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DEPRESSION AND LEARNED HELPLESSNESS

Y.P. ARVANITIS

M.Phil. 1985

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DEPRESSION AND LEARNED HELPLESSNESS

A PSYCHOPHYSIOLOGICAL COMPARISON

A thesis submitted to the
Council for National Academic Awards
in partial fulfilment of the requirements for the
Master of Philosophy degree in Psychology

by

YANNI PHILIPPOU ARVANITIS

October 1985

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To my parents, Philippos and Stella, and to my sister, Amalia, this piece of work is dedicated.

ACKNOWLEDGEMENTS

If this thesis is of any value at all, it is due to the patient guidance of Susan Grey who tolerated my erratic approach to the whole project and to the psychophysiological aspects of it in particular. It was she who was instrumental in putting some order to my confusion. I will be more than happy to know that she, now, thinks that she didn't waste her time after all. Putting all ideas into action was the responsibility of Mr. Dave Barton and Mr. Jack Whitcombe, both of whom devoted hours of their time in developing the computer programs that made the whole experimental procedure and data analysis appear so simple. In addition I would like to thank my Head of Department at Sunderland Polytechnic, Dr. N.F. Lemon, for being there to help with the numerous problems I had to face during the course of my work. My life would have been very difficult without the invaluable assistance of Mr. Spiros Vitouladitis who was responsible for the uninterrupted cash flow. Ismarie and her Angels brightened up my days and deserve my deepest thanks. Finally, it is the students, who voluntarily participated as subjects in this project, that I am grateful to, and it is to them that the thesis is primarily addressed.

Having said all that, I feel compelled to acknowledge the fact that all this is a reality due to the presence of Dr. D.C. Sanders who is morally responsible for this piece of work and whatever consequences it might have for the rest of my life.

Yanni Philippou Arvanitis, Sunderland, October 1985

DEPRESSION AND LEARNED HELPLESSNESS

A Psychophysiological Comparison

by Yanni Philippou Arvanitis

While clinical depression is not a unitary phenomenon, models of the disorder tend to evade the issue and provide monodimensional explanations of the symptoms and causes, and uniform approaches to the treatment of the various subtypes.

Psychophysiology, as a distinct approach to the study of depression, has provided evidence to support certain clinical distinctions between the subtypes and points to the necessity for a non-unitary model of depression and the development of a multidimensional classification system.

The Learned Helplessness model of depression, developed from observations in animal learning studies, has been another attempt to account for the phenomena of depression in terms of a simple learning relationship - a lack of correlation between response and outcome is cognitively represented by the subject which in turn perceives this as its own inability to control outcomes and this leads to 'learned helplessness' and depression.

Previously attempted psychophysiological comparisons between depression and LH have been rather inadequate in establishing a link between the two conditions. The present study is an investigation into the issues concerning the analogy between clinical depression and the LH state, particularly at the psychophysiological level.

For the purposes of the study, 30 subjects were randomly divided into three different groups: a naive group, a success group and a failure (helplessness) group. Electrodermal activity and heart rate were monitored in all three groups, during relaxation periods, before and after exposure to a neutral waiting period (Group 1), success in an electronic game (Group 2), and failure in the same game (Group 3).

Three-way analyses of variance with one repeated measure revealed a significant increase in Skin Conductance Level in Group 3 whereas Groups 1 and 2 showed no such changes. However, anagram performance, usually poor in 'helpless' subjects, did not differ between the groups.

The implications of these findings for the alleged analogy between clinical depression and the LH state are discussed.

PREFACE

Although depression, as a nosological entity, was described by Hippocrates more than 2000 years ago, its precise nature and causes are still a matter of controversy. Evidence from the fields of psychiatry, clinical and experimental psychology, and, more recently, pharmacology and psychophysiology, points to the existence of distinct subtypes within the main category, but still, disagreement over the various classification systems employed by different sections of the international scientific community, is the current state of affairs.

Various models of the disorder have been proposed, most of them based on clinical and psychological research. Attention has been drawn, in the past 20 years, to the Learned Helplessness model of depression and to the evidence that links it with the naturally occuring forms of the disorder. According to the exponents of the model, this evidence is cognitive, behavioural, pharmacological and psychophysiological.

The present study is an examination of the classification problems of depression, the utility of psychophysiology, as part of a multidimensional classification system, in the classification process, the relevance of the Learned Helplessness model in the study of depression and the evidence that links it with the disorder, particularly at the psychophysiological level.

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CHAPTER ONE

THE CLASSIFICATION OF DEPRESSION

Depression is classed as an affective disorder characterized by mood disturbances as well as somatic complaints, the whole group of the various subtypes being referred to as the Affective Disorders. According to the Committee on Nomenclature and Statistics of the American Psychiatric Association's (APA) Diagnostic and Statistical Manual of Mental Disorders (DSM-III), however, a more descriptive term whould have been "Mood Disorders" but the term "Affective Disorders" has been retained from earlier editions (DSM-I, 1952; DSM-II, 1968) for reasons of convenience and historical continuity (DSM-III, 1980, p.205).

A list of symptoms common in depression, compiled by Beck (1967), includes: sad face, stooped posture, crying in interview, slow speech, dejected mood, diurnal mood variation, suicidal wishes, indecisivenesss, feelings of inadequacy, conscious guilt, loss of interest, loss of motivation, fatiguability, disturbed sleep, appetite loss, constipation. The last four symptoms are physiological in nature rather than psychological. However, Beck, in reference to the use of affective adjective check lists in order to define and quantify depression, stated that "The representation of depression as an affective disorder is as misleading as it would be to designate scarlet fever as a 'disorder of the skin' or a 'primary febrile disorder'. There are many components to depression other than mood deviation. In a significant proportion of cases, no mood abnormality at all is elicited from the patient. In our present state of knowledge, we do not know which component of the clinical picture of depression is primary, or whether they are

all simply external manifestations of some unknown pathological process." (Beck, 1967, p.6). Actually, some depressed patients seem to consider their mood change of minor importance compared to other symptoms they experience during the depressive episode. Furthermore, according to patients, there is a qualitative difference between their feelings during a depressive episode and those they experience when not depressed. It seems that there is a rather important qualitative difference between the everyday transient mood changes that most of us experience and the dysphoric mood that characterizes the depressive individual (Beck, 1967).

The first official classification system of all mental disorders that appeared in the United States, (DSM-I, 1952), was a direct descendant of the Kraepelinian classification system that regarded mental disorders as definite disease entities (Blinder, 1966). The DSM-I system of classification divided depression into two major types: psychoneurotic depressive disorders and psychotic depressive disorders. Under the label psychotic depressive disorders were included the involutional psychotic reaction, the manic-depressive reaction, and the schizophrenic reaction, schizoaffective type, now classed as a separate psychotic disorder with no diagnostic criteria other than the uncertainty of the clinician to classify a particular case as an Affective Disorder, a Schizophreniform Disorder, or Schizophrenia (DSM-III, 1980, p.202).

A second, more refined and flexible edition of the DSM was published in 1968 (DSM-II). Also in the same year the eighth revision of the World Health Organization International Classification of Diseases (ICD-8), was published together with an edition adapted for use in the United States. The reason was the different systems of classification employed over the

years by European and American psychiatrists. Cooper et al. (1972) found significant differences in the diagnostic criteria used by American and British psychiatrists in particular. These differences have been described as 'too alarming' by Kendell, Pichot and von Cranagh (1973, p.27). These authors, in a study carried out in Europe, asked British, French and German psychiatrists to make a diagnosis based on five-minute videotaped interviews. It was found that despite overall similarities in the concepts of schizophrenia, alcoholism, neurotic illness and personality disorder, there were important differences in the concepts held by the British, French and German psychiatrists on the affective disorders. The differences, however, were not, according to the authors, as great as those that have been reported between American and British psychiatrists in the Cooper et al. (1972) study. An interesting point, illustrating the subjective element in clinical diagnosis, is that, since the general acceptance of lithium in the treatment of mania, American psychiatrists have been more willing to make a mania diagnosis instead of adopting the rather broad term 'schizophrenia' as they used to (Kendell, Pichot and von Cranach, 1973).

Under the general term 'depression' almost any combination of the symptoms listed by Beck (see p.1) can be found and several attempts have been made to quantify these symptoms and subject them to factor analysis in order to derive some objective system of classification. The benefit of such a system would be twofold. First, treatment would be tailored to each individual subtype of depression, a rather important issue since, as it will be pointed out later, there is a differential response to treatment between the various subtypes. Second, communication between researchers and clinicians, especially at the international level, would be improved.

This particular point is raised by van Praag (1976, p.197) when he attempts to "translate" the concepts that he personally uses "into the terms conventionally used in the literature, and particularly in the Anglo-American literature." It might even be shown that the various subtypes of depression have a different aetiology and thus prompt the development of even more effective therapeutic methods than currently available.

Factor analytic studies

Factor analysis is a "Statistical procedure aimed at the generation of hypothetical variables that are weighted sums of observed variates. The former, fewer in number than the latter, are usually expected to describe, summarize, or explain the latter." (Wolman, 1873, p.138). Its value lies in that existing systems of classification can be tested against data obtained from the clinical population the classification system under test is supposed to describe. In other words "we begin with a model and require to see whether it agrees with the data and, if so, to estimate its parameters" (Kendall, 1961).

Hamilton constructed a scale for rating depression and subjected the data generated by it to factor analysis (Hamilton and White, 1959; Hamilton, 1960). The analysis revealed six factors, four of which Hamilton interpreted as follows: The first factor "corresponded to the classical clinical description of Retarded Depression." (Hamilton and White, 1959, p.985), and had high loadings on depressed mood, guilt, retardation, loss of insight, suicide, loss of libido and loss of interest. The second factor was "vaguely like agitated depression" (Hamilton, 1960, p.59), with high loadings on somatic symptoms, insomnia, weight loss, retardation, hypochondriasis and anxiety. Hamilton

noted that this factor was "deficient in depression, the first factor having taken out most of the depressive variance." (p.59). The third factor was called "anxiety reaction" with high loadings on anxiety, agitation, loss of libido, somatic symptoms, guilt and insomnia. The fourth factor had high loadings on insomnia, somatic symptoms and anxiety. The patients who had high scores in factor four also had abnormal personalities and the factor was vaguely called "psychopathic depression" (Hamilton and White, 1959, p.896). The remaining two factors were considered of minor importance and were not interpreted.

Grinker et al. (1961) subjected to factor analysis a "Feelings and Concerns" check list and a "Current Behaviour" check list and extracted five factors from the first list and ten from the second. The first factor of the first list was considered to be a general factor of depression characterized by feelings of hopelessness, failure, sadness, unworthiness, guilt and internal suffering. The remaining four were considered to be Ego-defense mechanisms for dealing with the feeling of depression. When both lists were combined, four factors emerged: "retarded-empty", "anxious", "hypochondriacal", and "angry". The "retarded-empty" factor was characterized by dismal feelings, hopelessness, low self-esteem and guilt. The patient was withdrawn, apathetic and retarded. The response to somatic treatment was favourable. The second factor, "anxious", was characterized by similar feelings to those in factor one but anxiety was also noticeable. The behaviour of the patient was agitated, accompanied by demands for attention. Response to somatic treatment was good. The third factor, "hypochondriacal", was associated with feelings of not being loved, the patient complained of somatic symptoms, and was agitated and demanding. Drug therapy and supportive psychotherapy seemed the treatment of choice. Finally, the

fourth factor, "angry", was characterized by feelings of hopelessness and guilt with gloom and anxiety also being present. The patient was demanding and provocative, and somatic treatment was often required because of the high suicide risk.

Friedman et al. (1963) carried out a factor analysis of psychiatrists' ratings on the 60-item PPH Depression Rating Scale and obtained five factors. The first factor was identified as "classical mood, or affective depression with guilt, loss of esteem, doubting and psychological internalizing", the second as "retarded, withdrawn, apathetic depression", and the third as involving a "primarily 'biological' reaction with loss of apetite, sleep disturbance, constipation, work inhibition, and loss of satisfaction." The fourth factor was associated with a "querulous, hypochondriacal type, self-pre-occupied, demanding and complaining, irritable with marked body consciousness, and many physical compaints". The authors labelled this factor "oral-demanding depression" (Friedman et al., 1963, p.105). The fifth factor was not interpreted as it was related to the aging process rather than to depression and therefore considered to be of minor importance.

Kiloh and Garside (1963) isolated two factors in a check list of symptoms. The first was a general factor of depression and the second was a bipolar factor which differentiated between endogenous and neurotic depression. In another of the factor analytic studies (Rosenthal and Klerman, 1965), the existence of a general factor of endogenous depression was also shown. As endogenous are characterized depressions that are not associated with any external event, they simply appear and usually cycle over time between regular periods of normality. The symptoms are often severe and hallucinations and delusions are often present. Endogenous depressions respond

favourably to somatic treatments such as tricyclic antidepressants and electroconvulsive therapy (ECT), while neurotic depressions despite being, overall, milder with absence of any hallucinations and delusions do not respond well to somatic treatment (Carney, Roth and Garside, 1965; Kiloh, Ball and Garside, 1962; Kleinmuntz, 1980).

Rosenthal and Klerman's factor of endogenous depression was similar to Kiloh and Garside's second bipolar factor and Hamilton's first factor of retarded depression. So was the first, empty-retarded, factor in the Grinker et al. (1961) study.

