second session of the experiment ("receiver subjects") of the 30 videotaped excerpts of each of 3 subjects participating in the first session ("sender subjects").

Since there was a significant difference between the three diagnostic groups in age, it was of interest to determine whether or not there was any consistent relationship between the age variable and subjects' behaviour during the first and second sessions of the experiment. For this reason, composite scores were determined which reflected each subject's general performance as both sender and receiver. The sender score for each subject was taken as the mean number of correct identifications of the type of conditioning trial being experienced by that particular subject by each of the 3 receiver subjects who viewed her videotape. The receiver score was the mean accuracy of each receiver subject in identifying the type of conditioning trial being experienced by each of the 3 senders whose videotapes were being shown to her. Pearson r's were then calculated between the age variable and both of these accuracy scores. The results of both correlations were insignificant: the age-sender r being .03 and the age-receiver r being -.16. This finding suggests that any between-group mean differences cannot be attributed to a confound of age with diagnostic category.
Judgemental accuracy scores were subjected to a 4-factor mixed model analysis of variance. The between-groups factor was diagnostic category of receiver subject. The three within-groups factors were: diagnostic category of sender subject, trial blocks (obtained by dichotomizing the data for each receiver's judgements of each sender at the 15th trial), and type of conditioning trial.

The results of this analysis (Table 3) revealed significant main effects for the type of sender ($F(92, 54) = 18.04, p < .01$) and trial blocks ($F(1, 27) = 33.08, p < .01$) variables, and a significant receiver X sender X trial-blocks interaction ($F(4, 54) = 4.10, p < .05$). The significant main effect for the sender variable (Fig. 5) is accounted for by the poor performance of all three types of receiver subject in making judgements of D senders relative to both NC and PC senders. The trial-blocks main effect reflected the tendency for the accuracy of all receiver subjects' judgements to improve on the second block of 15 trials for each sender subject judged. The significant receivers X senders X trial-blocks interaction is depicted in Fig. 6. Post-hoc analyses of the simple effects of this significant interaction were performed using Cicchetti's (1972) modification of the Tukey "honestly significant difference" (HSD) procedure. Results of this analysis are presented in Table 4. All differences are significant at the $p < .05$ level. The major trends of this interaction can be summarized as follows. Performance of NC and PC receivers observing NC and PC subjects improved over trials. NC receivers observing D senders failed to improve over trials, while PC receivers observing D senders did. D receivers observing NC and D senders improved from trial block 1 to trial
Table 3. Results of the analysis of variance for judgemental accuracy data.

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<th>F</th>
<th>p</th>
</tr>
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<td>T X R</td>
<td>3.84</td>
<td>2</td>
<td>1.92</td>
<td>1.30</td>
<td>n.s.</td>
</tr>
<tr>
<td>Error</td>
<td>39.79</td>
<td>27</td>
<td>1.47</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>2.23</td>
<td>2</td>
<td>1.12</td>
<td>0.57</td>
<td>n.s.</td>
</tr>
<tr>
<td>C X R</td>
<td>4.16</td>
<td>4</td>
<td>1.04</td>
<td>0.53</td>
<td>n.s.</td>
</tr>
<tr>
<td>Error</td>
<td>105.28</td>
<td>54</td>
<td>1.95</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S X T</td>
<td>5.94</td>
<td>2</td>
<td>2.97</td>
<td>2.21</td>
<td>n.s.</td>
</tr>
<tr>
<td>S X T X R</td>
<td>22.07</td>
<td>4</td>
<td>5.52</td>
<td>4.10</td>
<td>p &lt; .05</td>
</tr>
<tr>
<td>Error</td>
<td>72.66</td>
<td>54</td>
<td>1.35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S X C</td>
<td>2.83</td>
<td>4</td>
<td>0.71</td>
<td>0.50</td>
<td>n.s.</td>
</tr>
<tr>
<td>S X C X R</td>
<td>7.88</td>
<td>8</td>
<td>0.98</td>
<td>0.70</td>
<td>n.s.</td>
</tr>
<tr>
<td>Error</td>
<td>152.62</td>
<td>108</td>
<td>1.41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T X C</td>
<td>0.29</td>
<td>2</td>
<td>0.15</td>
<td>0.18</td>
<td>n.s.</td>
</tr>
<tr>
<td>T X C X R</td>
<td>2.39</td>
<td>4</td>
<td>0.60</td>
<td>0.75</td>
<td>n.s.</td>
</tr>
<tr>
<td>Error</td>
<td>152.62</td>
<td>54</td>
<td>0.80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S X T X C</td>
<td>3.82</td>
<td>4</td>
<td>0.95</td>
<td>1.00</td>
<td>n.s.</td>
</tr>
<tr>
<td>S X T X C X R</td>
<td>6.60</td>
<td>8</td>
<td>0.83</td>
<td>0.87</td>
<td>n.s.</td>
</tr>
<tr>
<td>Error</td>
<td>102.91</td>
<td>108</td>
<td>0.95</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Fig. 5. Mean number of correct identifications by receiver subjects of the type of conditioning trial sender subjects were experiencing. NC = normal control, PC = psychiatric control, D = depressive.
Fig. 6. Mean number of correctly identified conditioning trials as a function of diagnostic category of receiver subject, diagnostic category of sender subject, and trial blocks. NC = normal control, PC = psychiatric control, D = depressive; T1 = trial block 1, T2 = trial block 2.
Table 4. Results of Tukey HSD analysis of simple effects of Receiver X Sender X Trial Block interaction (communication accuracy data). NC = normal control, PC = psychiatric control, D = depressive; $T_1$ = trial-block 1, $T_2$ = trial block 2.

A. Trial blocks simple effects

<table>
<thead>
<tr>
<th>Sender</th>
<th>Receiver</th>
<th>NC</th>
<th>PC</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>NS</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
<td></td>
</tr>
<tr>
<td>PC</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 = T_2$</td>
<td></td>
</tr>
<tr>
<td>D</td>
<td>$T_1 = T_2$</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
<td></td>
</tr>
</tbody>
</table>

B. Senders simple effects

<table>
<thead>
<tr>
<th>Receiver</th>
<th>Trial Block</th>
<th>NC</th>
<th>PC</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$T_1$</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
</tr>
<tr>
<td></td>
<td>$T_2$</td>
<td>$T_1 = T_2$</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
</tr>
</tbody>
</table>

C. Receivers simple effects

<table>
<thead>
<tr>
<th>Sender</th>
<th>Trial Block</th>
<th>NC</th>
<th>PC</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$T_1$</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
</tr>
<tr>
<td></td>
<td>$T_2$</td>
<td>$T_1 = T_2$</td>
<td>$T_1 &lt; T_2$</td>
<td>$T_1 &lt; T_2$</td>
</tr>
</tbody>
</table>
block 2, while D receivers observing PC senders did not. In general, NC and PC senders were significantly easier to judge accurately than were D senders. The only exception to this finding occurred among D receivers observing PC senders during trial block 2. In this instance, D and PC senders were equally difficult to judge relative to NC senders. With regard to the receivers variable, the only differences observed were significantly poorer performance for D receivers (relative to NC and PC receivers) judging PC senders during the second trial block and significantly poorer performance for NC receivers (relative to D receivers) judging D senders during trial block 2.

Because it was of further interest to evaluate the relationship between subjects' performance as senders and their performance as receivers, a Pearson $r$ was calculated between each subject's composite receiver and sender score. The results of this analysis yielded an insignificant $r$ value ($r = .08$).

Category choice analysis. Although the analyses of communication accuracy were of major interest, the information that they provided with respect to subjects' task performance was incomplete. It was possible that performance on the task was likely to be affected by stereotyped response predispositions on the part of both sender and receiver subjects. For example, it seemed plausible that depressed subjects would be relatively more likely than nondepressed subjects to emit facial cues that other people would label "aversive" (e.g., expressions of being hurt, etc.). Such a phenomenon might be expected to manifest itself in a tendency for all classes of receiver subject to choose a particular response class with greater frequency when describing depressives than when describing non-
depressives. That is, if, for example, depressed sender subjects tended to emit "aversive" cues in general, all classes of receiver subject might have been expected to choose the response class indicative of the presence of aversive stimulation with greater relative frequency when observing depressed subjects.

Similarly, it seemed plausible that receiver subjects might have evidenced stereotyped response predispositions in their choice of particular response categories. For example, depressed receiver subjects might be expected to perceive the presence of "aversive" cues in the behaviour of others and therefore choose a particular response category more frequently than other subjects.

In order to evaluate these questions, the judgemental data from each receiver subject were broken down into three different scores. The frequency with which receiver subjects chose each of the three possible response categories when judging each of three sender subjects (irrespective of whether or not the choice of a particular category was correct) was subjected to analysis of variance. Thus, three separate analyses of variance were performed: one for the frequency with which the "aversive" response category was chosen and one analysis each for the frequency with which the "pictorial" and "neutral" classes were chosen. Each analysis investigated the effects of the receivers, senders, and trial blocks variables. Stereotyped response predispositions on the part of a given class of receiver or sender subjects would, thus, be reflected in a significant main effect for that variable.

No significant effects were found in the analysis of the frequency with which the "aversive" response class was chosen. Analysis of the
frequency with which the "pictorial" response category was chosen revealed a
significant receiver X sender interaction ($F (4, 54) = 4.76, p < .05$) (Table
5) depicted in Fig. 7. Post hoc analyses of this interaction by the Neu-
man-Keuls technique showed that PC receivers judging D senders used the
"pictorial" response category significantly less frequently ($p < .05$) than
did NC and D receivers judging D senders and PC receivers judging PC senders.

Analysis of the frequency of choice of the "neutral" response cate-
gory (Table 6) yielded a significant main effect for the trial blocks
variable ($F (1, 27) = 7.26, p < .05$) indicating that the choice of the
"neutral" response category decreased from the first to the second trial block.

Thus, analyses of the judgemental data revealed that D subjects
were generally more difficult to judge accurately than were NC and PC sub-
jects, that performance on the judgemental task improved over time, and
that there was little evidence for the existence of stereotyped response
predispositions on the part of any of the classes of sender or receiver
subjects. Further, NC subjects' judgemental performance when observing Ds
did not improve over time, nor did D receivers' performance when judging PC
senders.