Cropley and Weckowicz (1966) subjected to factor analysis the Beck Depression Inventory (BDI - Beck et al., 1961). The inventory consists of 21 subscales and is the most widely used self-report instrument for measuring severity of depression. In this study, 20 of the 21 subscales were used, and six meaningful factors were interpreted. The first factor, "guilty depression", had high loadings on depressed mood, guilt feelings and self-punitive wishes; the second factor, "retardation", loaded highly on fatiguability, work inhibition, lack of satisfaction and loss of libido; the third factor, "aggression turned towards one's own body", showed high loadings on body image, self-punishment, pessimism and crying; the fourth factor, "sel-depreciation", had high loadings on self accusation, self hate and sense of failure - there was an almost zero loading on depressed mood; the fifth factor, "anhedonic", was characterized by a sense of failure and lack of satisfaction; finally, the sixth factor, "neurotic, irritable depression", was characterized by irritability, somatic preoccupation, indecisiveness, depressed mood and self hate, and a substantial degree of hypochondriasis.

Their first factor, "guilty depression", resembled the first factor in the Friedman et al. (1963) study and also Hamilton's first factor of "retarded depression", the Grinker et al. (1961) first, "empty-retarded" factor, Kiloh and Garside's (1963) second, bipolar factor, and Rosenthal and Klerman's (1965) factor of endogenous depression. It could well be that all these similar factors are reflecting one of the major depressive syndromes, i.e. endogenous depression.

In addition to the endogenous factor that was obtained, under various names, in all the studies mentioned above, there were other common factors reported. For instance, there was some similarity between Hamilton's second factor (agitated depression) and Cropley and Weckowicz's sixth factor (neurotic, irritable depression) in the sense that both factors had high loadings on hypochondriacal complaints with some degree of agitation present.

The fact that there were comparable factors in all these studies despite the differences in approach - there is an apparent psychoanalytic influence on the Grinker et al. (1961) and the Friedman et al. (1963) studies, while Hamilton (1960), Kiloh and Garside (1963), and Cropley and Weckowicz (1966) followed a rather more 'objective' approach - suggests that the factors may reflect real differences between depressives and points to the existence of distinct depressive syndromes. This finding has been strengthened by more recent studies employing a different statistical technique, cluster analysis, which is more suitable than factor analysis for classification purposes (Byrne, 1978).

Factor analysis simply provides criteria against which any individual may

be compared, while cluster analysis creates classes of individuals clustered together on the basis of similarity of symptom values. In other words, "Cluster analysis, a statistical technique whereby cases are assigned to groups, or clusters, by means of some similarity measure, may be used either to provide confirmation of existing clinical schemes, if the mathematically derived clusters are similar to existing schemes, or to create different and potentially better ones." (Andreasen and Grove, 1982, p.45-46). In a sense, cluster analysis resembles the approach in classification used by clinical medicine (Andreasen, Grove and Maurer, 1980).

Cluster analytic studies

Employing cluster analysis, Paykel (1971 and 1972) identified four groups of depressives: psychotic, anxious, hostile and young depressives with personality disorder. The psychotic group included patients with the highest scores in "typically psychotic or endogenous symptoms as distinct quality, guilt, anorexia, retardation and delayed insomnia." (Paykel, 1971, p.281). The anxious group included patients with the highest scores in psychic and somatic anxiety, depersonalization, obsessional symptoms and fatigue and were, overall, moderately depressed. The hostile group had high scores on hostility and self-pity and these were predominant in the otherwise mildly depressed group. Finally, the fourth group included the youngest patients of the whole sample with a mean age of 29 years as compared to 47 of the psychotic group, 40 of the anxious and 34 of the hostile group. They had the lowest scores in most symptoms but high scores on the Maudsley Personality Inventory (M.P.I. - Eysenck, 1959) neuroticism scale. It was shown that these clusters were a useful predictor of response to treatment with the psychotic group responding best to amitriptyline.

Raskin and Crook (1976), however, failed to find any significant differences in response to treatment between the four groups in their study, which they identified as endogenous, agitated, neurotic and personality disordered depressives. Nevertheless, they did identify a group which corresponded to the concept of endogenous depression.

Byrne (1978) also obtained four clusters using the Zung Self-Rating Depression Scale (SDS - Zung, 1965). Cluster one comprised of individuals who were severely depressed, suffering from feelings of hopelessness and worthlessness and displaying the motivational deficit evident in retardation. Cluster two was small and characterized by sadness, fatiguability, insomnia, agitation and irritability, dissatisfaction and emptiness. Cluster three was similar to cluster one but the symptoms were less severe. Cluster four composed of patients who reported minimal levels of depressive symptomatology. Byrne noted the absence of a cluster similar to the syndrome of "reactive or neurotic depression" (p.8). Since the Zung SDS has been used in this study, it is worth noting the comment by Carrol, Fielding and Blashki (1973) that "The Zung SDS is not recommended for use in research studies." (p.361) because of its lack of sensitivity. Carrol, Fielding and Blashki found that this scale failed to reflect the varying severity of the disorder and this might explain the failure of Byrne to find a "reactive or neurotic" cluster in his study.

In two recent cluster analytic studies, (Andreasen, Grove and Maurer, 1980; Andreasen and Grove, 1982), three and four clusters, respectively, were reported. The analyses were based on interviewers' ratings on the Schedule for Affective Disorders and Schizophrenia (SADS - Ednicott and Spitzer, 1978).

In the 1980 study the three clusters found were: cluster one, similar to severe, endogenous depression, with patients showing signs of severe depression, retardation, few somatic complaints and feelings of guilt; cluster two, with patients experiencing stressful life events and suffering from less severe depression; cluster three, with patients suffering from mild, chronic depression.

The 1982 study involved a larger sample of patients and yielded four clusters. The first cluster was similar to severe, endogenous depression, and, according to the authors "practically identical with DSM-III major depression with melancholia." The DSM-III list of symptoms for this category includes dysphoric mood or loss of interest and pleasure, poor appetite or significant weight loss (or exactly the opposite), insomnia or hypersomnia, psychomotor agitation or retardation, fatigue, feelings of worthlessness, suicidal wishes, diurnal variation in mood and early morning awakening. The second cluster comprised of patients with less severe or moderate depressions and was "like DSM-III major depression without melancholia, but the fit was not as good." (Andreasen and Grove, 1982, p.51). The DSM-III symptom list for this category is the same as above with the exception of diurnal mood variation and loss of interest and pleasure. Also, the two categories are distinguished by the depressed mood being "perceived as distinctly different from the kind of feeling experienced following the death of a loved one" (DSM-III, p.215), by the patients classed as suffering from major depression with melancholia. Beck had already pointed to this qualitative difference in the mood experience of some depressives (see p.2) and this 'distinct quality' has also been noted by Paykel (see p.9).

The third cluster included patients suffering from depressions that cycled to mania (bipolar) and almost all patients fitted the discription of the "DSM-III bipolar affective disorder, mixed type." In this category are classed patients who rapidly alternate between manic and depressive episodes (major depressive episodes and manic episodes) or exhibit a mixture of symptoms of both mania and depression. Among the symptoms characterizing a manic episode, according to the DSM-III, are: increase in activity or physical restlessness, more talkative than usual, flight of ideas, inflated self-esteem, decreased need for sleep, distractability and increased involvement in activities such as buying sprees, sexual indiscretions and reckless driving regardless of the consequences. Finally, the fourth cluster was the smallest with only 10 patients, compared to the 25 of cluster three, 67 of cluster two and 99 of cluster one. The patients in cluster four suffered from severe depression with psychotic features. This depression was "precisely DSM-III major depression with mood-congruent psychotic features." (Andreasen and Grove, 1982, p.51). This category is identical to the major depression without melancholia with the addition of psychotic features (delusions and hallucinations) "whose content is entirely consistent with the themes of either personal inadequacy, guilt, disease, health, nihilism or deserved punishment; depressive stupor (the individual is mute and unresponsive.)" (DSM-III, p.125).

Overall, clusters one and two in both the Andreasen studies corresponded well with each other while cluster three was markedly different in each study. This was due to differences in patient content in the two samples. There were very few patients with mild depression and a substantial number of patients with bipolar depression in the sample of the second study. Also, the presence of a fourth cluster became detectable in the second study

because of the larger sample involved. Oddly, the patients in cluster two, which seems to correspond to the concept of neurotic depression, did not show the more chronic and less episodic depression that is traditionally associated with the term (Andreasen and Grove, 1982). On the contrary, these depressions tended to be rather shotlived when compared to those of clusters one and four. The authors did not offer any possible explanation for this discrepancy apart from suggesting that cluster two was heterogenous and perhaps needed to be subtyped further.

Although it appears that the Andreasen studies provide support for the DSM-III system of classification and also point to the existence of a distinct endogenous category, it is interesting to note that the term 'endogenous' has been dropped from the DSM-III nomenclature as implying the absence of external precipitating factors for the disorder and being, therefore, misleading (DSM-III, p.205). Furthermore, note is made in the DSM-III introduction (p.1) of the "many fundamental ways in which DSM-III differs from .... its international contemporary, the mental disorders chapter of the ninth revision of the International Classification of Diseases (ICD-9)."

The issue of classification is far from simply academic. There is a definite link between diagnosis, treatment and outcome (van Praag, 1978; see also Grinker et al., 1961, p.5-6) and there is good evidence to suggest that there is a differential response to treatment between the various subtypes of depression.

<u>The relationship between diagnosis, treatment and outcome</u>

Kay et al. (1969) reported a 5- to 7-year follow-up study of 104 cases of

"endogenous" and "neurotic" depressives and attempted to correlate these two syndromes with various measures of outcome, i.e. immediate outcome, scores on the Hamilton Rating Scale for Depression (HRSD - Hamilton, 1960), the number of readmissions, prolonged ill-health, and favourable course of illness. They also looked for correlations between outcome and the individual symptoms that characterize and differentiate the two syndromes. The "endogenous" symptoms were: severe depression, retardation, guilt, nihilism, hopelessness and hallucinations; the "neurotic" symptoms were: somatic complaints, blaming others, psychogenesis and long duration. In addition to these two 'pure' groups of patients, a third, "undifferentiated" group was also included in the study.

After comparing outcome between the three groups and correlating it with the various symptoms distinguishing each group from the others, the authors concluded that, in the endogenous group, the retardation symptom was the one strongly connected with a favourable outcome and in the neurotic group, the syndrome as a whole, and the somatic complaints in particular, were associated with poor outcome. The undifferentiated group was intermediate in outcome as compared with the other two. The authors also pointed out that the presence of paranoid ideas and schizophrenia-like features had a negative influence on the outcome.

Additional support for the endogenous-neurotic distinction in respect to treatment outcome is provided by the studies of Carney, Roth and Garside (1965), and Mendels (1965), who found that neurotic depressives respond less favourably than endogenous depressives to ECT, and Kiloh, Ball and Garside (1962), and Greenblatt, Grosser and Wechsler (1964), who reported similar findings with tricyclic antidepressants: neurotic depressives did not respond

well to treatment with tricyclics while endogenous depressives did. More recently, Paykel, Klerman and Prusoff (1974) in a study similar to that of Kay et al. (1969) but of shorter duration follow-up, reported again the endgodenous-neurotic distinction "to be the main systematic predictor of outcome." (p.62). Also, after analysing data from 412 patients treated with tricyclic antidepressants, van Praag (1978) found that "the best results were obtained in the group of vital, and the least good results in that of personal depressions." (p.219). In van Praag's terminology, vital depression corresponds to endogenous depression and personal depression corresponds to the syndrome "described in Anglo-American literature as psychogenic, neurotic or reactive depression." (p.205).

The significance of the symptoms present, in the choice of treatment, is illustrated in an example given by Kelly and Walter (1969); when anxiety is present in depression, amitriptyline, as having greater sedative properties than imipramine, would be the treatment of choice, all other considerations being equal. Tricyclics may be combined with tranquillizers or even neuroleptics, if the concomitant anxiety is overwhelming (van Praag, 1978).

The studies in the classification of depression reviewed so far were based on symptomatology and certain of the syndromes have been related to treatment outcome, with some responding more favourably to somatic treatment (syndromes similar to the concept of endogenous depression) and some being more responsive to psychotherapy (syndromes similar to the concept of neurotic depression). Chronicity has also been linked with the endogenous-neurotic distinction, with endogenous features being persistently associated with favourable outcome and neurotic features often associated with chronic episodes. Symptoms have also been known to be good predic-

tors of outcome. Retardation has been linked with favourable outcome while psychotic features, such as paranoid ideas, have not. Unfavourable outcome has also been linked with the neurotic syndrome as a whole and the somatic complaints in particular.

However, the need for a multidimentional system of classification is rather strong (Overall et al., 1966a; van Praag, 1978) because, first, syndromes are not reliably linked with aetiology, and second, neither syndromes nor aetiology reliably predict the course of a depressive episode (unipolar or bipolar), thus not allowing a complete nosology of the depressive disorders. This view is not, apparently, shared by the exponents of certain cognitive/ behavioural models of depression who propose a common aetiological factor for all types of depression.

CHAPTER TWO

COGNITIVE/BEHAVIOURAL MODELS OF DEPRESSION

The evidence presented so far points to the existence of distinct depressive syndromes with differential response to treatment, leaving, nevertheless, the question of aetiology unaswered. The various aetiological/precipitating factors have been integrated (Akiskal and McKinney, 1973 and 1975) and the depressive response has been suggested to be the final, common pathway. Current cognitive/behavioural models of depression also tend to regard depression as a unitary phenomenon and attempt to account for the causes, explain the symptoms and propose ways of treatment based on this premise; this limits their scope and usefulness in the study of the disorder (Craighead, 1980). A single, different factor, is postulated by each model as being central in causing depression, and treatment is geared towards the modification or removal of that factor by psychotherapy.