Performance and predictions of performance

During the judgemental task all subjects were asked to estimate how
many trials out of 30 they would judge correctly prior to observing each
set of 3 videotapes. This was done in order to evaluate the prediction,
derived from Beck's theory, that D subjects, relative to NC's and PC's
would tend to underestimate their own performance. These data were sub-
jected to a 2-way between-within analysis of variance with diagnostic
Table 5. Results of the analysis of variance for category choice data: Frequency of choice of "pictorial response category."

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>420.06</td>
<td>179</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R</td>
<td>6.68</td>
<td>2</td>
<td>3.34</td>
<td>0.95</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>94.55</td>
<td>27</td>
<td>3.50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S</td>
<td>0.08</td>
<td>2</td>
<td>0.04</td>
<td>0.01</td>
<td>ns</td>
</tr>
<tr>
<td>S X R</td>
<td>13.55</td>
<td>4</td>
<td>3.39</td>
<td>4.76</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Error</td>
<td>153.70</td>
<td>54</td>
<td>2.85</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T</td>
<td>0.14</td>
<td>1</td>
<td>0.14</td>
<td>0.10</td>
<td>ns</td>
</tr>
<tr>
<td>T X R</td>
<td>1.01</td>
<td>2</td>
<td>0.51</td>
<td>0.70</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>39.02</td>
<td>27</td>
<td>1.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S X T</td>
<td>5.34</td>
<td>2</td>
<td>2.67</td>
<td>1.39</td>
<td>ns</td>
</tr>
<tr>
<td>S X T X R</td>
<td>1.96</td>
<td>4</td>
<td>0.49</td>
<td>0.25</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>104.03</td>
<td>54</td>
<td>1.93</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Fig. 7. Frequency of correct or incorrect judgements that sender subjects were undergoing a pictorial conditioning trial as a function of diagnostic category of sender and receiver subject.
Table 6. Results of the analysis of variance for category choice data: Frequency of choice of "neutral" response category.

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>566.20</td>
<td>179</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R</td>
<td>15.63</td>
<td>2</td>
<td>7.82</td>
<td>1.09</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>177.57</td>
<td>27</td>
<td>6.58</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S</td>
<td>11.63</td>
<td>2</td>
<td>5.82</td>
<td>2.19</td>
<td>ns</td>
</tr>
<tr>
<td>S X R</td>
<td>6.13</td>
<td>4</td>
<td>0.03</td>
<td>0.58</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>143.23</td>
<td>54</td>
<td>2.65</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T</td>
<td>9.80</td>
<td>1</td>
<td>9.80</td>
<td>7.26</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>T X R</td>
<td>0.10</td>
<td>2</td>
<td>0.05</td>
<td>0.04</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>36.43</td>
<td>27</td>
<td>1.35</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S X T</td>
<td>16.30</td>
<td>2</td>
<td>8.15</td>
<td>3.08</td>
<td>ns</td>
</tr>
<tr>
<td>S X T X R</td>
<td>6.40</td>
<td>4</td>
<td>1.60</td>
<td>0.60</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>142.97</td>
<td>54</td>
<td>2.65</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
category of receiver being the between groups variable and ordinal position of prediction being the within-groups variable. The results of this analysis revealed no significant differences for the class of receiver ($F = 0.23, \text{ ns}$), ordinal position of prediction ($F = 0.45, \text{ ns}$) or receiver X position ($F = 0.32, \text{ ns}$) terms.

It was of further interest to determine the extent to which subjects' predictions of their own performance changed as a function of the difference between their predictions and their actual performance. On the basis of Seligman's theory, it was expected that there would be little or no relationship between changes in subjects' predictions, and the differences between previous predictions and previous performance. In order to evaluate this, pairs of difference scores were calculated for each subject. The first pair of difference scores was acquired by subtracting subjects' predictions of performance for the first presentation of videotaped excerpts from their predictions for the second presentation and by subtracting subjects' predictions for the first presentation of videotapes from their actual performance on the first presentation. The second pair of difference scores was calculated in the same manner, and reflected change in predictions from the second to the third presentation of videotapes and the discrepancy between predictions and actual performance on the second presentation of videotapes. The third pair of difference scores reflected change in predictions from the second to the third presentation of videotapes, and the discrepancy between prediction and actual performance on the first presentation of videotapes. Pearson's $r$'s were then calculated between each of these pairs of difference scores, separately for each diagnostic group. Results of this analysis are presented in Table 7 and
Table 7. Correlations between difference scores reflecting (1) change in predictions and (2) discrepancy between predictions and performance

<table>
<thead>
<tr>
<th>Group</th>
<th>Difference 1</th>
<th>Difference 2</th>
<th>NC</th>
<th>PC</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$P_2 - P_1$</td>
<td>$A_1 - P_1$</td>
<td>.78**</td>
<td>.90**</td>
<td>.88**</td>
</tr>
<tr>
<td></td>
<td>$P_3 - P_2$</td>
<td>$A_2 - P_2$</td>
<td>.08</td>
<td>.77**</td>
<td>.71*</td>
</tr>
<tr>
<td></td>
<td>$P_3 - P_2$</td>
<td>$A_1 - P_1$</td>
<td>.47</td>
<td>-.13</td>
<td>-.05</td>
</tr>
</tbody>
</table>

$P = $ prediction of performance; $A = $ actual performance; subscripts refer to ordinal position of prediction or actual performance.

** $p < .01$

* $p < .05$
reveal some interesting findings. For all groups, the correlations between change in predictions from the first to the second presentation of videotapes and the discrepancy between predictions and actual performance on the first presentation of videotapes were strong, positive and significant. For the correlations between change in predictions from the second to the third presentation of videotapes and the discrepancy between predictions and actual performance on the second presentation of videotapes, both the PC and D groups still showed significant positive values. Only for the NC group was this correlation insignificant. For the correlation between change in predictions from the second to the third presentation of videotapes and the discrepancy between predictions and actual performance on the first presentation of videotapes, none of the $r$ values was significant.

Thus, while analysis of variance procedures revealed nothing in the way of systematic between-group differences in subjects' ratings of their anticipated performance, correlational procedures revealed significant strong and positive correlations between changes in predictions and discrepancies between predictions and actual performance. The magnitudes of these correlations, however, did not appear to vary in any systematic manner among the three groups.

**Psychophysiological data.**

**Skin conductance.** SC data were initially subjected to a four-factor, mixed analysis of variance with subjects' diagnostic category the between groups factor and type of conditioning trial, and scoring interval providing two of the three within groups factors. The third within groups factor was obtained by grouping the fifteen presentations of each of the three different
types of conditioning trial into blocks of five trials each. This was done in order to evaluate the development of response patterns as a function of increasing experience in the conditioning situation. Due to equipment problems, the SC recordings of two subjects in the D group were unscorable; therefore the results of this analysis are based on an N of 8 subjects in this group, while the NC and PC groups' data are based on an N of 10 in each group. Results of the analysis of variance (Table 8) revealed significant main effects for the type of conditioning trial ($F(4, 100) = 4.91, p < .05$), trial blocks ($F(2, 50) = 11.61, p < .01$) and scoring interval ($F(4, 100) = 17.57, p < .01$) variables and significant type of conditioning trial X scoring interval ($F(8, 200) = 12.35, p < .01$) and trial blocks X scoring interval ($F(8, 200) = 2.81, p < .01$) interactions in addition to significant conditioning trial X scoring interval X diagnostic group ($F(16, 200) = 2.30, p < .01$) and conditioning trial X trial block X scoring interval ($F(16, 400) = 3.59, p < .01$) third order interactions.

Subsequent to this initial analysis, a series of planned orthogonal contrasts were performed. Two orthogonal comparisons evaluated the effects of the type of conditioning trial variable. These comparisons demonstrated that SC values for the aversive auditory conditioning trial were significantly different from those for the pictorial and neutral trials ($t = 9.88, p < .01$) but the latter two types of conditioning trial did not differ ($t = .04, ns$). Inspection of mean SC values reveals that these values tended to be higher on aversive conditioning trials ($\bar{X} = 15.43 \mu$mho) than on pictorial ($\bar{X} = 15.10 \mu$mho) and neutral ($\bar{X} = 15.09 \mu$mho) trials. Orthogonal analyses of the trial blocks variable showed that the difference
Table 8. Results of the analysis of variance for skin conductance data.

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>90,443.58</td>
<td>1259</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>G (Diagnostic groups)</td>
<td>7,627.50</td>
<td>2</td>
<td>3813.75</td>
<td>1.36</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>70,360.00</td>
<td>25</td>
<td>2814.40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C (Type of conditioning)</td>
<td>12.56</td>
<td>2</td>
<td>6.28</td>
<td>4.90</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>C X G</td>
<td>2.24</td>
<td>4</td>
<td>0.56</td>
<td>0.44</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>63.99</td>
<td>50</td>
<td>1.28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T (Trial blocks)</td>
<td>3,543.93</td>
<td>2</td>
<td>1771.96</td>
<td>11.61</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>T X G</td>
<td>889.19</td>
<td>4</td>
<td>222.30</td>
<td>1.46</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>7,630.42</td>
<td>50</td>
<td>152.61</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C X T</td>
<td>2.30</td>
<td>4</td>
<td>0.57</td>
<td>0.86</td>
<td>ns</td>
</tr>
<tr>
<td>C X T X G</td>
<td>7.59</td>
<td>8</td>
<td>0.95</td>
<td>1.42</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>66.71</td>
<td>100</td>
<td>0.67</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I (Scoring interval)</td>
<td>17.12</td>
<td>4</td>
<td>4.28</td>
<td>17.57</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>I X G</td>
<td>2.08</td>
<td>8</td>
<td>0.26</td>
<td>1.07</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>24.36</td>
<td>100</td>
<td>0.24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C X I</td>
<td>35.42</td>
<td>8</td>
<td>4.43</td>
<td>12.35</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>C X I X G</td>
<td>13.21</td>
<td>16</td>
<td>0.83</td>
<td>2.30</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Error</td>
<td>71.72</td>
<td>200</td>
<td>0.36</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T X I</td>
<td>2.50</td>
<td>8</td>
<td>0.31</td>
<td>2.81</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>T X I X G</td>
<td>2.53</td>
<td>16</td>
<td>0.16</td>
<td>1.42</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>22.29</td>
<td>200</td>
<td>0.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C X T X I</td>
<td>5.44</td>
<td>16</td>
<td>0.34</td>
<td>3.59</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>C X T X I X G</td>
<td>2.66</td>
<td>32</td>
<td>0.08</td>
<td>0.88</td>
<td>ns</td>
</tr>
<tr>
<td>Error</td>
<td>37.82</td>
<td>400</td>
<td>0.09</td>
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</tbody>
</table>
in SC values between the first trial block and the second two trial blocks was significant ($t = 18.76, p < .01$) as was the difference between trial block two and trial block three ($t = 4.45, p < .05$). Inspection of means for the three trial blocks reveals an increase in SC with longer exposure to the experimental situation ($\bar{X}$ trial block 1 = 13.02 $\mu$mho, $\bar{X}$ trial block 2 = 15.34 $\mu$mho, $\bar{X}$ trial block 3 = 17.15 $\mu$mho). SC values over the five levels of the scoring intervals variable were subjected to a linear trend analysis which resulted in a significant $t$-value ($t = 58.81, p < .01$). This significant general trend reveals a tendency for skin conductance values to increase over the five scoring intervals as depicted in Fig. 8. Subsequent analyses of the interaction between this linear trend component and the other independent variables proved informative. The interaction between the linear trend of SC values over scoring intervals and the type of conditioning trial variable dichotomized by contrasting the aversive auditory conditioning condition with the neutral and pictorial conditions combined had a significant $t$ value of 84.56 ($p < .01$). However, the interaction of the linear trend component for scoring intervals with the type of conditioning trial variable dichotomized as pictorial contrasted with neutral conditioning trials was not significant ($t = 1.46$). The interaction of the linear trend over scoring intervals with the type of conditioning trial (aversive auditory vs. pictorial and neutral combined) is depicted in Fig. 9. From this figure it can be seen that on aversive auditory conditioning trials, SC values increased as far as the fourth scoring interval (corresponding to the period of UCS presentation) and then tended to decrease during the poststimulation interval (interval 5) to a level slightly below that at the fourth scoring interval. On the other
Fig. 8. Mean SC values over the five scoring intervals.
Fig. 9. Mean SC values over the five scoring intervals on aversive (A) trials and on the pictorial (P) and neutral (N) trials combined.
two types of conditioning trial little change in skin conductance values over trials is evident.