Beck's (1967) model attributes depression to a negative cognitive set. The depressed person sees himself as incapable of any form of success and has negative expectations about the future. Beck, as well as others (Ellis, 1962; Valins and Nisbet, 1971), regards these negative cognitions as primary, and depression as the secondary condition that ensues. Therapy is then based on techniques desinged to alter these cognitions and restore normal functioning in the person.

Lewinsohn (1974a and 1974b) regards depression as the result of inadequate response-contingent positive reinforcement and his approach to therapy is in designing a behaviour modification program that will increase the rate

of response-contingent positive reinforcement for the depressed individual. Lewinsohn makes it clear that it is not reinforcement per se that is missing, but reinforcement contingent with the depressed person's responses.

Seligman (1975) makes a similar point when he argues that the perception of non-control is the key factor in the development of the depressive response and this perception of non-control can be instilled in a person by exposure to either noncontingent success or failure. According to Seligman, depression results when an organism perceives a lack of control over its environment. Therapy is geared towards removing this perception of non-control by means of operant shaping programs, i.e. engaging in behaviour where the response-outcome contingency is apparent.

Rehm (1977) argued that although the perception of control over outcomes is important, it may be lost because of, first, inaccurate monitoring of outcomes, second, strict criteria applied to one's own self and, third, inaccurate attributions of responsibility. Treatment is, therefore, tailored to suit the patient's individual needs and also the patient under therapy is encouraged to increase the rate of self-reinforcement.

All these models can be regarded as being essentially correct in identifying one of the many possible causes of a depressive episode. Each treatment has a potential advantage over the others, provided it is applied to the person with the correct 'type' of depression. "The label, depression, would serve to cue the therapist as to which factors need to be included in the assessment. Treatment would then be assigned to match the problem area(s). If the problem is lack of social skills, then the client should receive social skills training; if the problem is a distortion of environmental feedback,

then cognitive therapy for this particular problem should be undertaken; if the problem is excessive standard setting, then more realistic standard setting skills should be taught; if the problem is poor self-monitoring of positive experiences, then self-monitoring skills should be taught; and so on." (Craighead, 1980, p.126).

In the cognitive/behavioural models, depression is considered as an undesirable trait, part of the depressive's personality, that can be eradicated or at least have its impact on the patient's life diminished so that it can be enjoyed again. Treatment, overall, is aimed at improving the patients' social skills and boosting their self-esteem (McConaghy, 1981).

The combined term 'cognitive/behavioural' has been used in this brief review since differentiation of the two concepts, particularly in respect to therapy, is rather subtle (Beck, 1970), and, in the opinion of the present writer, purely academic. Cognitive and behavioural approaches to therapy are complementary rather than mutually exclusive.

Seligman's model of depression is of particular interest, in this respect, since it was developed from observations in animal studies and has evolved, over the years, from a behavioural model into an entirely cognitive one incorporating concepts such as locus of control and attributions in its theoretical framework. Nevertheless, pharmacology and psychophysiology have also been implicated.

<u>The Learned Helplessness model of depression</u>

According to Seligman's original formulation (1975), the cause of all

depressions is perceived uncontrollability over events. The organism, after being exposed to noncontigent reinforcement (either positive or negative), perceives this noncontingency as its own inability to control outcomes. It subsequently ceases to respond, becomes passive, and, most important, fails to perceive future response-contingent reinforcement as such. The organism 'learns' that it is helpless. This Learned Helplessness, (LH), becomes a trait (Hiroto and Seligman, 1975) and takes the form recognized in humans as depression. Seligman also stated that "While reactive depressions are at the primary focus of the learned-helplessness model of depression, ... endogenous depressions have much in common psychologically with reactive depressions." (Seligman, 1975, p.79). He, in a sense, ignored the available evidence (e.g. Kiloh, Ball and Garside, 1962; Kiloh and Garside, 1963; Greenblatt, Grosser and Wechsler, 1964; Carney, Roth and Garside, 1965; Mendels, 1965; Kay et al., 1969; Paykel, 1971 and 1972; Paykel, Klerman and Prusoff, 1974) supporting the endogenous-neurotic distinction [neurotic depression is also called reactive depression (Depue and Monroe, 1978)] and went on to compile a comparison table of features, he regarded, common to LH and depression (see table one, p.21). His assertions received mixed support from both animal and human studies.

Animal studies

In the late sixties, Overmier and Seligman (1967) and Seligman and Maier (1967) reported a series of experiments where dogs exposed to inescapable electric shock failed to learn a subsequent escape/avoidance response, i.e. jumping over a shoulder-high barrier in a shuttlebox. Instead of trying to escape the shock, they remained passive and accepted it. The condition was termed 'learned helplessness' and these observations in the dog

Table one

Summary of features common to Learned Helplessness and Depression

	Learned Helplessness	Depression
Symptoms	Passivity	Passivity
	Difficulty learning that responses produce belief	Negative cognitive set
	Dissipates over time	Time course
	Lack of aggression	Introjected hostility
	Weight loss, appetite loss, social and sexual deficits	Weight loss, appetite loss, social and sexual deficits
	Noradrenaline depletion and cholinergic activity	Noradrenaline depletion and cholinergic activity
	Ulcers and stress	Ulcers(?) and stress
		Feelings of helplessness
Cause	Learning that responding and reinforcement are independent	Belief that responding is useless
Cure	Directive therapy: forced exposure to responses that produce reinforcement	Recovery of belief that responding produces reinforcement
	Electroconvulsive shock	Electroconvulsive shock
	Time	Time
	Anticholinergics; noradrenaline; stimulants(?)	Anticholinergics(?) noradrenaline; stimulants
Prevention	Immunization by mastery over reinforcement	(?)

Adapted from Seligman (1975).

experiments were the beginning of the Learned Helplessness Theory of Depression.

In the standard LH experiments a triadic design was employed. One group of dogs received, in a Pavlovian harness, electric shock which they coud terminate by making a simple motor response, i.e. lift a paw. Yoked to this group was a second group of dogs which received, by definition, the same amount of shock but no response that they could emit terminated it. This constituted the pretreatment phase. Both groups and an aditional, naive, group were tested, 24 hours later, in a shuttlebox escape/avoidance setting (testing phase). Almost invariably the yoked animals (uncontrollable shock group) were slow to learn to jump over the barrier to the safe side of the shuttlebox, if at all; the dogs were helpless. About two thirds of the animals in the yoked group just sat passively and took the shock. When forcibly dragged to the safe side of the shuttlebox (25 to 200 times were required)* the dogs were 'cured' of their learning deficit and started to respond on their own (Seligman, 1975). Also, the dogs could be 'immunized' against the debilitating effects of inescapable shock if they were exposed to escapable shock prior to the pretreatment phase (Seligman and Maier, 1967).

It was not passivity per se that made the dogs slow or unable to learn the responses necessary to avoid shock in the shuttlebox, but an aquired inability to perceive response-contingent reinforcement. The dogs simply

* There is some uncertainty concerning this figure. In his 1975 book, Seligman quotes the figure above, but in a later article (Alloy and Seligman, 1979) the figure quoted was 20 to 50. According to the original report, however, three out of the four dogs in the helpless group responded to this treatment and required 20, 35, and 50 trials, while the fourth dog required no such treatment at all (Seligman, Maier and Geer, 1968).

could not associate the termination of shock with their own responses. Maier (1970) demonstrated the point by training dogs to terminate shock in the pretreatment phase by holding still (or freezing). These dogs despite having been trained to be passive, still learned to avoid shock in the shuttlebox by emitting an active response, i.e. jump over the barrier.

The LH phenomenon has been demonstrated successfully in species other than dogs. Seward and Humphrey (1967) used cats in their experiments, and Padilla et al. (1970) used goldfish in an aquatic shuttlebox. Braud, Wepman and Russo (1969) used mice in a triadic design. The animals had to swim out of an alley flooded with water in the testing phase.

Findings in dog experiments have been replicated and extended in experiments with rats. Seligman, Rosellini and Kozak (1975) showed that simply dragging a rat randomly around the shuttlebox was not enough to reverse the helplessness deficit. When the rats were contingently dragged across the barrier, however, the deficit was reversed. So, it was the response-outcome contingency, rather, that was important in reversing helplessness. Also, Maier, Albin and Testa (1973) found, in the rat, that if the response required to escape shock in the shuttlebox was crossing the barrier once (Fixed Ratio 1 - FR1) then the inescapably shocked animals performed as well as controls. If, howeveer, an FR2 regime were employed, the rats became helpless and the usual performance deficits were observed. In a similar study Seligman and Beagley (1975) found that helpless rats could learn an FR1 lever-press response to terminate shock but not an FR3 one. It seemed that, in the FR2 and FR3 regimes, it was the difficulty in perceiving the contingency which made the rats helpless, and in order to rule out the effort requirement as an explanation for these results - FR2

and FR3 regimes required two and three times the effort of an FR1 - Maier and Testa (1975) introduced a delay (one to three seconds) between an FR1 crossing and shock termination, thus the effort requirements were the same in all conditions but the contingency more difficult to perceive in the three-second delay. Inescapably shocked rats failed to learn the escape routine in the three-second delay condition only. The effort required from the animals was not, it appeared, the crucial variable.

In addition to dragging the animals across the shuttlebox barrier, other methods of alleviating the helplessness deficit have been tried. Dorworth and Overmier (1977) found that a series of electroconvulsive shocks, (ECS), resulted in marked improvement in shuttlebox performance of three previously helpless dogs. Another three dogs that received the same ECS treatment showed no improvement. Dorworth and Overmier attributed their findings to noradrenaline increases in the brain of the helpless dogs which resulted from the ECS treatment; noradrenaline levels in the brain decrease when animals are exposed to inescapable shock (Weiss, Glazer and Pohorecky, 1976; Vogel and Haubrick, 1973). An alternative interpretation has been put forward by Lewis (1969 and 1979) who suggested that it is the memory interference of the ECS which is responsible for the improvement in the performance of the helpless dogs and not the increase of noradrenaline levels in the brain of the animals.

Brett, Burling and Pavlik (1981) attempted a replication of the Dorworth and Overmier (1977) dog study. The used ECS to alleviate LH in rats, in three different experiments. They employed the standard triadic design and the procedure involved three distinct phases for experiment 1, and two phases for experiments 2 and 3. The first two phases were the same in all three

experiments. First, in the training phase, all animals were placed in automated Skinner boxes and according to group allocation they were exposed to escapable or inescapable shock. A third, naive, group was placed in a Skinner box for the mean duration time of the training session of the other two groups. After 24 hours, a testing phase followed, with the animals being tested in a shuttlebox escape/avoidance task. In experiment 1, a retesting phase was included, identical to the testing phase, 24 hours later. In experiment 1, ECS was administered to half the animals in each group, immediately after the testing phase. In experiment 2, one third of the animals in each group were given an ECS immediately after the testing phase and another third received the ECS 30 minutes prior to the testing phase. The remaining third of the animals received no ECS at all. In experiment 3, half the animals were given an ECS 30 minutes prior to the training phase. In all three experiments, one ECS was administered to each of the ECS animals, whereas 64 ECSs were administered to the dogs in the Dorworth and Overmier study.

The results indicated an overall alleviation of the LH deficit in the helpless animals following ECS treatment. In experiment 2, however, all animals that received ECS 30 minutes prior to the testing phase were slower to learn the avoidance response in the shuttlebox, except those in the inescapable group which showed a LH effect during early trials but improved considerably later on in the test. In experiment 3 the results were rather strange and difficult to interpret. For instance, the inescapable groups, regardless of whether they received ECS or not, showed a LH effect but the pattern was different from that observed in experiments 1 and 2. The helpless animals in experiment 3 started responding in a quite efficient way and their performance progressively worsened across trials, while the helpless

animals in experiments 1 and 2 started off with slow responses which were maintained across trials.

Experiment 3 was run one year after experiments 1 and 2, and in a different laboratory using the same stock of animals and equipment; but whilst the same equipment was used, it had to be rewired and reprogrammed and the authors attributed the diverse findings in experiment 3 to subtle changes in either the equipment or the animal population. An interesting observation was that the LH pattern seen in experiment 3 was more typical of the LH seen in other rat experiments while the pattern in experiments 1 and 2 resembled more the pattern seen in dog experiments (Brett, Burling and Pavlik, 1981).

Results that do not support the LH theory have been reported by Glazer and Weiss (1976a and 1976b) who, incidentally, use the phrase "long-term interference effect" to refer to the debilitating effects of inescapable shock (Glazer and Weiss, 1976a, p.202). They found that rats who failed to cross a barrier in a shuttlebox at FR2 and press a lever at FR3 to escape shock, showed superior performance in an escape/avoidance test that required little movement, i.e. a nosing response. This is not consistent with the Maier and Testa (1975) findings which have been interpreted as supporting the cognitive deficit hypothesis rather than a motivational or activity deficit. Also, in another study, the same authors found that duration of shock in the pretreatment phase was as critical as noncontingency. "The interference effect only became evident if animals were exposed to inescapable shocks that lasted 5 sec or more. Thus, duration of shock, not simply noncontingency, is critical." (Glazer and Weiss, 1976a, p.203).

Cogan and Frye (1981) using a Sprague-Dawley strain of rats, found a general facilitating effect in all three groups of animals, i.e. escapable, inescapable, and control. Referring to similar findings by Beatty (1979), Brookshire, Littleman and Stewart (1961) and deToledo and Black (1967) and to the coments of Seligman and Beagley (1975), who implied that these contradictory findings may be the result of small procedural differences between experiments, the authors remarked that "any effect that lacks the robustness to overcome such small discrepancies may well be an artifact of the specific procedures that yield it. On the basis of the evidence reported here, we seem inescapably drawn to the depressing conclusion that learned helplessness, in the rat, is not a reliable enough phenomenon to be considered an analog of depression." (Cogan and Frye, 1981, p.288).