The final set of orthogonal contrasts examined the interaction between type of conditioning trial, diagnostic groups and the linear component over scoring intervals. The first of these examined the interaction between type of conditioning trial dichotomized as aversive auditory vs. pictorial and neutral trials combined, diagnostic groups dichotomized as group D vs. groups PC and NC combined, and the linear trend over scoring intervals and yielded a significant $t$ value ($t = 29.47$, $p < .01$). The interaction between type of conditioning trial (pictorial vs. neutral), diagnostic groups (group D vs. groups PC and NC combined) was insignificant ($t = 0.13$) as were the interaction terms contrasting groups PC and NC as a function of aversive versus pictorial and neutral trials and the linear trend over intervals ($t = 0.46$) and the interaction term for groups PC vs. NC X pictorial vs. neutral conditioning trials X linear trend ($t = 0.47$). The relationship between diagnostic category of subject, type of conditioning trial and, the linear trend over scoring intervals is depicted graphically in Fig. 10. From this figure it can be seen that for the combined PC and NC group and for the D group SC values on non-aversive types of conditioning trial remain fairly stable over each of the five scoring intervals. Cicchetti's (1972) modification of the Tukey HSD multiple comparison procedure was used after the orthogonal analyses in order to clarify the nature of the difference observed. These analyses were first performed on the difference between scoring intervals 1 through 5 on non-aversive trials for the D group and the combined PC and NC group separately. No differences between scoring intervals were found. Next, differences in mean
Fig. 10. Mean SC values over the five scoring intervals on aversive (A) trials and on the pictorial (P) and neutral (N) trials combined as a function of subjects' diagnostic categories. NC = normal control, PC = psychiatric control, D = depressive.
SC values between scoring intervals 1 through 5 on aversive trials were examined. This analysis, performed on the scores of the combined NC and PC group, revealed only that scoring interval 4 (UCS presentation) differed from scoring interval 1 (prestimulation). In contrast, among D subjects, intervals 1, 2, and 3 (prestimulation, WS, CS) differed significantly from intervals 4 and 5 (UCS, poststimulation), but not from one another. Contrasts between aversive and nonaversive trials at each of the five scoring intervals were conducted separately for groups PC and NC combined and group D. These contrasts showed significantly higher mean SC values on aversive trials only during UCS presentation for NC and PC subjects, while D subjects' mean SC on aversive trials was significantly higher than on non-aversive trials during UCS presentation and during the poststimulation interval.

Since no specific predictions had been made beforehand with respect to the interaction between trial blocks and scoring intervals and the interaction between type of conditioning trial, trial blocks and scoring intervals, analyses of these significant interactions were carried out using the modified Tukey procedure.

For the trial blocks X scoring intervals interaction SC values were compared across blocks at each of the five scoring intervals, and then across each of the five scoring intervals within trial blocks. This analysis revealed higher SC values during trial block 3 than during trial block 2, and higher SC values during trial block 2 than during trial block 1 at each level of the scoring intervals variable. Patterns of SC change, over the five scoring intervals differed slightly during each trial block. The most marked changes occurred during the first trial block, where SC
values were significantly higher in intervals 4 and 5 (UCS presentation and poststimulation) than during intervals 1-3. During trial blocks 2 and 3 SC values were higher during interval 4 than during interval 1 (pre-stimulation); no other differences were significant.

The analysis for the type of conditioning trial X trial blocks X scoring intervals interaction revealed that superimposed upon a significant general increase in SC values over each trial block was a significant increase in SC over scoring intervals during aversive conditioning trials. There were no reliable changes in SC values over scoring intervals during pictorial and neutral trials. The pattern of SC change during aversive trials was one of increasing SC during those scoring intervals corresponding to CS and UCS presentation, with elevations being maintained during the post-stimulation interval. A significant increase in SC during WS presentation was observed on the first trial block but not thereafter. Further, SC values increased during those scoring intervals corresponding to UCS presentation and the poststimulation interval relative to the interval corresponding to the period of CS presentation on trial blocks 1 and 3, but not on trial block 2. These data are depicted graphically in Fig. 11.

Heart rate. HR data were initially subjected to a 3 X 3 X 3 X 13 mixed model analysis of variance. As with SC, diagnostic category of subjects comprised the between groups factor while type of conditioning trial, trial blocks, and scoring intervals comprised the within-groups factors. This analysis (Table 9) revealed significant main effects for diagnostic category of subject ($F(2, 27) = 3.43, p < .05$), type of con-
Fig. 11. Mean SC values over the five scoring intervals as a function of type of conditioning trial and trial blocks. A = aversive, P = pictorial, N = neutral; TB1 = trial block 1, TB2 = trial block 2, TB3 = trial block 3.
Table 9. Results of the analysis of variance for heart-rate data.

<table>
<thead>
<tr>
<th>Source</th>
<th>SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>p</th>
</tr>
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<tr>
<td>Total</td>
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<td>3509</td>
<td>44,078.69</td>
<td>3.43</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>G (Diagnostic groups)</td>
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<td>2</td>
<td>44,078.69</td>
<td>3.43</td>
<td>&lt;.05</td>
</tr>
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<td>Error</td>
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<td>27</td>
<td>12,838.13</td>
<td>3.43</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>C (Type of conditioning trial)</td>
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<td>2</td>
<td>83.88</td>
<td>3.93</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>C X G</td>
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<td>4</td>
<td>21.71</td>
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<tr>
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<td>54</td>
<td>21.37</td>
<td>1.02</td>
<td>ns</td>
</tr>
<tr>
<td>T (Trial blocks)</td>
<td>485.36</td>
<td>2</td>
<td>242.68</td>
<td>1.70</td>
<td>ns</td>
</tr>
<tr>
<td>T X G</td>
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<tr>
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<td>54</td>
<td>142.76</td>
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<td>ns</td>
</tr>
<tr>
<td>C X T</td>
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<td>4</td>
<td>3.61</td>
<td>0.12</td>
<td>ns</td>
</tr>
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<td>C X T X G</td>
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<td>8</td>
<td>35.79</td>
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<td>Error</td>
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<td>108</td>
<td>29.16</td>
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<td>ns</td>
</tr>
<tr>
<td>I (Scoring Intervals)</td>
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<td>12</td>
<td>42.88</td>
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</tr>
<tr>
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<td>ns</td>
</tr>
<tr>
<td>Error</td>
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<td>324</td>
<td>7.77</td>
<td>0.96</td>
<td>ns</td>
</tr>
<tr>
<td>C X I</td>
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<td>0.96</td>
<td>ns</td>
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<td>C X I X G</td>
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<td>8.05</td>
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<tr>
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<td>648</td>
<td>6.12</td>
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<td>ns</td>
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<td>T X I X G</td>
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<tr>
<td>Error</td>
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<td>5.79</td>
<td>1.16</td>
<td>ns</td>
</tr>
<tr>
<td>C X T X I</td>
<td>247.32</td>
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<td>5.15</td>
<td>0.88</td>
<td>ns</td>
</tr>
<tr>
<td>C X T X I X G</td>
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<td>96</td>
<td>6.13</td>
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<td>&lt;.001</td>
</tr>
<tr>
<td>Error</td>
<td>7,570.54</td>
<td>1296</td>
<td>5.84</td>
<td>1.05</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>
ditioning trial ($F (2, 54) = 3.92, p < .05$), and scoring intervals ($F (12, 324) = 5.32, p < .01$), and significant trial blocks X scoring intervals ($F (24, 648) = 2.00, p < .01$), and type of conditioning trial X trial blocks X scoring intervals X diagnostic groups ($F (48, 1296) = 1.05, p < .05$) interactions (Fig. 12). A series of planned orthogonal comparisons was then carried out in order to evaluate some of the main effects. These comparisons indicated that mean HR for group D was significantly different from that for groups PC and NC combined ($t = 2.62, p < .05$) but groups PC and NC did not differ from one another ($t = .10, ns$). Mean HR was higher for group D than for groups PC and NC ($\bar{X} = 91.94, 81.82, 81.50$, respectively). Orthogonal analyses of the class of conditioning trial variable showed that mean HR on aversive conditioning trials differed significantly from that on pictorial and neutral trials combined ($t = 28.58, p < .01$); the latter two types of conditioning trial not differing from each other ($t = 0.46, ns$). Mean HR was higher on aversive than on pictorial and neutral trials ($\bar{X} = 85.16, 84.66, 84.75$, respectively). As in the overall analysis of variance, no significant differences were noted in orthogonal analyses of the trial blocks variable.

Since no specific predictions had been generated with respect to the significant main effect obtained for scoring intervals, Tukey HSD analysis was employed in order to evaluate the significance of differences between all possible pairwise comparisons of the 13 levels of this factor. This analysis showed a significant HR deceleration during intervals 10, 11, and 12 relative to intervals 1-9. The significant trial blocks X scoring intervals interaction was shown on post-hoc analysis to derive from the development of a pattern of HR deceleration over trial blocks. During trial block 1, only
Fig. 12. Depiction of the significant interaction between subjects' diagnostic category, type of conditioning trial, trial blocks, and scoring intervals for HR data. Each column depicts the data from one group of subjects, whereas each row represents one type of conditioning trial. NC = normal control, PC = psychiatric control, D = depressive; A = aversive, P = pictorial, N = neutral; TB 1 = trial block 1, TB 2 = trial block 2; TB 3 = trial block 3.
interval 12 was significantly lower than interval 1, while during trial block 2 HR on interval 12 was lower than during intervals 7 and 8. During intervals 10, 11, and 12, HR was significantly lower than during intervals 2 and 4, 2, 3, 4, and 7 and 2 through 7, respectively.

The significant four-factor interaction between diagnostic groups, type of conditioning trial, trial blocks, and scoring intervals is shown graphically in Fig. 12. Analyses of this interaction were carried out by performing Cicchetti's (1972) modification of the Tukey HSD procedure on the main effects of each factor within this interaction. Because the results of this analysis were extremely complex, only the major trends in the data will be summarized here; the complex analysis is presented in Appendix B.

On aversive conditioning trials NC subjects evidenced the development of a significant accelerative change in HR values occurring during the CS interval on trial block 3. PC subjects evidenced the development of a similar accelerative HR change that did not attain statistical significance. D subjects evidenced no systematic changes in HR on aversive conditioning trials.

On pictorial conditioning trials, PC subjects evidenced the development of a significant decelerative HR change during the first post-stimulation interval on the second trial block and a similar, but non-significant pattern on the third. NC subjects evidenced somewhat lower HR values during the interval of UCS presentation on trial block 3, but this trend was, again, nonsignificant. D subjects exhibited no systematic HR changes.

On neutral conditioning trials, the only HR difference that occurred was a somewhat perplexing HR "spike" among NC subjects during the last interval of CS presentation on the first trial block.
During all scoring intervals and trial blocks, D subjects exhibited significantly higher HR than NC and PC subjects.

By way of summary, the following seem to be the most important of the psychophysiological findings. Tonic SC levels did not differ significantly between groups. A significant increment in tonic SC values was observed from early to later trials in the conditioning situation. A significant type of conditioning trial main effect and type of conditioning trial X scoring interval interaction revealed greater SC activity on aversive than on pictorial and neutral trials. Finally, a significant interaction between subjects' diagnostic category, type of conditioning trial, and scoring intervals seemed to reflect greater SC activity on aversive conditioning trials among D subjects than among NC and PC subjects.

HR data revealed greater tonic HR level among D subjects than among NC and PC subjects, greater HR level on aversive than on pictorial and neutral trials, and a significant 4-factor interaction between subjects' diagnostic group, type of conditioning trial, trial blocks, and scoring intervals. This latter finding seemed to reflect restricted variability of HR for D subjects as contrasted with NC and PC subjects and the apparent development of HR acceleration on aversive trials and HR deceleration on pictorial trials among non-D subjects.

Psychometric data - Self-Monitoring Scale

Snyder's (1975) scale assessing the self-monitoring of expressive behaviour (SM; see Appendix A) was administered to all subjects. This scale was developed in order to differentiate people according to their ability to "...observe and control their expressive behaviour and self-presentation" (Snyder, 1975, p.527). The aforementioned ability seemed
conceptually related to the processes involved in the experimental task. In particular, it seemed likely that subjects high in the ability putatively measured by this scale should exhibit better performance as receiver and/or sender subjects. To the extent that one group of subjects was characterized by exceptional performance as sender or receiver subjects, the same group should have exhibited correspondingly exceptional scores on the SM scale.

A one-way analysis of variance compared scores on this scale as a function of diagnostic groups. No significant effect was noted ($F(2, 27) = 0.22$). Two further correlational analyses assessed the degree of relationship between subjects SM scores and their composite scores as receivers and senders. Pearson $r$ values for these correlations were $-.06$ and $.13$ respectively; both nonsignificant.
Chapter V
DISCUSSION

Subject characteristics.

The extent to which valid conclusions about depression or depressed people can be drawn from the present study is dependent upon the presentation of evidence that the variable of "depression" was a meaningful discriminator of the three groups in the study. Psychometric evidence collected from the subjects in this study supports the assertion that the three groups did vary in level of depression. It is not surprising that the D group exhibited higher BDI scores than both the PC and NC groups in light of the fact that scores on this inventory were used as selection criteria. While a multivariate analysis revealed no significant between-group differences in either level or profile of MMPI scales, inspection of Fig.2 shows that the three groups' mean scores on the D scale were ordered appropriately. Subjects in the D group evidenced the highest scores on scale D, while PC and NC subjects exhibited lower scores, respectively. Moreover, while the multivariate analysis resulted in nonsignificant findings, a simple one-way analysis of variance of D scale scores resulted in a significant F value. The evidence, then, is consistent with the assertion that the variable of depression was a meaningful discriminator of the groups. Inspection of Fig. 2 reveals that the mean D scale score of the PC group was elevated above a T-score of 70. This elevation was largely accounted for by extreme scores on the part of 2 subjects. It is further likely that this elevation reflects the fact that the MMPI D-scale is multifactorial, and is not a particularly discriminating
measure of depression. Comrey (1957), for example, found in a factor analytic study that items from this scale load on at least a dozen factors, only 2 of which seem closely related to the concept of depression.

Beck (1967) devoted a great deal of attention to the problem of age differences between groups in research on depression. He noted in this regard that the risk for depression increases with increasing age, and that in much of the research which has compared groups of depressed individuals with groups of subjects belonging to other psychodiagnostic categories, depressive subjects often were significantly older. As a result of this, any differences found could not be attributed specifically to the effects of depression. In the present study, the average age of group PC was significantly lower than those of groups D and NC; however, the mean ages of the latter two groups did not differ. The significantly younger mean age of group PC is not surprising considering that the major proportion of these subjects were being treated in a sexual dysfunction clinic. This significant difference does not seem to be particularly troublesome methodologically in that the age variable did not correlate with subjects' efficacy in emitting or identifying nonverbal cues discriminating between the three types of conditioning trials. The fact that the NC and D groups were approximately equal on the age variable allows for further disclaiming the possibility that differences between groups on the experimental task were primarily accountable for in terms of age differences.

Judgemental data.

Communication accuracy. As can be seen in Fig. 5, depressed subjects were the most difficult "senders" to judge correctly. Multiple comparison
analyses showed that the differences between the NC and PC senders was not significant. This finding provides clear support for the hypothesis that depressed individuals exhibit a deficit in nonverbal communicative behaviour and provides further support for Lewinsohn's (1974, 1975; Lewinsohn, Weinstein, and Shaw, 1969) hypothesis that a lack of social skill represents a major antecedent condition for the genesis of depressive behaviour. The present findings are consistent in this regard with those of several previous studies (Aronson and Weintraub, 1967; Hinchcliffe et al., 1971; Rosenberry et al., 1969; Lewinsohn et al., 1968; Lewinsohn et al., 1970; Libet and Lewinsohn, 1973; Lewinsohn and Graf, 1973; Libet et al., 1973; Schaeffer amd Lewinsohn, 1971; Ekman and Friesen, 1974) all of which having found evidence for the presence of deviant patterns of verbal or nonverbal communicative behaviour among depressed subjects.

The significant main effect for trial blocks reflects the fact that subjects on the whole were easier to judge on the second 15 trials judged (actually the third of 3 blocks of conditioning trials in session I, since the middle 15 conditioning trials were not videotaped) than on the first 15. This finding may reflect either or both of two processes: (1) the development over time of more readily discriminable changes in facial expressive behaviour on the part of sender subjects and (2) the adaptation of receiver subjects to idiosyncratic nuances in the expressive behaviour of sender subjects over the 30 trials presented per sender. Subjectively, it was clear from viewing the videotapes of session I that the former process was critical. This finding provides evidence, therefore, for the development over time of discriminative responses under the control of the various CS's used in this study.
The significant receivers X senders X trial blocks interaction is somewhat more complex. Inspection of Fig. 6, along with multiple comparison data reveal that this interaction is primarily accounted for by non-D receivers lack of improvement in judgemental accuracy on the second trial block when judging D senders and the differences in judgemental performance between D and non-D receivers. Apparently D receivers were better able to accurately judge D senders on the second block of trials than were non-D receivers. At the same time, D receivers appeared relatively poor at judging PC senders on the second trial block in comparison to non-D receivers. Why this might have occurred is difficult to ascertain. It is possible that D receivers, because of the similarity of their own behaviour to that of other D senders were somehow better able to discriminate subtle cues emitted by these subjects. Perhaps self-observational processes operative while D subjects were themselves going through session I of the experiment contributed toward their being more attuned to the particular types of behaviour likely to be emitted by subjects exhibiting behavioural characteristics similar to their own. At the same time subjects in the PC group, exhibiting behaviour pathology of a different nature may have developed idiosyncratic behaviour patterns over the course of the experiment which resulted in the failure of D receivers to improve judgemental accuracy when judging PC senders during the second trial block.

Altogether, these findings suggest that the hypothesis that depressed persons exhibit a deficit in nonverbal social communicative behaviour may apply specifically to the expressive component of this process as defined in the introduction. The depressive deficit, as it appeared on the experimental task, was a result of the relatively poor performance of all subjects
in correctly identifying the stimulus conditions D subjects were undergoing. It had been expected, on the basis of Ferster's (1973) speculation that depressed persons are characterized by a "limited repertoire of observation" that D subjects in the present study would exhibit relatively poor performance while serving as receiver subjects in session 2. Since, according to this formulation, depressive behaviour is functionally related to a failure of the individual's behaviour to come under the control of environmental discriminative stimuli, in the present study it was expected that D subjects, in comparison to non-D subjects, would be relatively unable to respond appropriately to other subjects' changes in facial expression. That is, D subjects should have exhibited deficient "receptive" social communication. Since no significant differences were found between any of the 3 groups of receiver subjects, the present findings fail to support Ferster's contention. It may have been, however, that the receiver's task in the present study did not adequately relate to what Ferster identified as the "basic behavioural process" underlying the development of a depressive repertoire. Future research might profitably attempt to identify more specifically the conditions under which the physical and social environment exert relatively limited control over the behaviour of depressed individuals.