Nevertheless, only a year later, Sherman, Saquitne and Petty (1982) reported a study testing the LH model with antidepressant drugs and psychoactive drugs that are not effective in the treatment of depression, using Sprague-Dawley rats. They found antidepressants to reverse helplessness, while other psychoactive drugs did not. Among the antidepressants used were Imipramine, Desipramine, Amitriptyline, Noritriptyline and Iproniazid. In the psychoactive group, Diazepam, Chlorpromazine, Haloperidol, Amphetamine and Phenobarbital were among the drugs used. Most drugs were administered in two different concentrations. When the animals were tested, inconsistent results were obtained during early trials. When the number of trials was increased - five or less escape failures in 15 trials served as the criterion - the reversal of LH by the antidepressants was clear with the exception of Iproniazid at 10mg/Kg (low concentration). None of the psychoactive drugs appeared to reverse helplessness under this criterion.

The demonstration of the LH phenomenon in animals has been far from consistent. The deficit was evident only when certain types of response were employed as the dependent variable, and small procedural differences between studies were enough to yield contradicting results. Facilitation instead of a performance deficit has been found whenever the response requirement in the testing phase was minimal and noncontingency has been shown to be insufficient, on its own, to induce helplessness. In several of the studies where LH has been demonstrated the sample sizes were small (e.g. Maier and Testa, 1975; Seligman, Maier and Geer, 1968; Seligman, Rosellini and Kozak, 1975; Seligman and Groves, 1970), and, furthermore, several alternative explanations for the performance deficit of helpless animals have been proposed [see Maier and Jackson (1979) for a review].

Human studies

Research on LH has been carried out with human subjects and two early studies did not provide the expected support for the LH model. Thornton and Jacobs (1971) found that escapable electric shock was as effective in producing deficits in subsequent performance as was inescapable shock. Out of the four groups inlcuded in their study - escapable, yoked active, yoked inactive and control - only the control group differed significantly from the other three by being faster in a reaction time task. The task, pressing buttons to terminate electric shock, was similar in both the pretreatment and testing phases. They also found that variable intensity shock was more effective in inducing performance deficits than fixed intensity shock. In a subsequent paper, the same authors reported facilitation on intellectual performance - as measured by scores on the Wonderlic Personel Test - following inescapable shock. The effect was

stronger in the group that received variable intensity inescapable shock.

Miller and Seligman (1975) and Hiroto and Seligman (1975) found that subjects treated with inescapable aversive noise (90db) did show performance deficits when tested on an anagram solving task, compared to subjects who were exposed to the same amount of noise that they could control, and naive controls. The standard experimental design, as it become established, involves an escape group that can control and escape aversive noise stimulation, a yoked group that receives the same amount of noise without the ability to control it, and a third, naive, group that receives no noise treatment at all. All three groups are tested for performance deficits in an anagram solving task following the pretreatment phase.

Hiroto and Seligman (1975) also employed failure in a cognitive task as the helplessness induction procedure on the grounds that "insolubility in a cognitive task is formally analogous to inescapability, since in both the probability of reinforcement (correct or incorrect, or shock or no shock) is independent of responding." (p.313). Their findings were similar to those obtained from subjects treated with inescapable aversive noise.

Miller and Seligman (1975) compared depressed and nondepressed college students (the distinction was based upon BDI scores) in a standard LH induction procedure. Subjects that scored 8 or less on the BDI were assigned to the nondepressed group and subjects that scored 9 or above were assigned to the depressed group. Subsequently, two independent experiments were run, one with depressed and one with nondepressed subjects, following the paradigm described briefly above. The results from the experiment with the nondepressed subjects paralleled those of Hiroto

and Seligman (1975), i.e. inescapable noise subjects showed poor anagram performance compared to escapable noise and naive control subjects. Inescapable noise subjects also showed an increase in anxiety and hostility scores on the Multiple Affect Adjective Check List, today form (MAACL - Zuckerman and Lubin, 1965). Also, Gatchel, Paulus and Maples (1975) found significant increases in depression scores in addition to anxiety and hostility scores, following pretreatment with inescapable noise, using the same check list.

The results from the experiment with the depressed subjects were not as straight forward. The naive control group mimicked the poor anagram performance of the inescapable noise nondepressed group but there was considerable facilitation and slight facilitation found in the escapable and inescapable depressed groups respectively. No mood changes were observed in any of the three depressed groups. Miller and Seligman concluded that "depression and inescapability were not additive." (p.233). This is rather embarrassing for the LH theory since inescapability is additive in animals (Seligman and Groves, 1970); the longer the animal is exposed to inescapable shock the greater the LH effect. It logically follows, since inescapability eventually leads to depression and since the observed performance deficits, following exposure to inescapable stimulation (electric shock, aversive noise, etc.), are early manifestations of the depressive response, that performance deficits due to inescapability or depression are qualitatively similar and, therefore, should be additive, or, at least, the theory should predict so. In other words, LH should enhance the depressive symptoms; a person who becomes mildly depressed because of inescapability <u>must</u> become more depressed by more inescapability, if the model is to have any value in the study of human depression.

Griffith (1977) who used failure in a cognitive task to induce helplessness, assessed the relative balance of anxiety and depression in the subjects, by using the Paired Anxiety and Depression Scale (PADS - Mould, 1975). Five groups, in all, were incorporated in the design. A control group which was simply exposed to the task material without being asked to provide any solutions to the problems, a solvable group, a noncontingent failure and a noncontingent success group and, finally, a naive group. Noncontingent success is, according to Seligman's original formulation (1975), equivalent to noncontingent failure.

The results of the anagram test were as predicted by the LH theory. Noncontingent groups performed poorer than contingent and naive groups. Unexpectedly, the control group performed even more poorly than the noncontingent groups. The mood data showed no differences in the pre- and post-scores of the contingent and control groups. The predicted increase in depression relative to anxiety scores was only confirmed in the noncontingent failure group. The opposite was found in the noncontingent success group; anxiety increased relative to depression. This result is rather strange since the noncontingent success group showed a stronger LH effect than the noncontingent failure group. Seligman (1975), however, claimed that anxiety is the first reaction in a LH setting and this gives way to depression later on. On the other hand, it could well be that it was the presence of anxiety that produced the greater LH effect in the noncontingent success group and not the lack of contingency as such.

Dyck and Breen (1978) found task importance to be a factor that contributes to the effectiveness of the pretreatment phase in LH. When subjects were led to think of the pretreatment task as being of high importance, they

showed the expected anagram performance deficits, while subjects who were led to think of the pretreatment task as being of minor importance, did not. Also, paralleling studies in animals (e.g. Seligman and Maier, 1967; Seligman, Rosellini and Kozak, 1975), they reported immunization against helplessness by having subjects experience success in a task, prior to the pretreatment phase. Similar effects of immunization against the effects of failure have been reported by Klee and Meyer (1979). Skinner (1979) also found the LH effect to be a function of task significance and Williams and Teasdale (1982) demonstrated an interaction between perceived difficulty and importance of a task.

LH in humans proved to be specific to certain types of testing task - i.e. anagram solving - and a function not simply of perceived uncontrollability but also of attributed task difficulty and significance. Tennen and Eller (1977) manipulated the subjects' attributions and obtained both helplessness and facilitation as a result of initial failure in a cognitive task. Their design, in addition to the three standard groups - solvable, unsolvable, control - included two double helplessness (DH) groups. These groups were treated with three sets of unsolvable discrimination problems as opposed to one set for the standard, single helplessness (SH) group. One of the DH groups was led to believe that each subsequent set of problems was easier than the previous one (DH-easier) and the other was led to believe that the subsequent set was harder (DH-harder). The DH-harder subjects solved more anagrams in less time than any other group in the study. DH-easier and SH subjects, on the other hand, showed the expected poor anagram performance with DH-easier being the slowest of the two groups. It was suggested that when subjects believe that their failure is specific to the situation they double their efforts in subsequent tasks.

Although facilitation has been found in animals (e.g. Glazer and Weiss, 1976a; Cogan and Frye, 1981), obviously, it cannot be explained by attributions the animals might make concerning task significance etc. Alloy and Seligman (1979) in reviewing the animal literature stated that "We feel a bit uncomfortable talking about perceptions and epxectations in animals because such entities are a bit less observable than stimuli, responses, and reinforcers, which are themselves not directly observable. We are more uncomfortable ascribing attributions to animals because they are even more inferential and may require a level of cognitive complexity which animals do not have, and most importantly because no operational definitions exist for attributions in animals." (p.241).

In human LH the issue of attributions became rather central and it was incorporated in the reformulated LH model proposed by Abramson, Seligman and Teasdale (1978), in an attempt to account for the discrepant results, in human LH, reported in the literature.

The reformulated model of Learned Helplessness

The reformulated model (Abramson, Seligman and Teasdale, 1978; Abramson, Garber and Seligman, 1980) was an extension rather than a revision of the original model (Seligman, 1975); "Both the old and the reformulated hypotheses hold the expectation of nocontingency to be the crucial determinant of the symptoms of LH." (Abramson, Seligman and Teasdale, 1978, p.52). The original model did not make explicit predictions concerning the subjects' attributions about the uncontrollable situations they were experiencing. Reference has been made, however, of the possible role attributions can play in human LH (Seligman, 1975, p.30) with externality linked with greater

susceptibility to helplessness (Hiroto, 1974).

Three dimensions are taken into account in the reformulated model in order to describe the possible attributions formed by the subjects in a LH setting: Internal-External, Stable-Unstable, Global-Specific. Uncontrollability might be attributed to factors internal or external to the subject, i.e. inside or outside the subject's sphere of influence. In addition, uncontrollability might be attributed to stable or unstable factors, i.e. factors that persist or wither over time. Furthermore, the subject might attribute uncontrollability to global or specific factors, i.e. factors that apply across situations or to the situation at hand only.

There are eight possible combinations under this scheme: 1. Internal-Stable-Global; the subject attributes his condition to factors which are inside his sphere of influence, persist over time and apply across situations, 2. Internal-Unstable-Global; the subject attributes his condition to factors which are inside his sphere of influence and dissipate over time but apply across situations as long as they last, 3. Internal-Stable-Specific; the subject attributes his condition to internal factors which persist over time but are specific to the present situation, 4. Internal-Unstable-Specific; the subject attributes his condition to internal factors which dissipate over time and are specific to the present situation, 5. External-Stable-Global; as in case 1 but the factors are outside the subject's influence, 6. External-Unstable-Global; as in case 2 but the factors are again outside the subject's influence, 7. External-Stable-Specific; as in case 3 but with factors being external, 8. External-Unstable-Specific; as in case 4 with external factors involved.

The model predicts that when global attributions are made, helplessness results and generalizes across situations. When, on the other hand, specific attributions are made, helplessness results only for the particular or a very similar situation. Furthermore, stable attributions will produce chronic helplessness across situations, if global, or transient helplessness, if specific. Finally, internal attributions - the subject's own fault - will produce loss of self-esteem, while external attributions - fault attributed to others - will not. A similar account has been proposed by Miller and Norman (1979) with the addition of pretreatment task importance as a fourth dimension.

Seligman et al. (1979) tested the reformulated model in a college population. Students were divided in two main groups (depressed and nondepressed) on the basis of BDI (short form) and MAACL (today form) scores. An attributional style scale was used to assess differences in attributions between depressed and nondepressed subjects. The results supported the reformulated model. Depressed subjects tended to attribute bad outcomes to internal, stable and global factors, while good outcomes were attributed to external, unstable factors. The higher the depression scores in both the BDI and the MAACL, the greater the ratings of internality, stability and globality of causes of bad outcomes. BDI scores correlated negatively with ratings of internality and stability of causes for good outcomes. This was not the case for globality ratings. Also, no correlations between attributions for good outcomes and MAACL scores were found.

Seligman et al. interpreted these findings as supporting the reformulated model and pointing to the existence of an attributional style which "does not by itself result in depression: only when a bad outcome actually occurs

or is anticipated and is attributed to global, stable, and internal factors, does depression ensue." (Seligman et al., 1979, p.246). Raps et al. (1982) replicated these findings, using the same attributional style scale, in an all male population of unipolar depressives. A group of nondepressed schizophrenics and a group of nondepressed medical patients served as control. The depressed patients made more internal, stable and global attributions concerning bad events, compared to the nondepressed schizophrenics and medical patients, but, as in the Seligman et al. (1979) study, the depressive attributional style for good events, i.e. external, unstable, specific attributions, was not as "robustly associated with depression [as] was the style for bad events." (Raps et al., 1982, p.105).

Harvey (1981) found partial support for the reformulated model with a nonclinically depressed, all female sample. The support was partial since the predicted differences between depressed and nondepressed subjects emerged only in the external-internal dimension; "No differences were found for the stable dimension, and the global dimension of causes could not be reliably identified." (p.140).

All these studies were correlational in nature rather than experimental. Golin, Sweeney and Shaeffer (1981), on the other hand, applied cross-lagged correlational analysis in an attempt to show some temporal precedence of attributions over depression. The authors did acknowledge the fact that "Cross-lagged analysis does not demonstrate causality in the same way as true experiments" (p.15). Their analysis showed that stable, global attributions for bad outcomes may be a cause of depression. This was not the case for internal attributions. They also found that unstable attributions may be a cause of depression. The subjects in this study were college

students selected on the basis of BDI scores. The same scale as in the Seligman et al. (1979) and Raps et al. (1982) studies, was used to quantify the attributions made by the subjects.