Category choice analyses. The category choice analyses were conducted in order to evaluate the extent to which consistencies in the tendency to emit particular types of cue on the part of sender subjects, or in the tendency to perceive particular types of cue on the part of receiver subjects played a part in the results obtained. More specifically, an analysis of the nature of this experiment suggests that there are at least two different ways for a given class of sender subject to produce poor performance on the
part of the receiver subjects: (1) by failing to acquire differentiated responses which would allow the receiver to accurately identify the stimulus conditions to which senders were being exposed, or (2) by emitting a particular kind of response with a relatively high frequency, thus producing a consistent, predominantly incorrect manner of responding on the part of the receiver subject. With regard to the latter, for example, it might be suggested that depressed subjects would frequently be likely to emit a particular type of cue that would lead to an observer to say that the depressed person was undergoing "aversive" stimulus conditions.

The results of the category choice analyses were largely negative, and provide little support for the notion that any given class of sender or receiver has a tendency to emit or perceive a particular stereotyped class of nonverbal cue. The analysis of the frequency with which receiver subjects selected the response indicating that sender subjects were undergoing an aversive conditioning trial revealed no significant differences. Thus, it would appear that there was no tendency on the part of any given class of sender subject to predominantly emit cues which receiver subjects in general would label as belonging to a class of responses indicative of aversive trials. Conversely, there was no observable tendency for any particular class of receiver subject to predominantly perceive in others' behaviour cues indicative of aversive trials. The analysis of the frequency with which the "pictorial" response class was chosen resulted in no significant main effects but a significant senders X receivers interaction (Fig. 7) reflecting a tendency on the part of PC receivers to perceive fewer cues than NC or D receivers indicative of the presence of such a trial when observing D senders. No immediate interpretation of this
finding is readily apparent. Impressionistic data suggest one possible reason for this finding. It was noted that several PC receivers while making judgements of D senders spontaneously verbalized distress at being unable to "read" or "get into" the other subject. Comments such as "she's really a stone-face" or "her expression doesn't change" were not uncommon and these subjects were occasionally observed to remind themselves that "there were pictures, too". Perhaps there was a particular ambiguity about the behaviour of D subjects which caused PC subjects to selectively choose the responses indicative of aversive and neutral trials at the expense of choosing the response indicative of the presence of a pictorial trial. It seems likely that, among non-D senders, certain very specific cues controlled the use of the "pictorial" response by receiver subjects. If such specificity of behaviour was infrequent among D subjects, it could account for the relatively infrequent use of the "pictorial" response by PC receivers judging D senders. Why this phenomenon was observed only among PC receivers is difficult to say. The analysis of the frequency with which the "neutral" response choice was used revealed a significant main effect for the trial blocks variable, reflecting a reduced frequency of the choice of this response alternative during the second block of 15 trials relative to the first. This finding undoubtedly reflects the development of more differentiated patterns of response on the part of the sender subjects from the earlier to the later trials of the experiment. Since during the first block of 15 trials sender subjects had not had time to develop appropriate CR's it is likely that their behaviour was relatively diffuse relative to their behaviour on the second trial block, while by the second trial block they would have had sufficient experience with the experimental situation
to have acquired relatively more specific response patterns. The relatively nonspecific behaviour emitted by these subjects during the first trial block was probably more likely to provoke the choice of "neutral" response alternative on the part of receiver subjects. As senders' responses became more differentiable toward the later trials, receiver subjects would have been more likely to choose response alternatives appropriate to the other two types of conditioning trial.

The category choice analyses, considered in conjunction with the judgemental accuracy data suggest that the evident deficit among depressives in the emission of nonverbal cues which may be discriminated by others results not from a tendency to emit a particular class of behaviour to an excessive degree. Rather, it seems that the depressive may serve as an ambiguous social stimulus whose emission of behaviour is not differentiated enough to control the behaviour of others. The likely social consequences of such a deficit fit in quite well with current behavioural theories of depression. Given that the depression-prone person is characterized by a relative deficit in the extent to which she emits behaviour which others can identify and respond discriminatively to, much of her behaviour in social situations is likely to "miss the mark". In situations in which a particular behaviour or set of behaviours emitted by another person might be reinforcing, the depression-prone person's behaviour because of its ineffectiveness in controlling others' behaviour will fail to produce the reinforcing consequence. In situations where aversive contingencies prevail, the depression-prone person's behaviour is likely to be ineffective in removing the aversive state of affairs. Thus the individual may experience a low rate of positive reinforcement in addition
to being exposed to an aversive, punitive environment. Further probable sequelae, resulting from the effects of such a deficit in expressive social communication are identifiable. It has been noted, that, through the process under discussion, the depression-prone individual is likely to (a) fail to obtain positive reinforcement and (b) fail to avoid aversive experiences through engaging in interpersonal behaviours. The former defines the basic characteristic of an extinction schedule, while the latter effectively defines a punishment paradigm; both being applied to attempts to engage in interpersonal behaviour. Thus, the individual exposed to such contingencies could be expected to engage in fewer and fewer interpersonal behaviours with the effect of producing further social isolation. Clinical descriptions of depressed individuals and factor analytic research have devoted a great deal of attention to what has been termed the depressive's "loss of interest in the social environment" (Beck. 1967; Costello, 1972; Grinker et al., 1961). Attributing the restricted range of interpersonal contacts and variety of interpersonal behaviour to such an internal process as "loss of interest" seems to miss the point that the behaviours implied by such a term are related to the contingencies of the social environment of which they are a function, and that the likelihood of being exposed to such contingencies may be to a significant extent related to the deficit in expressive social communication observed in the present study.

Although these data seem clearly supportive of the hypothesized social communication deficit, alternative explanations are possible. Although the present findings have been applied to a partial formulation of the etiology of depression, and the data are consistent with this for-
mulation, the methodology employed was inadequate for the purpose of drawing any unequivocal conclusions about the etiological significance of the present findings. The information obtained in the present study was correlational in the sense that a deficit was observed in a group of people defined as being depressed relative to groups of people who were not. It is possible that this deficit may be a consequence of a central depressive "disease state", or of engaging in depressive behaviour. If this were a more adequate interpretation of the present findings, two alternative conclusions would be possible: (a) a deficit in expressive social communication has little, if any, relevance to the etiology of depression, or (b) a deficit in the expressive social communication has little relevance to the origin of depression, but may serve as a factor in the maintenance of the disorders. In the case of the latter, it could be assumed that the same unknown factors which are responsible for the production of depression are also responsible for the production of a deficit in social communication skills. It would then be possible to argue that this superimposition of a communication deficit atop an already present depressive disorder compounds the depressive's problems by reducing reinforcement and increasing aversive experiences and social isolation and thus either lengthens or makes more severe the depressive episode. It might be possible to evaluate the etiological significance of a deficit in expressive social communication by modifying the present paradigm with the inclusion of a group of "remitted" depressives, or retesting a group of "formerly ill" depressives at some specified point after an apparent improvement in their clinical condition. Another strategy would be to select a group of subjects at "high risk" for depression and, utilizing the appropriate control groups evaluate whether
or not high risk-poor communication subjects are more susceptible to episodes of clinical depression than high risk-good communication subjects.

Performance and predictions of performance.

Clinical descriptions of depressed patients heavily emphasize the high frequency of pessimistic statements to be observed among them and often note that such patients tend to approach situations with what has been termed a "defeatist attitude". Such notions have been incorporated into certain cognitive theories of depression, particularly that of Beck (1967). As noted in Chapter 1, Beck's theory considers depression to be a disorder of thinking, characterized by a "primary triad" of cognitive schemata consisting of a negative conception of the self, the external world, and the future. Research relevant to this formulation has employed subjects' self-ratings of probability of success and level of aspiration while manipulating success and failure on experimental tasks. In general, findings from this line of research have been consistent with the conclusions that depressed subjects tend to be relatively pessimistic with regard to their perceived probability of success, tend to exhibit level of aspiration ratings which are similar to those of non-depressed subjects, and tend to evaluate their performance more negatively than non-depressed subjects (Loeb et al., 1967; Loeb et al., 1971).

It will be recalled that in the present study, all subjects serving in the receiver condition of session 2 were required to make predictions regarding the number of correct judgements they would make on each set of 30 videotaped excerpts of each of 3 sender subjects presented to them. The data thus acquired may be seen to be roughly analogous to the probability of success estimates used by Loeb and his colleagues. Analysis of variance
performed upon these data revealed no significant differences as a function of diagnostic groups or ordinal position of prediction. Thus, to the extent that the predictions of performance used in the present study may be said to be a similar dependent variable to Loeb et al.'s probability of success estimates, the present findings failed to replicate those of the previous studies.

Further correlational analyses were performed on these data in an attempt to test some predictions derived from Seligman's (1972, 1975) theory of depression. According to Seligman's formulation, depression occurs when a person may be said to have acquired "learned helplessness". Whatever the origins of this phenomenon, its critical feature is said to be the individual's "expectation" that behaviour and its consequences are independent, noncontingent events. Once this "expectancy" has been acquired, the various phenomena of depression follow. As noted in Chapter 1, a test of several predictions of Seligman's theory (Miller and Seligman, 1973) found that depressed subjects showed less change in ratings of their probability of success on a "skill" task than did nondepressed subjects. From Seligman's position it would seem to follow that relative to nondepressed subjects, depressed subjects' ratings of their expectancies for success at a given task should be random, and should remain relatively unaffected by feedback regarding their performance. With regard to the present study, it would be predicted from Seligman's theory that depressed subjects' predictions of their own performance should remain stable over trials, and that changes in predictions should not be significantly correlated with the discrepancy between their predictions and their actual performance on previous trials. As can be seen in Table 7, the results of the present study are strongly
disconfirmatory with respect to this hypothesis. Difference scores reflecting changes in subjects' predictions of their performance were, for the most part, significantly correlated with difference scores reflecting the discrepancy between subjects' predictions and their actual performance and these correlations were of a large magnitude for depressed subjects. Thus, depressed subjects' predictions of their own performance showed systematic changes in the direction of more closely approximating their actual performance on previous trials -- a finding exactly opposite to what would be expected from Seligman's theory. It would seem therefore that depressed individuals do not always behave as if their behaviour and its outcome were independent.

Psychophysiological data.

Skin conductance. SC values increased significantly as a function of increased exposure to the experimental situation during the classical conditioning phase of this study. This finding was unexpected in that level of SC activity is commonly interpreted as reflecting level of arousal and, as such, should have evidenced habituation during the latter trials.

The significant main effect for the type of conditioning trial variable was partially consistent with experimental expectations. SC values were generally elevated on aversive conditioning trials thus confirming the provocative qualities of the type of stimulation employed in this study. On the other hand, SC values on pictorial trials were in general indistinguishable from those on neutral trials. Discussion of this finding in addition to the finding of a significant linear trend component for SC values may be more fruitfully considered in relation to several of the interaction effects observed and therefore will be postponed until these.
interactions are considered.

Initially, those interaction terms about which no a priori predictions had been made will be discussed. These two interactions -- trial block X scoring intervals and type of conditioning trial X trial blocks X scoring intervals -- are best treated together since the patterns of SC change observed in the two-way interaction seem to be accounted for by those patterns of change observed in the third-order interaction. For the interaction between trial blocks and scoring intervals, a general increase in SC values was observed between the earlier and later scoring intervals. This pattern was most marked during the first trial block, and became less pronounced on the second and third trial blocks. Analysis of the 3-way conditioning trial X trial blocks X scoring intervals interaction shown in Fig. 11, reveals that this significant increase over scoring intervals is primarily due to the effects of aversive stimulation. From Fig.11 it can be seen that little change in SC values occurs over scoring intervals on neutral and pictorial conditioning trials. However, on aversive trials, a significant increase in SC values is observed from the early to the later scoring intervals.

Orthogonal analyses of the interaction between type of conditioning trial and the linear trend of SC change over scoring intervals confirmed in general what was specifically observed in the 3-way type of conditioning trial X trial block X scoring intervals interaction. As can be seen in Fig. 9 , an increasing linear trend of SC values was found on aversive conditioning trials, but not on pictorial or neutral conditioning trials. Thus, only on aversive conditioning trials was a conditioning-like phenomenon observed in SC values.
The pattern of SC change on aversive conditioning trials is the typical pattern observed in trace conditioning procedures using SC as a dependent variable. It had been expected, however, on the basis of a study reported by Corah and Tomkiewicz (1971) that a similar pattern of SC change would be observed on pictorial conditioning trials. The pictorial stimuli used in this study were selected on the basis of their similarity to the stimuli used by Corah and Tomkiewicz. This study failed to replicate these authors' finding that electrodermal conditioning can be produced through the use of pictorial stimuli of moderate "interest" value. Several reasons for this failure to replicate may be proposed. One possible reason may be that the pictorial stimuli used in this study were not of sufficient "interest" value to produce any consistent effect on electrodermal responding. Corah and Tomkiewicz had their subjects rate the interest value of the slides employed prior to taking part in the study. On the basis of these ratings, pictorial stimuli were chosen which had been found to have some degree of interest value for the sample of subjects they employed in their study. A second reason for the discrepancy between the findings of this study and those of Corah and Tomkiewicz may relate to differences in subject samples. Corah and Tomkiewicz' sample consisted of female college students with a mean age lower than that of any of the groups in the present study. It may have been that the type of stimulation employed was differentially effective in producing changes in electrodermal responding for the different subject samples involved. A third possible explanation for the discrepancy between the two studies relates to contextual differences in the paradigms employed. Corah and Tomkiewicz' procedure involved the comparison of two different types of trial (pictorial and
neutral) while the present study employed three (pictorial, neutral and aversive). It may have been that presentation of pictorial conditioning trials within the same context of and relatively recent continuity with aversive conditioning trials resulted in the failure to observe a pattern of SC change that might otherwise have occurred. An alternative interpretation might be that in the type of conditioning procedure employed in this study and that of Corah and Tomkiewicz, conditioning-like SC changes are most likely to occur on those trials employing the most intense stimulation. One final point worthy of emphasis is that while the pictorial conditioning trials in this study did not produce any reliable SC changes, this does not necessarily mean that this form of stimulation was not effective in producing reliable physiologic or behavioural effects. Clearly, the fact that subjects were able to accurately identify pictorial trials at a better than chance level during the judgemental task of session 2 strongly indicates that reliable behavioural changes occurred during pictorial conditioning trials. Evidence to be discussed later suggests that other physiological effects occurred on pictorial trials.

Of greatest interest for the purposes of the present study was the finding of a significant interaction between diagnostic groups, type of conditioning trial and scoring intervals. Orthogonal analyses of this interaction revealed that neither the NC and PC groups, nor the pictorial and neutral conditioning trials differed in SC values over the 5 scoring intervals. Inspection of Fig.10 reveals that what change in SC values occurred took place on aversive conditioning trials, and the pattern and magnitude of SC change differed as a function of diagnostic groups. Thus the D group exhibited a greater magnitude of change in SC values from scoring
interval 3 to scoring interval 4 on aversive conditioning trials than did the combined control groups. Thus the D group exhibited a greater increase in SC values during the presentation of the aversive UCS than did the NC and PC groups. Additionally, rather than observing a decrease in the SC values during the post-stimulation interval as was seen in the PC and NC groups, D group subjects exhibited further increases in SC during this period.

These results are consistent with those of Lewinsohn, Lobitz and Wilson (1973), who reported that depressed college students, relative to psychiatric and normal controls exhibited greater electrodermal response to aversive (electric shock) stimulation in a non-differential classical conditioning paradigm. Unlike the results reported by Lewinsohn et al., in the present study, SC values continued to rise among D subjects during the post-stimulation interval, while a partial return to prestimulation SC levels was observed among PC and NC subjects. Together with those of Lewinsohn et al., the findings of the present study are largely inconsistent with those of other studies dealing with the electrodermal activity of depressed subjects during classical conditioning procedures. As reported in Chapter 3, Ban et al. (1966) reported differences between depressives and normals in the direction of diminished responsivity among members of the former group in UCR amplitude. Similarly, Dawson et al. (1974) reported decreased UCR magnitude among depressed subjects compared with a matched group of normals.

Given the inconsistencies between these studies, interpretive statements regarding the meaning of the findings obtained in the present study are hazardous. It may be, as Lewinsohn (1974a) suggests, that aversive stimulation produces a greater autonomic (electrodermal) response
among other selected diagnostic groups, but the inconsistent findings reviewed above argue against uncritical acceptance of this hypothesis. The inconsistencies observed between these studies might reflect differences in some of the characteristics of the various samples of depressives utilized. In the Ban et al. study, depressed subjects were differentiated into 3 groups: "neurotic", "endogenous", and "schizo-affective", while in the Dawson et al. study all depressed subjects were diagnosed as suffering from a "primary monopolar depressive illness", but, more importantly, all had been recommended for ECT. Lewinsohn et al.'s subjects, on the other hand, were all unhospitalized college students, while in the present study a wide variety of patients participated; ranging from outpatients to one who was being considered for ECT. It is possible that the discrepant findings of these studies might reflect the operation of a severity of depression variables with the former two studies employing more severely depressed subjects, while the present study and that of Lewinsohn et al. might have used less severely depressed subjects. In this regard, it might be noted that a severity or chronicity variable has been profitably applied to the analysis of discrepant findings in studies of electrodermal behaviour among schizophrenic subjects (DePue and Fowles, 1973).

Heart rate. Subjects in the D group were found to exhibit a greater mean HR than did subjects in either the NC or PC groups, replicating the findings of Kelly and Walter (1968), McCarron (1973) and Dawson et al. (1974).

With regard to the psychophysiological mechanisms that might be reflected in this finding, it need hardly be noted that heart data are commonly taken to reflect the effects of conditions referred to as "stressful". Similarly, a common interpretation of between-groups differences in tonic
HR level suggests that the differences observed reflect disparate levels of "arousal". In accordance with this interpretation, it might be suggested on the basis of the present findings that depressives in general tend to be physiologically hyperaroused. What the implications of such a conclusion for an explanation of the origins and nature of depressive behaviour might be, however, are unclear. Elevated tonic HR levels have been found to characterize several clinical groups, including schizophrenics (Buss, 1966; Venables, 1966) and anxiety neurotics (Martin and Sroufe, 1970). Whether these findings reflect the operation of the same process in all these groups or different processes producing the same ultimate psychophysiological effect in each specific group is not known. It must be pointed out, further, that interpretations of psychophysiological data as reflecting differences in level of arousal rest upon the general utility concept of arousal. This concept has come under attack in recent years for being vague and failing to account for the complexities of psychophysiological response patterns (Lacey, 1967; Lacey and Lacey, 1970, 1974). Thus, the heuristic value of concluding that depressives in general tend to be hyperaroused is questionable.

Lacey's (1967; Lacey and Lacey, 1970, 1974) speculations with respect to the meaning and significance of variations in HR provide the basis for an alternative means of accounting for the differences observed in the present study. This position argues that HR levels reflect a dimension of "acceptance" or "rejection" of environmental information by the organism. Elevations in HR according to this formulation are indicative of environmental rejection, while decreased HR values reflect "environmental intake" or "attention". Thus, it might be argued that D subjects' elevated tonic HR level
reflects an active process of "rejection of the environment". Such a position has some appeal in that it corresponds with informal descriptions of depressed patients as "tuning out" the environment. Further, certain theoretical positions on depression such as Lazarus' (1972) notion that the depressive exhibits elevated sensory thresholds, and Ferster's (1973) hypothesis that the depressive is a poor observer of the environment seem to be pointing toward something similar. The validity of such an explanation, however, depends upon the validity of Lacey's hypothesis, a hypothesis which has been subjected to a number of lengthy criticisms (Elliot, 1972, 1974; Hahn, 1973).

One alternative to Lacey's explanation of the phenomenon of HR acceleration-deceleration is particularly apropos here. Obrist, Webb, Sutterer, and Howard (1970) have argued that HR variation represents a response to variations in the organism's energy demands such that HR elevation occurs when increased somatic activity requires extra energy mobilization. In this regard, the most consistent psychophysiological finding among depressed patients is that of elevated EMG levels. Thus, the finding of elevated tonic HR level among depressed patients could simply be a by-product of elevated somatic (EMG) activity.