The reformulated model has been tested 'in vivo' by Rothewell and Williams (1983) in a sample of working and nonworking men. The BDI was used to assess severity of depression and the attributional style scale (Seligman et al., 1979) provided a measure of attributions. Only internality correlated with depression after a stressful life event (redundancy).

Causal attributions about stressful life events have been examined in various real life situations by Hammen, Krantz and Cochran (1981) who found mixed support for the reformulated model of LH. The did not confirm the prediction that internal, stable and global attributions were related to depression but they did find a positive correlation between experienced uncontrollability and depression.

The reformulated model of LH has not generated the amount and diversity of research that the original model did. A great deal of attention has been focused on the possible role of attributions in LH and depression whereas the important issue of the analogy between LH and clinical depression seems to have been neglected. The reformulated model has been proposed in an attempt to account for the discrepant results of earlier studies. By introducing, however, variables the nature of which is not yet clear, much of the precision, elegance and falsiability of the earlier model has been sacrificed (Huesmann, 1978).

A different approach has been adopted by certain investigators to strengthen

the behavioural findings in LH experiments. Donovan (1981) used taped infant crying as the aversive stimulus, in the pretreatment phase, on a group of mothers. She found that infant crying is as effective in inducing helplessness, when the subject has no control over it, as is white aversive noise. Her results replicated earlier findings (Kevill and Kirkland, 1979) using infant crying as the aversive stimulus on a nonmother sample. The results were supported by a physiological measure (cardiac deceleration) that differentiated the escapable group from the inescapable and control groups; cardiac deceleration has been associated with stimulus intake (Lacey, 1967).

Physiological measures have been adopted as dependent variables in the study of LH by Krantz, Glass and Snyder (1974), Gatchel and Proctor (1976), Gatchel, McKinney and Koebernick (1977) and Suarez, Crowe and Adams (1978), who compared and contrasted their findings with those from similar studies in the depressive disorders. This psychophysiological approach to depression and LH will be discussed next.

CHAPTER THREE

THE PSYCHOPHYSIOLOGY OF DEPRESSION AND LH

Psychophysiology is not a new approach to the study of Man. Hassett (1978) makes the point that "Psychophysiological observation is as old as the first young man who noted a woman's blush" and "In this ancient and honourable event lay the seeds of several modern scientific conceptions. A mental state was inferred from a well-defined physiological change (increasing blood flow to the face). Further, the physiological change was believed to be more valid than words could ever tell." (p.1).

Lader starting his introduction to "The Psychophysiology of Mental Illness" quotes Darrow (1964a) who defined psychophysiology as "the science which concerns those physiological activities which underlie or relate to psychic function" and adds his own definition that "Psychophysiology is the study of physiological variables which have a relevant correlation with behavioural events." (Lader, 1975, p.1).

Once technology could deliver the means by which the relevant body responses could be monitored, psychophysiology grew as a separate branch of the behavioural sciences with its own specific terminology and methodological approach. The object was to find out how the body responds when the organism, Man, behaves in a specific way and if these responses have any behavioural significance. It was soon realized that certain mental states show a characteristic response pattern and that certain responses are common across states.

The behavioural significance of Electrodermal Activity and Heart Rate

The history of Electrodermal Activity (EDA) begun in 1888 when Féré reported changes in the skin resistance of a hysterical anorexic patient, when a weak electric current was applied on the patient's forearm (exosomatic method). Two years later, Tarchanoff (1890) independently reported similar electric changes without the imposition of external electric current (endosomatic method). These changes became known as the Galvanic Skin Response (GSR), a term now obsolete but still found in some literature.

The changes Féré was referring to, are now known as Skin Resistance Responses (SRRs) and are changes in the electrical resistance of the skin brought about by sweat gland activity. Electrical models of the skin regard sweat glands as electrical resistors connected in parallel and changes in their combined activity are thought to be reflected in the SRRs (Venables and Christie, 1980). For statistical and biological reasons Skin Conductance (SC), as a unit of quantification of EDA is preferable to Skin Resistance (SR). SC units - μmhos - tend to give a better approximation to normal distribution than SR units - Kohms - (Hassett, 1978; Venables and Christie, 1980). Also, electrical conductance is defined as the reciprocal of electrical resistance ($C = 1/R$) and in the case of resistors connected in parallel - such as the sweat glands of the skin - their conductances are additive ($1/R_{total} = 1/R_1 + 1/R_2 + ... + 1/R_n$) and for this reason SC is a more direct index of sweat gland activity (Darrow, 1964b; Tregear, 1966; Lader, 1970).

Tarchanoff, on the other hand, was measuring changes in the electrical potential difference between two skin sites. In this case Skin Potential Responses (SPRs) were being measured. Because of the different mechanisms

involved, the two variables, SC and SP, do not yield the same results and it is important to remember that SC and SP are not interchangeable (Hassett, 1978; Venables and Christie, 1980).

The overall electrical activity of the skin is referred to as Electrodermal Activity (EDA) with tonic activity being referred to as Skin Conductance Level (SCL) or Skin Potential Level (SPL), depending on whether exosomatic or endosomatic methods of measurement are being used and also on the physiological mechanisms involved. When SCRs or SPRs are observed after the presentation of a definite stimulus to the subject, they are simply called SCRs or SPRs, while when the presence of a stimulus is not apparent, these responses are known as Spontaneous SCRs (SSCRs) or Spontaneous SPRs (SSPRs). SCRs and SPRs, whether spontaneous or not, constitute the phasic component of the EDA (Venables and Christie, 1980). The above terminology will be used throughout the present text, although different, and now obsolete, terms have been used in some of the literature that will be reviewed and discussed.

Also, for the purposes of the present study, Heart Rate (HR) will always denote beats per minute (bpm). The cardiac cycle produces a rather complex electrical wave but the theoretical and practical arguments over its measurement and quantification will not be discussed here - suffice to say that HR, like EDA, shows phasic changes over the tonic level.

Katkin (1965) suggested that SSRRs were emotional responses to the experimental conditions while changes in SRL reflected non-emotional or cognitive responses. Miller and Shmavonian (1965) although they replicated Katkin's findings, expressed some concern about his rather premature conclusions.

Claridge (1967) postulated a tonic and a phasic activation system of arousal. If the tonic activation system is stable, the stability is reflected in the relatively constant SCL. If the system is labile, SCL changes greatly over short periods of time. The differences in stability are also reflected in the case of intensive stimulation with a stable system showing little change as a result of intensive stimulation and the labile system showing big changes for the same amount of stimulation. The phasic activation system can be either activated or inhibited. When activated we see high reactivity to weak stimuli and many SSCRs as compared to low reactivity to weak stimuli and few, if any, SSCRs when the phasic activation system is inhibited. Crider (1979, p.42) discussing the relationship between electrodermal lability and arousal, proposed that "electrodermal lability reflects more general individual differences in discriminative ability or, in Katkin's (1975) words 'in higher central processes involved in attending to and processing information' (p.171)".

Obrist, Lawler and Gaebelein (1974) reported a positive correlation between HR and somatic activity and Epstein, Boudreau and Kling (1975) concluded that "heart rate varies more directly and reliably with motor output than skin conductance" which is "more sensitive to small cognitive than to small motor effects." (p.15). Nevertheless, Obrist (1976) has argued that under certain conditions, e.g. active avoidance of aversive stimuli, cardiac-somatic coupling can be dissociated and HR can reach levels unrelated to the demands of somatic activity.

The psychophysiology of Depression

EDA and, to a lesser degree, HR, have been used as indices of depressive states, linked with particular syndromes, and symptoms. Riazansky (1965)

reported differences in EDA between various depressive subtypes (depressive phase of manic-depressive psychosis, cyclothymic depression, involutional depression, psychogenic depression and depressive-paranoid syndrome in schizophrenia). He assessed the patients' SRRs to tactile, auditory and verbal stimuli, measuring the magnitude, the latency, the speed and the duration of each SRR. His results indicated a greatly diminished EDA, as indicated by the low reactivity to the various stimuli, in the "deep depressive states of cyclical character, the apatico-aboulitic, and the stuporous states" (p.382). On the other hand, "The mean values of GSR .... [were] more pronounced in the less deep depressions - the cyclothymic and the reactive depressions" (p.381).

Martin and Rees (1966) compared a group of female depressives suffering from primary depression with a group of non-psychiatric controls in a reaction time test and in two physiological measures (SR and muscle action potentials - maps) in response to auditory stimuli preceded by a warning light. The results from three different experiments indicated that although, overall, the reaction times of endogenous depressives were the slowest and those of controls were the fastest, with mixed depressives occupying an intermediate position, the physiological measures did not differentiate the three groups. The authors questioned whether "the sub-groups employed .... represent much more than different severities of depression." (p.380).

Bagg and Crookes (1966) employed a rather unusual method of assessing sweat gland activity. They applied black paint on the fingertip of subjects and, under low magnification (20X) they counted the active sweat glands; active sweat glands show as holes on the black paint when it is peeled off the skin. Their subjects were women suffering from depression and their

sweat gland activity was assessed before and after they received ECT. They found that sweat gland activity increased with clinical recovery.

Zuckerman, Persky and Curtis (1968) compared a group of 29 male psychiatric patients and 25 normal male controls, in anxiety, depression, hostility and autonomic variables. Anxiety, depression and hostility were assessed by raters using a series of 10-point scales. The autonomic variables included HR, breathing rate, blood pressure, SCL and SSCRs. These measurements were taken before and after a cold-pressor stress test. The results indicated that anxiety in patients, controls and in both groups combined, correlated positively with baseline and post-stress SSCRs. Depression correlated positively with SC change and with SSCRs before and after the cold-pressor stress test. In controls, depression correlated positively with resting HR and SCL. Depression correlated positively with SSCRs before and after the cold-pressor stress test for both groups combined and correlated negatively with SCL. The authors concluded that SSCRs "show a specific relationship to the dysphoria effects, anxiety and depression." (p.485). They went on to postulate that SCL reflects a general level of arousal while SSCRs "are more closely related to some kind of oscillating effect in the autonomic system." (p.486). Although rather vague about this point, they suggested that SCL and SSCRs, despite being monitored through the same set of electrodes, and being moderately correlated, may reflect different aspects of psychological functioning. Schwartz (1974) also suggested that SCL may be more responsive to general arousal and SSCRs may express emotional or limbic system stress. Finally, they pointed out that HR may indicate general arousal and does not seem to be related to anxiety.

Lader and Wing (1969) made a direct physiological comparison between 35

depressed patients and 35 normal controls. The patients were divided into three groups: retarded, agitated and an intermediate group of 'uncomplicated' depressives. The retarded patients were characterized by a diminution of purposive movements and speech, were slow to obey simple commands and to answer questions, and had few complaints, although they appeared pre-occupied with their problems. The agitated patients were characterized by an excess of non-purposive movements and speech, were restless and complained of feelings of tension and anguish. The uncomplicated group, although moderately depressed, showed neither retardation nor agitation. The physiological variables, along which the three groups were compared, included SCL, SSCRs, HR and electromyogram (EMG) of the right forearm extensor muscles.

During the experimental session a series of auditory stimuli were presented to the subjects. At the beginning of the session the agitated patients had significantly higher and the retarded patients had significantly lower SCLs than the normals. At the end of the session the differences between the groups were more pronounced than at the beginning. SSCRs also discriminated the groups, the agitated patients having more SSCRs than both the controls and the retarded patients. The retarded patients had significantly fewer SSCRs than the controls. HR was higher in the patient groups but the difference reached statistical significance for the agitated group only, which had significantly higher HR than the retarded group as well. No significant differences were found between the groups in respect to EMG.

Overall, the uncomplicated group showed responses similar to those of the normal group. In respect to SCL at the beginning and at the end of the experimental session, the change in SCL during the session and the total

number of SCRs and SSCRs, the agitated and retarded groups were on either side of the normal and uncomplicated groups. There was complete discrimination between the agitated and the retarded groups in respect to SSCRs and habituation rate of SCRs elicited by the 20 auditory stimuli presented to the subjects during the experimental session. The agitated patients showed, in this sense, increased sensitivity to the stimuli compared to the retarded patients.

Kelly and Walter (1969) compared a group of 60 normal controls with a group of 56 retarded depressives, 25 agitated depressives and 45 anxiety patients. The variables employed were scores on a self-rating anxiety scale, the Taylor scale for manifest anxiety (Taylor, 1953), the scores on the neuroticism and extraversion scales of the MPI, forearm blood flow and HR. The physiological measurements were taken, and the self-rating anxiety scale completed, during relaxation and under induced stress (2½ minutes of mental arithmetic - Kelly, 1967).

Basal forearm blood flow differentiated anxiety patients and agitated depressives from retarded depressives and controls, as did HR. The retarded depressives did not differ from controls in basal forearm blood flow but controls had the lowest HR in all groups. The most important finding was that agitated depressives had higher basal forearm blood flow, HR and self-rated anxiety than retarded depressives, thus supporting the clinical distinction. Normal controls and retarded depressives had similar values of basal forearm blood flow. This finding suggests that increased forearm blood flow might be an indication of anxiety rather than depression. The effects of stress - mental arithmetic - did not positively differentiate the four groups.

Noble and Lader (1971a) in a study similar to that of Kelly and Walter (1969) found no significant correlation between anxiety and HR. They did find, however, a significant increase in forearm blood flow following ECT and they suggested an inhibitory effect of depression on muscle blood flow, mediated by hypothalamic control (Pollit, 1965). Their group consisted solely of depressives who were tested before and after ECT and possible correlations between the psychological and physiological measures were sought within the same group, thus their results are not immediately comparable with those of Lader and Wing (1969) and Kelly and Walter (1969).