One possibly important implication of the present finding of elevated tonic HR among depressives relates to the possibility of using HR measures as indices of therapeutic change in depression. If tonic HR level is an important correlate of depression, changes in depressive behaviour, brought about as the result of some effective form of therapeutic intervention might be reflected in changes in tonic HR level. Furthermore, to the extent that deviant psychophysiological response patterns are a component of
depressive disorders, therapeutic change could potentially be affected through the use of biofeedback procedures. Such a strategy seems rather indirect, however.

Orthogonal analysis of the type of conditioning trial variable revealed that mean HR on aversive conditioning trials was greater than that on pictorial and neutral trials. As with the SC data, these results are at least partially in accordance with experimental expectations, and again confirm the provocative properties of the aversive stimulation employed. It had been expected, on the basis of Lacey's speculations regarding the pattern of HR activity among subjects exposed to stimulation of some "interest value" that HR values on pictorial conditioning trials might be significantly lower than on aversive or neutral trials. This prediction failed to be confirmed when considering the overall main effect of the type of conditioning trial. It must be pointed out, however, that this main effect term was obtained by collapsing over 13 scoring intervals, 3 classes of subject, and 3 trial blocks; thus failing to account for a large amount of variance taken up by these factors. Analyses of interactions between the type of conditioning trial variable and the other 3 variables suggest that the anticipated "Lacey effect" may be present, though attenuated, in the data.

The significant main effect for the scoring intervals variable and the significant trial blocks X scoring intervals interaction both reflect decreasing HR values during later scoring intervals. This pattern of HR deceleration became more marked during later trial blocks. Thus, increasing experience in the conditioning situation was accompanied by greater HR deceleration during later scoring intervals. Since the deceleration in-
variably occurred during the UCS and poststimulation intervals, this phenomenon seems related to the end of anticipatory sequence and may reflect decreasing arousal, or a decrease in somatic energy demands (Obrist et al., 1970).

The significant four-factor interaction between diagnostic groups, type of conditioning trial, trial-blocks, and scoring intervals, though highly complex, seems amenable to interpretation. As can be seen in Fig. and as confirmed by post hoc analysis, HR levels for D subjects were significantly higher than they were for NC and PC subjects at all levels of the type of conditioning trial, trial-blocks, and scoring intervals variables. Furthermore, as is evident from Fig.12, the greatest interval-to-interval variability in HR scores occurred during aversive conditioning trials. During the first block of aversive conditioning trials, NC subjects exhibited an elevated mean HR during the first prestimulation interval that dropped out on the following two trial blocks. During the second trial block, an anticipatory HR increment is evident during the first 3 intervals of CS presentation, and thereafter a decrement beginning during the last interval of CS presentation and continuing to the last poststimulation interval was observed. During the third trial-block a HR increment occurred during the second prestimulation interval and continued until the second interval of CS presentation, after which a decrement was observed. While post-hoc analysis revealed no significant differences between scoring intervals on aversive conditioning trials for PC subjects, inspection of Fig. reveals roughly similar patterns of HR change to those observed in NC subjects, with nonsignificant increments in HR values occurring during the period of CS presentation. No such pattern is readily discernible for D subjects on the 3 blocks of aversive conditioning trials. These data on HR
change demonstrate that for NC subjects, and possibly for PC subjects, a pattern of initial HR acceleration, then deceleration prior to UCS onset characterized HR response on aversive conditioning trials. This is consistent with the pattern often observed in studies of HR conditioning (Dronsejko, 1973). Why the response should have occurred as early as the second prestimulation interval during trial-block 3 for NC subjects is problematical in that, at that point in the conditioning sequence, no external cue reliably associated with UCS-A onset was presented. While this could simply reflect random variance, since there were only 3 different types of conditioning trial, it is possible that subjects, having been exposed to prior pictorial and neutral trials, were responding to the implicit CS involved in the contingency that 1/3 of all trials were of an aversive nature. This post-hoc interpretation is obviously highly speculative.

On pictorial conditioning trials, HR values remained fairly stable over the first 11 scoring intervals during all 3 trial blocks for PC subjects. However, during the second and third trial blocks, a decelerative response occurred during the first poststimulation interval (12th scoring interval). Although for NC subjects there were no significant differences between any of the scoring intervals within each trial block on post-hoc tests, investigation of Fig. 12 reveals a roughly similar pattern of HR deceleration occurring during later scoring intervals on the second and third trial blocks. D subjects undergoing pictorial conditioning trials exhibited almost identical HR scores during all 13 scoring intervals. These data demonstrate the occurrence of a decelerative HR response during a period of time roughly contiguous with the presentation of pictorial stimulation among PC subjects. In addition, it appears possible that a similar pattern
occurred among NC subjects in spite of the fact that post-hoc statistical comparisons failed to show that this pattern was statistically significant. These observations suggest that a "Lacey effect" of HR deceleration in the presence of environmental stimuli having "interest value" occurred among PC subjects, possibly occurred among NC subjects, but did not occur at all among D subjects. Unfortunately, the present data do not provide any evidence at all for the development of conditional HR change to stimuli reliably preceding the presentation of pictorial stimuli.

The only significant HR change observed on neutral trials was the occurrence of an accelerative HR "spike" during the ninth scoring interval of the first trial block for NC subjects. This spike is difficult to interpret in light of theoretical expectations on aspects of the experimental situation which might have elicited it. From Fig. 12 it can be seen that nothing resembling this spike was observable during later trial blocks or on any other trial block for the other two groups of subjects. The only other HR changes of similar magnitude that occurred in the experiment were observed among NC subjects during aversive conditioning trials. The only apparent interpretation of this phenomenon takes into account the context within which neutral trials were presented. It may have been that during early trials, NC subjects had not acquired fully discriminated patterns of HR responding. If these subjects had failed to acquire a discrimination between aversive and neutral trials at this point in time, this spike may have been an accelerative response in anticipation of the presentation of an aversive UCS. This interpretation, however, is highly speculative.

Taken together, the psychophysiological data from this study lead to a number of conclusions. The electrodermal data from the present study and
that of Lewinsohn et al. (1973) provide disconfirmatory evidence for Lazarus' (1968, 1972) hypothesis that depressed persons may be characterized as "relatively refractory to most forms of stimulation". While the data of Ban et al. (1966) and Dawson et al. (1974) may be used to argue that Lazarus' description may sometimes be the case, clearly such a generalization is unwarranted on the basis of the present findings. With regard to the HR data, the 4-factor interaction seems to reflect the development over time of consistent accelerative and decelerative response patterns on aversive and pictorial conditioning trials respectively but no consistent pattern of HR change for D subjects. Although this finding could be taken as support for Lazarus' hypothesis, it seems more likely that the failure to observe any consistent patterns of HR change among D subjects simply reflects the operation of a "ceiling effect". In fact the finding of a significantly elevated tonic HR level among depressed subjects also seems inconsistent with Lazarus.

Psychometric data: Self-monitoring of expressive behaviour.

Prior to participation in the experiment, all subjects completed Snyder's (1975) scale assessing the self-monitoring of expressive behaviour (SM). This scale was developed in order to assess individual differences in the extent to which people are able to "...observe and control their expressive behaviour and self-presentation" (Snyder, 1975, p.527 ). Studies of the psychometric characteristics of the scale demonstrated internal consistency, temporal reliability, and discriminant validity, while a series of other studies provided good support for its construct validity. Conceptually, it appeared that the construct putatively measured by this scale should be related to various aspects of subject's performance on the
experimental task in the present study. If people scoring high on the SM scale demonstrate better control over their expressive behaviour and a greater ability to use this control in order to effectively manage the impact they have on others than subjects scoring low on the scale, it would seem reasonable that SM scores should be significantly correlated with (at least) subjects' performance as senders in the present study, and possibly with their performance as receivers. Moreover, if, as was hypothesized in the present study, depressives are deficient in the "ability" to emit or perceive expressive behaviours controlled by various stimulus conditions, they could be expected to score lower on the SM scale than other groups not so deficient. This expectation was strengthened by Snyder's (1975) finding that a mixed group of psychiatric patients scored significantly lower, while a group of theatre actors scored significantly higher than a normative sample of university students on the SM scale.

In regard to these proposals, the present findings were completely negative. Between-group comparisons of SM scores by analysis of variance revealed no significant differences, while correlations between subjects' SM scores and composite scores reflecting subjects' performance as senders and receivers revealed no significant relationships.

A variety of reasons for this failure to find either significant between-group differences or significant correlations between the SM scores and subjects' experimental performance can be suggested. It is possible that the construct putatively measured by the SM involves the operation of processes entirely independent of those involved in the present experimental situation. Alternatively, the present findings could be taken as simply another demonstration of the often found poor relationship
between personality tests designed to measure hypothetical internal traits and actual behavioural performances, variations in which should reflect the operation of similar processes (cf. Mischel, 1968). A third interpretation of these data suggests that the failure to find any significant relationships can be attributed to sampling characteristics. The subjects with which Snyder performed his reliability and validity studies were, for the most part, drawn from samples with characteristics quite markedly different from those of subjects employed in the present study. Snyder's subjects were primarily drawn from college populations, and the only study using somewhat different subjects employed theatrical actors and psychiatric patients; all such groups, with the exception of the latter, varying in a number of important respects from the groups used in the present study. The possibility that sampling characteristics largely accounted for the results obtained in the present study gains credence when the mean scores for the two psychiatric groups employed in the present study (PC $\bar{X} = 10.20$; D $\bar{X} = 10.60$) are compared to the mean score of Snyder's psychiatric group ($\bar{X} = 10.19$). Comparison of these values with the mean SM score for the NC group in the study ($\bar{X} = 9.60$) shows that all these values are quite similar, while comparisons with the means of Snyder's actor sample ($\bar{X} = 18.41$) suggests that sampling differences played an important role in the disparities between the two studies.

As an alternative to all these speculations, it could also be argued that the present findings call into question the generalizability and clinical meaningfulness of Snyder's findings. In the absence of further data, it is difficult to favour one explanation over another.
Methodological and interpretive problems.

This section deals with difficulties in experimental design which provide potential sources of bias and restrict the generalizability of the present study and discusses interpretational issues arising from uncontrolled aspects of the study.