They also studied the same group of patients (Noble and Lader, 1972) in respect to EMG, SCL, SSCRs, forearm blood flow and salivation rate, along the endogenous-reactive dimension. The patients were allocated to each diagnostic group according to the criteria set by Carney, Roth and Garside (1965). The forearm extensor EMG, the forearm blood flow and the salivation rate were not significantly different in the two groups. However, the endogenous group had significantly lower SCLs and significantly fewer SSCRs than the reactive group. In this study there was a significant positive correlation (0.41) between retardation and endogenous depression. The results indicated that the concept of retarded depression was heuristically more useful than the concept of endogenous depression since the differences that emerged between the two groups were due, apparently, to the high proportion of retarded features in the endogenous group.

Significant differences in various physiological measures have been found whenever the retarded-agitated dichotomy was observed (Lader and Wing, 1969; Brown, 1970; Noble and Lader, 1971a and 1971b). Also, Overall et al. (1966b), Hollister et al. (1967) and Kay et al. (1969) showed that retardation

is positively correlated with favourable outcome and better response to drug treatment more consistently than any other endogenous feature, i.e. severe depression, feelings of guilt, nihilism, hopelessness, and delusions and hallucinations.

In a more recent study, Mirkin and Coppen (1980) using the Newcastle Scale (Carney, Roth and Garside, 1965) to identify patients with endogenous depression, yielded results similar to those of Noble and Lader (1972) in respect to EDA, but Toone, Cooke and Lader (1981) failed to replicate previous findings suggesting that higher SCLs and increased number of SSCRs are "typical of anxiety, whether viewed as a distinct syndrome (Lader and Wing, 1966; Raskin, 1975) or as a morbid characteristic in a mixed psychiatric population (Tan, 1964)." They also failed to find any significant differences between depressed patients and controls in respect to EDA. In their words "this is at variance with some previous reports and was unexpected." (p.503).

Lapierre and Butter (1980) also reported results that were not in agreement with the current views on the psychophysiology of depression. Their study included a group of 20 agitated depressives, 20 retarded depressives and 20 controls. Their diagnosis was based on the DSM-II and, according to these criteria, "All depressions were of endogenous characteristics and depressions of reactive origin were excluded from the study." (p.128). The results revealed differences in EDA between depressives and controls during relaxation but not during stimulation. Agitated and retarded depressives had significantly lower SCLs before and after sensory stimulation and, although the SCRs during the sensory stimulation did not differ among the groups, the retarded depressives showed more SCRs to the stimuli (photic, auditory and tactile), compared to controls.

It is possible that the discrepant results in this study were due to the selection criteria that were employed. The entire depressed group was characterized as endogenous and, since retardation is one of the main features of this category, the 'agitated' and 'retarded' groups in the study were more homogeneous than suggested in respect to the agitated-retarded dimension. Symptoms of anxiety, which differentiate agitated from retarded patients, have not been considered when patients were allocated to the 'agitated' and 'retarded' groups. The authors themselves pointed out that "Because clear physiological and motoric differentiation of agitated and retarded depression was not statistically demonstrable in spite of trends being suggested, a replication of the present study with more refined experimental procedure is imperative" (p.223).

Also worth noting is the fact that EDA was monitored in this study from the subjects' right hand and there is evidence to suggest that there are bilateral differences in EDA reflecting, apparently, differences in hemispheric functioning, the right hemisphere being subfunctional in depressives (Gruzelier and Venables, 1974; Dimond, Farrington and Johnson, 1976; Toone, Cooke and Lader, 1981) and, therefore, the left hand would have been, methodologically, the 'correct' hand to use in monitoring EDA in depressives; this is common practice in most studies since the subjects' non-preferred hand is used.

Giedke, Bolz and Heimann (1980) found no differences in SCLs and SSCRs between a group of depressed patients and healthy controls during rest. However, during a forechecking prior to the experimental session, patients did show lower SCLs and fewer SSCRs than controls, and the authors suggested that this may indicate that small changes in the experimental procedure

might be responsible for the varying results among different studies. Their results might have been confounded, however, by the fact that 9 out of the 18 patients in their sample were being treated at the time of the experiment with "antidepressives, neuroleptics, tranquillizers, hypnotics or combinations thereof." (p.92).

A possible correlation between clinical recovery and physiological recovery

Noble and Lader (1971b) investigated changes in EDA subsequent to ECT. First, they found a significant negative correlation between SCL, SSCRs and severity of depression. Second, no changes in reactivity to stress (mental arithmetic - Kelly, 1967) were found subsequent to ECT, but reactivity of SC to stress was significantly reduced. Third, agitated depressives had significantly more SSCRs than retarded depressives prior to ECT and subsequent to ECT both values converged towards a common mean. These changes, however, did not reach statistical significance. The authors suggested that 'physiological recovery' may not parallel clinical recovery. EDA did not correlate significantly with the anxiety assessments (self and experimenter ratings) but it did correlate with increased anxiety under the stress condition. Probably EDA is best at differentiating between conditions in the same group of subjects than in the same condition between different subjects (Noble and Lader, 1971b). Finally, there were two correlations the authors did draw attention to. First, sweat gland activity correlated negatively with weight loss and both, it has been suggested (Pollit, 1965), may be mediated by hypothalamic control which may be disturbed in depression. Second, sweat gland activity correlated negatively with retardation which may be "a key variable in interpreting the physiology of depression." (Noble and Lader, 1971b, p.68).

Stability of EDA and HR over time and regardless of clinical recovery has been reported in some studies (Mirkin and Coppen, 1980; Dawson, Schell and Catania, 1977; Storie, Doerr and Johnson, 1981). This stability has been interpreted as evidence for a trait description of depression rather than a state.

Breyer-Pfaff, Gaertner and Giedke (1982) and Heimann (1978), however, found significant shifts towards 'normality' in the EDA of depressed patients following clinical improvement in response to amitriptyline. It could be that the reported changes are due to the different treatment used in the latter studies. Mirkin and Coppen (1980) and Storrie, Doerr and Johnson (1981) did not specify the exact nature of the treatment their samples were receiving and Dawson, Schell and Catania (1977) used ECT on their group of depressives, although Bagg and Crookes (1966) did report increased sweat gland activity following ECT. Noble and Lader (1971b) who found limited support for 'physiological recovery' following ECT, suggested that "it is possible that in some patients physiological activity does not alter until, or even after, clinical recovery has taken place." (p.67).

In recapitulation, it seems that, overall, depression is accompanied by a general decrease in EDA compared to normals and the increased EDA found in agitated depressives is due to the high concomitant anxiety rather than to depression. Also, HR has been found to be higher in depressives compared to normal controls (Dawson, Schell and Catania, 1977) and increased HR has been linked with reactive (neurotic) depression (McCarron, 1973). In addition Bonis de and Freixa i Baqué (1978) reported a linear relationship between HR and depressed mood. The psychophysiological dimension supports certain clinical distinctions (agitated vs retarded) and points to possible neural

mechanisms (right hemisphere - hypothalamic mediation) involved in the manifestation of certain symptoms (weight loss, reduced sweat gland activity, retardation). Finally, the increased physiological responsivity of depressives reported in certain studies seems again to be linked with concomitant anxiety rather than depression and, furthermore, supports the agitated-retarded dichotomy since retarded depressives have been reported to be hyporesponsive compared to agitated depressives and normals.

Psychophysiological approaches have also been employed in the study of LH in humans, in an attempt to establish a link between the model and the actual depressive disorder, at the psychophysiological level.

The psychophysiology of Learned Helplessness

Direct psychophysiological comparison between controls, helpless subjects and nonclinically depressed subjects has been carried out by Gatchel, McKinney and Koebernick (1977). Previous to this study, Gatchel and Proctor (1976) reported differences in EDA of normal and helpless subjects.

In the 1976 study, Gatchel and Proctor continuously monitored EDA and HR from subjects participating in a standard LH experiment, using aversive noise in the pretreatment phase. As expected, the group treated with inescapable noise showed impaired anagram performance compared to the escapable and control groups. HR did not differentiate the three groups but decreased, following pretreatment, in all groups. Compared with the escapable group, the inescapable group showed reduced SCLs, reduced SCRs to the pretreatment noise and increased SSCRs during later pretreatment trials. The authors interpreted these results as indicating reduced autonomic arousal and

increased 'emotionality', in line with the alleged motivational deficit in LH and the rise in anxiety scores in helpless subjects.

The 1977 study (Gatchel, McKinney and Koebernick) included a third group of depressed college students. The presence of depression was assessed by the BDI. The results of this study indicated that there was no direct parallel between depression and LH in respect to EDA. "Learned Helplessness is associated with less electrodermal responding, while depression is associated with greater responding to uncontrollable events. The difference suggests that there may be different underlying mechanisms involved in these two phenomena." (p.30).

The LH model predicts that controllability reduces stress associated with shock, and this hypothesis has been tested, indirectly, by Geer, Davison and Gatchel (1970) who found that SCRs to electric shock habituated more rapidly in subjects who believed they had some control over the shock, while SCRs failed to habituate in subjects who believed they had no control over the shock. Also, perceived control subjects showed fewer SSCRs than perceived noncontrol. HR did not differ between the groups. The perception of control is central in LH theory and in this study, perception of control has been shown to reduce autonomic responding (fewer SSCRs) and reactivity (faster SCRs habituation rate) to aversive stimuli.

Finally, Suarez, Crowe and Adams (1978) tested depressed and nondepressed students and nonstudents in active and passive avoidance learning conditions. They found distinct differences in the performance of the depressed and nondepressed subjects. Depressed subjects were superior to nondepressed subjects in the passive avoidance task. In the active avoidance task the

depressed subjects made significantly more errors than nondepressed subjects. Physiological data suggested that depressed females were more emotionally responsive than males, having more SRRs and the effect was greater in the nonstudent sample. The data further suggested that the student sample was consistently more aroused than the nonstudent sample as indicated by their lower SRLs. Another important finding was that students were superior to nonstudents in the aquisition of the active avoidance response. Since students, as a whole, proved to be different than nonstudents in both the behavioural and physiological measures, the authors suggested that "extreme caution must be exercised when generalizing from a college student analog of depression." (Suarez, Crowe and Adams, 1978, p.31).

Reasons for the present study

The Gatchel studies, instead of clarifying, further confused the analogy between LH and depression. In 1976, Gatchel and Proctor stated that "the learned-helplessness group demonstrated lower tonic skin conductance levels, smaller phasic skin conductance responses, and more spontaneous electrodermal activity relative to the group pretreated with escapable tones. These are symptoms which some researchers have claimed to be associated with clinical depression." (p.27). In 1977, "findings indicated that learned helplessness was associated with less phasic skin conductance responding, while depression was associated with greater responding to uncontrollable aversive events." (Gatchel, McKinney and Koebernick, 1977, p.25). As has already been discussed, depression appears to be a nonunitary phenomenon and, according to the degree of concomitant anxiety, EDA in depressives can be either decreased (retarded depression) or increased (agitated depression) compared to the EDA of a normal population. Furthermore, Gatchel,

McKinney and Koebernick based their comparison on two analog populations and, as it has already been noted, analog populations should not be treated as if they represent the actual disorder, since there is a distinct qualitative difference between depressed patients and depressed normals, as well as between depressed students and depressed nonstudents (Suarez, Crowe and Adams, 1978; Weissman, Prusoff and Pincus, 1975; Beck, 1967). Gatchel and his colleagues made a methodological error in comparing two analog populations (depressed students and helpless students) as if one of them, the depressed students, represented the actual depressive disorder in general and the other, the helpless students, were the analog population in the study.

A replication of the Gatchel studies was felt necessary for three reasons. First, because a comparison was needed between the psychophysiology of LH and that of clinical depression. Second, because of the limited number of studies available on the psychophysiology of LH, replication and cross-validation of the reported findings was considered imperative (Smith, 1970). Third, the extenion of the Gatchel findings in a 'failure in a task' paradigm would be another necessary step towards demonstrating the generality of the findings and accepting them as actual physiological concomitants of LH in humans.

CHAPTER FOUR

A STUDY OF EDA AND HR AS CONCOMITANTS OF THE LH STATE

Rationale

The main premise of the LH theory of depression is that when subjects are exposed to uncontrollable situations they develop cogntive/motivational deficits and perform worse than control subjects in subsequent tasks. As a result of prolonged exposure to unctontrollability or even after a short but intensive shock (disaster, loss of loves ones, etc.), depression ensues. The LH state, it has been claimed, has certain distinct physiological concomitants that differentiate it from naturally occurring depression, despite the behavioural analogies between the two conditions claimed by the exponents of the LH theory.

EDA is regarded as an overall index of arousal or anxiety and severe depressive states have been associated with low SCLs and few SSCRs, while depressive states where anxiety is also present have been associated with higher than normal SCLs and increased number of SSCRs. The LH theory does not make explicit predictions about the psychophysiology of the LH state but since a cognitive/motivational deficit is postulated and since a "heightened state of emotionality" which "will increase and be replaced with depression" is also contended (Seligman, 1975, p.53-54.), certain hypotheses can be safely derived. First, helpless subjects should show an increase in SCL and SSCRs after the pretreatment phase since "reactive depressions are at the primary focus of the learned-helplessness model" (see p.20) and reactive depression has a high anxiety content (see chapters one and three). Second, lower SCLs

and fewer SSCRs should be found if the LH state is closer to endogenous depression since this category is characterized by retardation and, as a consequence, by reduced EDA.