The first question to be dealt with relates to the potential confounding of depressed and nondepressed psychiatric groups with inpatient-outpatient status. It will be recalled that in the present study, the bulk (8/10) of subjects included in group D were inpatients, while most (7/10) of the PC subjects were outpatients. It could be argued that the differences observed between group PC and D on judgemental and physiological data could just as likely be an artifact of inpatient-outpatient status as a result of depressive status. In an attempt to clarify this issue, inpatients and outpatients within the D and PC groups were contrasted in terms of their composite "sender" scores. These means are presented in Table 10 where it can be seen that within group PC, inpatient subjects performed somewhat better than did outpatients, whereas within group D, outpatients performed better than inpatients. Between diagnostic categories, however, D subjects, regardless of whether they were inpatients or outpatients performed more poorly than did PC subjects. Thus, taking into account the inpatient-outpatient status of subjects within each group still shows that D subjects performed more poorly as senders than did PC subjects, and supports the position that the differences observed are at least partly a function of depressive status.

Another major source of difficulty in the present study relates to
Table 10. Mean composite sender scores for PC and D outpatients and inpatients.

<table>
<thead>
<tr>
<th></th>
<th>Outpatients</th>
<th>Inpatients</th>
</tr>
</thead>
<tbody>
<tr>
<td>PC</td>
<td>17.48</td>
<td>17.55</td>
</tr>
<tr>
<td>D</td>
<td>16.17</td>
<td>12.13</td>
</tr>
</tbody>
</table>
the fact that patients' medication regimens were not controlled. Psychoactive medications could reasonably be expected to affect most, if not all, of the dependent variables in this study. Thus, it is clearly possible that this uncontrolled source of bias could have affected the data in unknown ways. Further, there do not appear to be any data that might clarify the contribution of medications to the results obtained. The findings of this study are therefore open to criticism from this angle.
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Appendix A. Snyder's (1975) Self-Monitoring Scale

Personal Reaction Inventory

The statements on the following pages concern your personal reactions to a number of different situations. No two statements are exactly alike, so consider each statement carefully before answering. If a statement is TRUE or MOSTLY TRUE as applied to you, blacken the space marked T on the answer sheet. If a statement if FALSE or NOT USUALLY TRUE as applied to you, blacken the space marked F. Do not put your answers on this test booklet itself.

It is important that you answer as frankly and as honestly as you can. Your answers will be kept in the strictest confidence.

1. I find it hard to imitate the behavior of other people.
2. My behavior is usually an expression of my true inner feelings, attitudes, and beliefs.
3. At parties and social gatherings, I do not attempt to do or say things that others will like.
4. I can only argue for ideas which I already believe.
5. I can make impromptu speeches even on topics about which I have almost no information.
6. I guess I put on a show to impress or entertain people.
7. When I am uncertain how to act in a social situation, I look to the behavior of others for cues.
8. I would probably make a good actor.
9. I rarely need the advice of my friends to choose movies, books or music.
10. I sometimes appear to others to be experiencing deeper emotions than I actually am.
11. I laugh more when I watch a comedy with others than when alone.
12. In a group of people I am rarely the centre of attention.
13. In different situations and with different people, I often act like very different persons.
14. I am not particularly good at making other people like me.
15. Even if I am not enjoying myself, I often pretend to be having a good time.
16. I'm not always the person I appear to be.
17. I would not change my opinions (or the way I do things) in order to please someone else or win their favour.
18. I have considered being an entertainer.
19. In order to get along and be liked, I tend to be what people expect me to be rather than anything else.
20. I have never been good at games like charades or improvisational acting.
21. I have trouble changing my behaviour to suit different people and different situations.
22. At a party I let others keep the jokes and stories going.
23. I feel a bit awkward in company and do not show up quite so well as I should.
24. I can look anyone in the eye and tell a lie with a straight face (if for a right end).
25. I may deceive people by being friendly when I really dislike them.
Appendix B. Post-hoc analysis of the 4-factor heart-rate interaction

This appendix describes the results of the post-hoc analyses of the significant four-factor HR interaction. All post hoc comparisons were performed using Cichetti's (1972) modification of the Tukey HSD procedure.

The results of these analyses will be described first in terms of the significance of differences in mean HR for each level of the groups, type of conditioning trial, and trial blocks variables. During the first trial block on aversive conditioning trials, NC subjects significantly elevated HR during the first 2 second scoring interval (corresponding to the first of two pre-stimulation intervals) relative to all other scoring intervals. This HR increment occurred during CS presentation. On the third aversive conditioning trial block for NC subjects, a significant HR increment occurred during the second prestimulation interval and was maintained as far as the second interval of Cs presentation.

For both PC and D subjects no significant differences in mean HR were observed between any of the 13 scoring intervals on the three trial blocks during aversive conditioning trials.

During conditioning trials on which a pictorial UCS was presented, there were no significant HR changes for NC and D subjects on any of the three trial blocks. For PC subjects, however, a significant decrement in HR was observed during the 12th (first prestimulation) scoring interval.
on the second and third blocks of conditioning trials on which a pictorial UCS was presented.

On neutral conditioning trials, the only significant effect noted was an increase in HR during the 9th scoring interval (last interval of CS presentation) on the first trial block for NC subjects.

Differences between mean HR on each of the three trial blocks were then compared within diagnostic groups, type of conditioning trial and scoring intervals. This analysis will be described first for NC subjects undergoing aversive conditioning trials. During the first (first prestimulation) scoring interval, HR was significantly greater during the first trial block than on the second and third which did not differ from one another. This pattern then reversed itself, so that on the second (second prestimulation) scoring intervals, mean HR on trial block 3 was significantly greater than on trial blocks 1 and 2 which did not differ. During the 6th and 7th scoring interval, HR was significantly greater on trial blocks 2 and 3 than on trial block 1. During both these intervals, HR values on trial blocks 2 and 3 did not differ significantly from one another. During the 8th (third during CS presentation) scoring interval, HR values during the second trial block were greater than on the first and third which did not differ. During the 9th and 10th (last during CS presentation and first during UCS presentation) scoring intervals, there were no significant differences in HR on any of the three trials. During the 11th (second during UCS presentation) scoring interval, HR was significantly greater on the first trial block than on the second and third trial blocks which did not differ. During two poststimulation scoring intervals there were no significant differences between any of the trial blocks.
For PC subjects undergoing the aversive conditioning phase of the experiment there were no significant HR differences between the three trial blocks during intervals 1 (first presentation), 3 (first during WS presentation), 6, 7, 8, 9 (all intervals during CS presentation), 12 and 13 (both poststimulation). During interval 2 (second prestimulation), HR was significantly higher on the first trial block than on the second two trial blocks (which did not differ). During intervals 4 and 5 (second and third during WS presentation), HR was significantly greater on trial block 1 than on trial block 3, but trial block 1 did not differ from trial block 2, nor did trial block 2 differ from trial block 3. During interval 10 (first during UCS presentation), HR was significantly higher on trial block 2 than on trial block 3, while trial blocks 2 and 1 and 1 and 3 did not differ. During interval 11 (second during UCS presentation) trial block 1 was significantly higher than trial block 3 while trial blocks 2 and 3 and 1 and 2 did not differ from one another.

For D subjects undergoing the aversive conditioning phase of the experiment the only significant HR difference over the three trial blocks was observed during the 12th (first poststimulation) interval where HR on trial block 1 was significantly greater than on trial blocks 2 and 3 (which did not differ).

No significant HR differences between the three trial blocks were found for any of the three diagnostic groups during conditioning trials on which a pictorial UCS was presented. On neutral conditioning trials, the only between-trial-blocks difference found was a significantly greater HR on trial block 1 than on trial blocks 2 and 3 (which did not differ) during the 9th scoring interval for NC subjects.
The next set of multiple comparisons examined differences in HR between the three different types of conditioning trials within each of the 3 diagnostics groups, trial blocks, and 13 scoring intervals. For NC subjects during the first trial block, there were no significant HR differences between the three different types of conditioning trial during intervals 2 (second prestimulation), 3, 4, 5 (all intervals during WS presentation), 6, 7, 8 (first through third intervals during CS presentation), 10, 11 (both intervals during UCS presentation), 12 and 13 (both poststimulation). During the first scoring interval, HR was significantly greater on aversive conditioning trials than on pictorial and neutral trials (which did not differ). During the 9th scoring interval, HR was greater on neutral than on aversive and pictorial trials (which did not differ).

For NC subjects during the second trial block, the only significant differences noted occurred in intervals 6, 7, and 8 (first 3 intervals during which CS was presented), in all of which HR was greater on aversive than on pictorial and neutral trials (which did not differ). During trial block 3, NC subjects' mean HR was greater on aversive than on pictorial and neutral trials for intervals 2 (last prestimulation), 3, 4, 5 (all intervals during WS presentation), 6 and 7 (first two intervals during which CS presented). During all other intervals there were no significant differences.

For PC subjects, during the first trial block, there were no significant differences between the 3 different types of conditioning trial. During the second trial block, the only significant difference occurred during interval 12 (first poststimulation) in which HR was higher on both aversive and neutral trials than on pictorial trials. The first two types of conditioning trial did not differ. During the third trial block, the only signifi-
cant difference occurred in the third interval where HR was higher on pictorial and neutral trials than on aversive trials, the pictorial and neutral trials, again, failing to differ,

For D subjects there were no significant differences between types of conditioning trial during any scoring interval in any of the three trial blocks.

Finally, HR differences between the three diagnostic groups were evaluated within each type of conditioning trial, trial block and scoring interval. Most noteworthy was the fact that, at all levels of the latter three variables, D subjects' HR was significantly greater than that for the NC and PC groups. On the first aversive conditioning trial block, NC subjects had a greater HR during interval 1 (first prestimulation) than did PC subjects, while the reverse was true during interval 2 (second prestimulation) no further significant differences between groups NC and PC occurred during intervals 3 through 13. During the second aversive conditioning trial block, groups NC and PC did not differ except at intervals 6 and 7 (first and second intervals during which CS was presented) wherein NC subjects' HR was greater than that for PC subjects. During the third trial block, NC subjects' HR exceeded PC subjects during intervals 2 (second prestimulation), 3, 4, 5, (WS presentation), 6, 7, and 8 (first three intervals during CS presentation).

During the first trial block on which a pictorial UCS was presented, the only significant difference between groups NC and PC occurred during interval 13 (last poststimulation), where NC subjects' HR exceeded that of PC subjects. No other significant differences occurred between NC and PC subjects during any scoring interval in the second and third blocks of trials on which a pictorial UCS was presented.
On neutral conditioning trials, the only significant difference between NC and PC subjects occurred during the 9th (last interval during which CS presented) scoring interval in the first block of trials, where NC subjects' HR exceeded that of PC subjects.