Thus the main hypotheses of the present study were: First, helpless subjects would show either a general increase in EDA, if LH is an analog of reactive depression, or, helpless subjects would show a general decrease in EDA, if LH is an analog of endogenous depression, compared to control subjects, following failure in a task. Second, HR would increase in helpless subjects after the pretreatment phase since increased HR in reactive depression has been reported by McCarron (1973) and a linear relationship between depressive mood and HR has been reported by Bonis de and Freixa i Baqué (1978). HR, however, has not been positively linked, in other studies, with any depressive subtype (see chapter three) and is not regarded as reliable an index of depressive states as EDA. Finally, anagram performance deficits were expected to indicate the success of the LH treatment.

As secondary to the main hypotheses, mood was expected to deteriorate after the pretreatment phase in the helpless group with a possible elation in the success group.

Method

Three groups were included in the study. Group 1 was a naive control group which was exposed to neither success nor failure in the pretreatment phase; subjects were simply asked to 'take a break' and browse through some magazines instead. Group 2, the success group, had to play an electronic game, and Group 3, the failure group, had to play the unsolvable version

of the same game. EDA and HR were monitored, and mood was assessed before and after the pretreatment phase and, finally, all subjects had to solve a series of anagrams in order to assess the effectiveness of the LH induction procedure.

Subjects were recruited by class anouncements at Sunderland Polytechnic and participation was voluntary. Allocation of subjects to groups was random with the only restriction that there would be equal numbers of subjects in each of the three groups. In all, 37 subjects were recruited; 2 were excluded because the procedure was interrupted due to equipment failure and 1 for failing to succeed in the pretreatment task; 4 more subjects failed to complete the experiment by not attending the second session. So, the final sample consisted of 30 subjects, 10 in each group. In more detail:
Group 1 included 6 females and 4 males, mean age was 22.75 years - sd=5.60;
Group 2 included 4 females and 6 males, mean age was 21.17 years - sd=3.06;
Group 3 included 4 females and 6 males, mean age was 21.50 years - sd=4.69.

Because of the nature of the experiment some degree of deception was necessary. The subjects were told that they were to participate in a study concerned with the putative connection between mood states, performance and physiological responding. All subjects were shown the experimental room and the adjacent room where the recording equipment was housed, the requirements and procedure of the experiment were clearly explained to them and it was made clear that they could terminate the procedure and withdraw from the experiment at any moment, if they so wished. Finally, all subjects were debriefed after the experiment was completed, the deception was removed and any questions they had were answered.

Materials

Mood was assessed by using the Eight State Questionnaire (8SQ) developed by Barton, Cattell and Conner (1972), Cattell (1973a), and Barton, Cattell and Curran (1973). The 8SQ was chosen instead of the MAACL, which is more sensitive to transient mood changes, for two reasons. First, because of the detailed description obtained of the subject's mood - eight states are measured, i.e. anxiety, stress, depression, fatigue, guilt, extraversion and arousal. The eight states and their behavioural correlates are fully described in the manual for the 8SQ (Institute for Personality and Ability Testing, 1975 - see also appendix 1). Second, the reliability of the various Multiple Adjective Check Lists (MACLs) has been questioned by Cattell on theoretical and methodological grounds; 'R-technique' factor analysis, instead of the more appropriate 'dR-technique' and 'P-technique' has been used in their development (Cattell, 1963, 1966 and 1972). The so-called R-technique deals with correlations among variables over people and "the term can appropriately cover all kinds of correlational and covariance analysis of absolute, single occasion measures" (Cattell, 1972, p.123). In dR-technique one analyzes the change of scores of many individuals over two occasions and in P-technique one measures the change of scores of one individual over many occasions. So, R-technique counfounds trait and state responses while dR- and P-techniques do not, since they "knock the trait variance out altogether .... [and] the dR- or P-technique domain of measurement is thus purely concerned with the process of state change - the flow of mood in response to changing occasions." (Cattell, 1972, p.124). Mackay (1980) discussing the various mood measurement instruments, also points out the fact that, because of the employment of R-technique in their development, MACLs assess traits rather than states and this renders them inappropriate for the purposes

of the present study.

The 8SQ comes in two, fully interchangeable forms, A and B, one of which (Form A) was used before and the other (Form B) after the pretreatment phase, so the subjects did not have to answer identical questions twice in the space of 30 minutes.

Administration of the 8SQ was completely automated. The subjects had to type their answers into a computer (Sinclair ZX-81) thus the need for an answer sheet was eliminated. Raw scores were calculated by the program and later they were converted to standard ten (sten) scores using the Males+Females table provided in the 8SQ manual. Computer controlled administration of self-report questionnaires has been shown to be a legitimate method of obtaining data (Carr et al., 1981).

Electrodermal Activity and Heart Rate were recorded on paper using a LECTROMED MX6-6 polygraph. Skin resistance was measured using lead electrodes (tetrapolar) which are free from polarization effects (Venables and Martin, 1967). The electrodes were placed on the subject's left hand and, therefore, right cerebral hemisphere reactions to the stress of failure were being monitored. This was considered important since there is evidence to suggest that there are differences in responding to cognitive and emotional material between the two hemispheres and these are reflected in differential electrodermal responding of the two hands. The right hemisphere is more responsive to unpleasant material than the left and, therefore, seems a more potent indicator of the subject's emotional state (Dimond, Farrington and Johnson, 1976; Lacroix and Comper, 1979). Handedness was not taken into consideration because lateralization of functions of the

cerebral hemispheres is, generally, irrelevant to the subject's preferred hand (Kolb and Whishaw, 1980). The exact placement of the EDA electrodes is shown in figure 1, p.62.

Two electrodes were used for monitoring HR; one was placed on the subject's chest, on the left hand side, approximately 5cm above the lower rib, and the other on the right wrist. Neptic gel was used for the EDA and HR electrodes. Scoring was done manually from the chart and skin resistance data were converted to skin conductance before any further analysis was undertaken (conductance = 1/resistance or C = 1/R). The range used was 100Kohms for all subjects except one for whom the range was set at 250Kohms in order to keep the reading within the range of the pen recorder; SSRRs of 2Kohms or more were counted and are being referred to as SSCRs in the following discussion.

For the Helplessness Induction Procedure, a computer game, Golf was specially adapted from the original version supplied for the Sinclair ZX-81. At the bottom of a TV screen a solid line appears representing the golf course and a ball rests on the left hand side of it. The line is broken somewhere along its length in such a way as to represent a hole in the ground (see appendix 2, figure 2a). The object of the game is to decide on the distance between the ball and the hole, and to type into the computer the value of the force required - expressed in units of 35 to 70 - to make the ball land in the hole. The path of the ball is visible to the player (figure 2b). After a successful shot the player is informed of the number of shots he had altogether (figure 2c) and the game continues. There is an automatic reset and usually, but not always, the hole 'moves' along the course (i.e. along the line at the bottom of the screen) to simulate the approach towards the hole that occurs

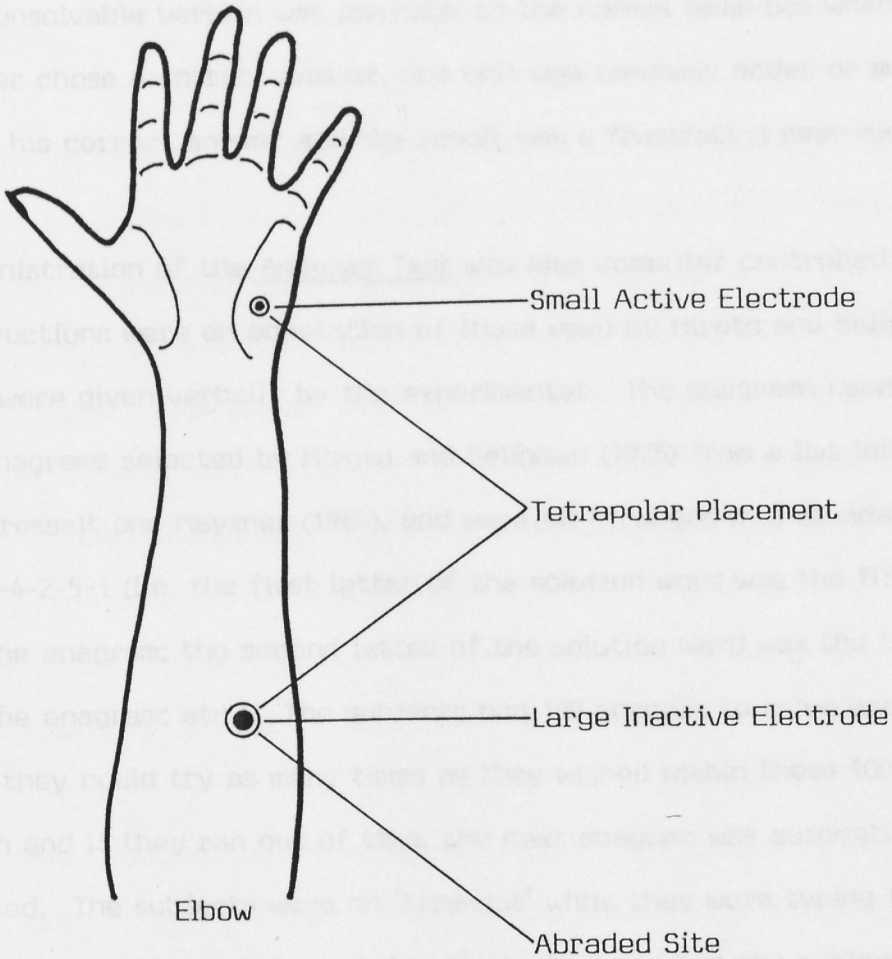

Figure 1. Electrode sites used (Sketch adapted from Venables and Christie, 1980, p.29).

in real golf. There is no time limit but the subjects were given 10 minutes to play and they were told before they started that 8, or more, hits within 10 minutes were well above average. One subject failed to attain the required minimum of 8 hits and was excluded from the sample.

The unsolvable version was identical to the normal game but whenever the player chose a correct answer, one unit was randomly added or subtracted from his correct answer and the result was a frustrating near miss (figure 2d).

Administration of the Anagram Test was also computer controlled. The instructions were an adaptation of those used by Hiroto and Seligman (1975) and were given verbally by the experimenter. The anagrams used were the 20 anagrams selected by Hiroto and Seligman (1975) from a list initially devised by Tresselt and Mayzner (1966), and were all arranged in a standard sequence of 3-4-2-5-1 (i.e. the first letter of the solution word was the fifth letter of the anagram; the second letter of the solution word was the third letter of the anagram; etc.). The subjects had 100 seconds to solve each anagram and they could try as many times as they wished within these 100 seconds. When and if they ran out of time, the next anagram was automatically presented. The subjects were on 'time-out' while they were typing their answer into the computer. The computer clock stopped when the subjects pressed "G" - for 'guess' - and the instruction "Guess now" appeared on the screen (see appendix 3, figures 3a and 3b). After each attempt the subjects were informed of their performance (figures 3c and 3d) and presented with the next anagram if their answer was correct, or if they ran out of time; otherwise the same anagram was presented again. Subjects could use the zero (0) key to 'pass' and proceed to the next anagram, if they so wished, before the elapse of the 100 seconds. In this case the phrase "You have passed"

appeared on the screen for a few seconds, before the next anagram was presented (figure 3e). None of the subjects communicated having any difficulty with the use of the computer keyboard (ZX-81) or problems related to the instructions given on the screen or by the experimenter.

Procedure

In order to familiarize the subjects with the environment of the experimental room, a trial session was arranged prior to the experiment proper. In the trial session the subjects were asked to complete Form A of the 8SQ and then the EDA and HR electrodes were attached and 5 minutes recording took place. Subjects allocated to Groups 2 and 3 were given a chance to play Golf for a couple of minutes in order to understand the requirements of the game. It was made certain that each subject had one successful shot before they left in order to avoid any later suspicions. The trial session lasted approximately 45 minutes and at the end the subjects were thanked and an appointment for the second and final session was made.

The second session took place within a week from the trial session and lasted approximately 2 hours. First, the subjects were asked to complete Form A again and then 10 minutes of recording followed. EDA electrode sites were first cleaned with mild detergent (Fairy liquid was used in all occasions) and then with surgical spirit. The site of the inactive electrode was abraded with cotton wool impregnated in surgical spirit until it reddened. During the recording the lights were dimmed and the subjects were asked to close their eyes and relax. The procedure described so far was familiar to the subjects from the trial session.

After the first recording session was over, Group 1 had to take a 10 minute break and Groups 2 and 3 played solvable and unsolvable Golf for the same length of time. Meanwhile the EDA electrodes were removed to avoid any possible hydration artifacts (Bundy and Mangan, 1979). Next, the subjects had to complete Form B and were told that despite the similarities between the two forms they should not consciously try to replicate their previous answers but to, simply, respond to the questions according to how they felt at the time. After completion of Form B another 10 minutes recording took place under the same relaxed conditions as the first one.

Last, all subjects had to solve the 20 anagrams. The anagram test concluded the session and after the necessary debriefing the subjects were thanked for their participation and left.

During the sessions the subjects were left alone in the experimental room and they could call the experimenter if something went wrong or if they, for any reason, felt uncomfortable. In practice, the subjects called for help only when a genuine reason, such as accidental misuse of the keyboard was present.

The individual stages of the trial and experimental sessions, in their sequence and approximate duration time, were as follows: trial session - 8SQ, Form A, 20 minutes; physiological recording; 5 minutes; introduction to the Golf game (Groups 2 and 3 only), 5 minutes; total time, including electrode site preparation etc., 45 minutes; experimental session - 8SQ, Form A, 20 minutes; physiological recording, 10 minutes; break (Group 1), Golf (Groups 2 and 3), 10 minutes; 8SQ, Form B, 20 minutes; physiological recording, 10 minutes; anagram test, maximum 35 minutes; debriefing; total time, including electrode

site preparation, debriefing, etc., 2 hours.

Data transformation

Some modifications were introduced in the scoring of the anagram test. In previous studies in LH, the subjects had to reach a criterion of three consecutive solutions in less than 15 seconds each. In the present study, the subjects had to solve all 20 anagrams since in earlier piloting (see appendix 4) it was noticed that some subjects did meet the criterion at some stage but, for some reason or other, their performance dropped in subsequent attempts. Similar reservations about the validity of the criterion as an indication of the subjects having discovered the pattern of the anagrams have been expressed by Tennen and Eller (1977). Instead, the total number of correct solutions, the mean time taken for each anagram solved, the number of times the subjects opted to pass (gave up), and the number of anagrams not solved (subjects ran out of time), were taken into account when the performances of the three groups were compared. Separate analyses were carried out for each of these variables and also, they were all combined to yield an overall Anagram Performance Index (API) using the formula: API = Number of Anagrams Solved / Mean Time per Anagram Solved + Number of Anagrams Passed + Time Out.

SR was first converted to SC and then these data, in μmhos, were transformed to log conductance (base 10). This transformation was necessary in order to obtain a better approximation to a normal distribution (Venables and Christie, 1980) although Edelberg (1972) claimed an over-correction for skewness when this transformation is carried out. SSCRs were transformed to log(X+1) for the same reasons.

All analyses were based on Winer (1962), Siegel (1965) and Lawson, Goldstein and Musty (1975). Raw data are given in appendix 5.

Results

Trial session One-way analyses of variance, separately carried out for each of the 8 mood states measured by the 8SQ, the SCL and the HR data obtained during the trial session (baseline data), revealed no differences between the three groups. The results of the analyses are summarized in tables two and three, p.68-69.

Experimental session Mood did not change in any of the three groups following the pretreatment phase, nor were there any significant differences between the groups either before or after the pretreatment phase, as revealed by two-way analyses of variance with one repeated measure, separately carried out for each of the 8 mood states (see tables four and five, p.70-72, for summary).

Tests for the homogeneity of the sources of variation of the physiological data were carried out before any further analysis was undertaken. The results showed the cell variances to be homogeneous (see table six, p.73, for summary). Subsequent two-way analyses of variance with one repeated measure were separately carried out for logSCL, SSCRs - log(X+1) and HR.

The analysis of the logSCL data showed a significant treatments x before-after measures effect ($F = 3.86 > F.95(2,27) = 3.36$) and a subsequent analysis of simple effects showed this to be due to a significant increase in SCL in Group 3 ($F = 9.50 > F.99(1,27) = 7.69$). No changes in SCL following the pre-

Table two

Means ($\bar{x}$) and standard deviations (s) of baseline variables

Variable	Group 1 baseline	Group 2 baseline	Group 3 baseline
Anxiety	$\bar{x}$=5.10 s=2.18	4.50 2.27	4.90 2.85
Stress	$\bar{x}$=4.70 s=3.02	5.40 2.63	3.90 3.07
Depression	$\bar{x}$=4.70 s=1.57	5.70 2.50	5.10 2.96
Regression	$\bar{x}$=4.80 s=2.04	5.10 2.51	4.90 2.38
Fatigue	$\bar{x}$=4.10 s=2.42	5.00 3.20	4.50 3.14
Guilt	$\bar{x}$=4.70 s=1.64	5.00 2.36	5.50 2.37
Extraversion	$\bar{x}$=6.00 s=2.05	5.30 2.91	5.80 2.39
Arousal	$\bar{x}$=6.70 s=1.49	5.80 2.62	6.00 2.05
LogSCL (μmhos)	$\bar{x}$=1.38 s=0.31	1.38 0.30	1.19 0.38
Heart Rate (bpm)	$\bar{x}$=71.80 s=10.29	69.20 11.17	70.30 9.43

Table three

Summary of Analysis of Variance (baseline data)

	Source of variation	SS	df	MS	F
Anxiety	between groups	1.87	2	0.94	0.16
	experimental error	162.30	27	6.01	
Stress	between groups	11.27	2	5.34	0.66
	experimental error	229.40	27	8.50	
Depression	between groups	5.07	2	2.54	0.44
	experimental error	157.10	27	5.82	
Regression	between groups	0.47	2	0.24	0.04
	experimental error	145.40	27	5.39	
Fatigue	between groups	4.07	2	2.04	0.24
	experimental error	233.40	27	8.64	
Guilt	between groups	3.27	2	1.64	0.36
	experimental error	124.60	27	4.61	
Extraversion	between groups	2.60	2	1.30	0.21
	experimental error	165.70	27	6.14	
Arousal	between groups	4.47	2	2.24	0.51
	experimental error	119.70	27	4.43	
LogSCL (µmhos)	between groups	0.24	2	0.12	1.09
	experimental error	2.98	27	0.11	
Heart Rate (bpm)	between groups	34.07	2	17.04	0.16
	experimental error	2877.30	27	106.57	

Table four

Means ($\tilde{x}$) and standard deviations (s) of experimental variables

	Group 1		Group 2		Group 3	
Variable	before	after	before	after	before	after
Anxiety	$\tilde{x}$=4.90	5.00	4.20	4.40	3.50	3.70
	s=2.56	2.16	3.19	2.72	1.58	1.83
Stress	$\tilde{x}$=4.70	4.60	4.20	4.30	4.10	4.20
	s=2.79	2.46	2.78	2.63	2.51	1.99
Depression	$\tilde{x}$=5.20	5.00	5.50	4.50	5.20	4.50
	s=2.39	2.40	2.80	2.51	2.49	1.72
Regression	$\tilde{x}$=5.10	5.60	5.30	4.90	4.30	4.70
	s=2.02	2.72	2.63	3.25	1.64	1.49
Fatigue	$\tilde{x}$=4.70	4.60	5.20	5.30	5.20	5.00
	s=2.16	2.01	2.90	2.54	2.30	2.49
Guilt	$\tilde{x}$=5.20	5.50	4.00	4.80	4.90	5.10
	s=2.53	2.42	2.94	2.86	2.08	2.13
Extraversion	$\tilde{x}$=5.60	5.70	5.40	5.40	5.30	5.70
	s=2.95	2.11	2.84	2.76	2.31	2.21
Arousal	$\tilde{x}$=5.60	5.90	6.00	6.00	5.20	5.20
	s=1.48	2.60	2.40	2.70	1.87	1.99
LogSCL	$\tilde{x}$=1.14	1.08	1.08	1.08	0.83	1.02
(μmhos)	s=0.38	0.42	0.28	0.24	0.39	0.32
SSCRs -	$\tilde{x}$=0.42	0.41	0.40	0.45	0.49	0.68
log(X+1)	s=0.49	0.44	0.52	0.50	0.64	0.67
Heart Rate	$\tilde{x}$=70.60	68.60	69.30	65.40	67.70	65.10
(bpm)	s= 8.60	9.30	9.65	10.18	7.67	9.11

Table five

Summary of Analysis of Variance of 8SQ data

	Source of variation	SS	df	MS	F
Anxiety	between subjects	295.68	29		
	A (treatments)	18.23	2	9.12	0.89
	Ss within groups	277.45	27	10.28	
	within subjects	34.50	30		
	B (before - after)	0.42	1	0.42	0.33
	AB	0.03	2	0.02	0.01
	B x Ss within groups	34.05	27	1.26	
Stress	between subjects	309.15	29		
	A (treatments)	2.80	2	1.40	0.12
	Ss within groups	306.35	27	11.35	
	within subjects	42.50	30		
	B (before - after)	0.02	1	0.02	0.01
	AB	0.13	2	0.07	0.04
	B x Ss within groups	42.35	27	1.57	
Depression	between subjects	255.48	29		
	A (treatments)	0.63	2	0.32	0.03
	Ss within groups	254.85	27	9.44	
	within subjects	65.50	30		
	B (before -after)	6.02	1	6.02	2.81
	AB	1.63	2	0.82	0.38
	B x Ss within groups	57.85	27	2.14	
Regression	between subjects	267.48	29		
	A (treatments)	7.63	2	3.82	0.40
	Ss within groups	259.85	27	9.62	
	within subjects	47.50	30		
	B (before - after)	0.42	1	0.42	0.25
	AB	2.43	2	1.22	0.74
	B x Ss within groups	44.65	27	1.65	

Table five (continued)

Summary of Analysis of Variance of 8SQ data

	Source of variation	SS	df	MS	F
Fatigue	between subjects	282.00	29		
	A (treatments)	3.90	2	1.95	0.19
	Ss within groups	278.10	27	10.30	
	within subjects	38.00	30		
	B (before - after)	0.07	1	0.07	0.05
	AB	0.23	2	0.12	0.08
	B x Ss within groups	37.70	27	1.40	
Guilt	between subjects	316.08	29		
	A (treatments)	9.23	2	4.62	0.41
	Ss within groups	306.85	27	11.36	
	within subjects	38.50	30		
	B (before - after)	2.82	1	2.82	2.19
	AB	1.03	2	0.52	0.41
	B x Ss within groups	34.65	27	1.28	
Extraversion	between subjects	295.48	29		
	A (treatments)	0.63	2	0.32	0.03
	Ss within groups	294.85	27	10.92	
	within subjects	57.50	30		
	B (before - after)	0.42	1	0.42	0.20
	AB	0.43	2	0.22	0.10
	B x Ss within groups	56.65	27	2.10	
Arousal	between subjects	235.68	29		
	A (treatments)	8.63	2	4.32	0.51
	Ss within groups	227.05	27	8.41	
	within subjects	38.25	30		
	B (before - after)	0.15	1	0.15	0.11
	AB	0.10	2	0.05	0.04
	B x Ss within groups	38.00	27	1.41	

Table six

Tests for homogeneity of sources of variation of logSCL, SSCRs - log(X+1), and Heart Rate

LogSCL SSsubjects within groups: Fmax = 2.27 < Fmax.95(3,9) = 5.34

SSB x subjects within groups: Fmax = 2.25 < Fmax.95(3,9) = 5.34

SSCRs - log(X+1) SSsubjects within groups: Fmax = 2.26 < Fmax.95(3,9) = 5.34

SSB x subjects within groups: Fmax = 1.09 < Fmax.95(3,9) = 5.34

Heart Rate SSsubjects within groups: Fmax = 1.40 < Fmax.95(3,9) = 5.34

SSB x subjects within groups: Fmax = 1.22 < Fmax.95(3,9) = 5.34

Table seven

Summary of Analysis of Variance of logSCL

	Source of variation	SS	df	MS	F
LogSCL	between subjects	6.11	29		
(μmhos)	A (treatments)	0.41	2	0.21	0.98
	Ss within groups	5.70	27	0.21	
	within subjects	0.82	30		
	B (before - after)	0.03	1	0.03	1.30
	AB	0.18	2	0.09	3.86*
	B x Ss within groups	0.61	27	0.02	

* F = 3.86 > F.95(2,27) = 3.36

Table eight

Test for homogeneity of Error Variance of logSCL (μmhos)

Fmax = 3.08 < Fmax.95(6,9) = 7.80

Analysis of Simple Effects of logSCL (μmhos)

Source of variation	SS	df	MS	F
A (treatments)	0.41	2	0.21	
A for b1	0.56	2	0.28	2.33
A for b2	0.03	2	0.02	0.17
SSsubjects within groups + B x subjects within groups	6.31	54	0.12	
B (before - after)	0.31	1	0.03	
B for a1	0.02	1	0.02	1.00
B for a2	0.000026 (negligible)			
B for a3	0.19	1	0.19	9.50*
B x subjects within groups	0.61	27	0.02	

* F = 9.50 > F.99(1,27) = 7.69

Table nine

Summary of Analysis of Variance of SSCRs - log(X+1)

	Source of variation	SS	df	MS	F
SSCRs -	between subjects	13.70	29		
log(X+1)	A (treatments)	0.35	2	0.18	0.36
	Ss within groups	13.35	27	0.49	
	within subjects	3.23	30		
	B (before - after)	0.10	1	0.10	0.88
	AB	0.10	2	0.05	0.46
	B x Ss within groups	3.03	27	0.11	

Table ten

Summary of Analysis of Variance of Heart Rate (bpm)

	Source of variation	SS	df	MS	F
Heart Rate	between subjects	4468.88	29		
(bpm)	A (treatments)	108.03	2	54.02	0.33
	Ss within groups	4360.65	27	161.51	
	within subjects	261.50	30		
	B (before - after)	120.42	1	120.42	24.70*
	AB	9.43	2	4.72	0.97
	B x Ss within groups	131.65	27	4.88	

* $F = 24.70 > F.99(2,27) = 5.49$

Table eleven

Means ($\tilde{x}$) and standard deviations (s) of Anagram Test data

Variable	Group 1	Group 2	Group 3
Mean Time per Anagram Solved (seconds)	$\tilde{x}$=24.57 s=12.58	20.69 10.45	28.99 16.80
Anagrams Solved	$\tilde{x}$=16.50 s= 4.65	17.40 3.06	13.00 6.53
Anagrams Passed	$\tilde{x}$= 1.90 s= 3.70	0.90 1.60	1.00 1.41
Time Out	$\tilde{x}$= 1.60 s=2.46	1.70 1.57	6.00 5.64
Anagram Performance Index	$\tilde{x}$= 0.86 s= 0.55	0.98 0.48	0.66 0.67

Table twelve

Summary of Analysis of Variance of Anagram Performance Index

	Source of variation	SS	df	MS	F
Anagram Performance Index	between groups	0.54	2	0.27	0.82
	experimental error	8.82	27	0.33	

treatment phase emerged in Groups 1 and 2. There were no significant differences between the three groups either before or after the pretreatment phase. The results are summarized in tables seven and eight, p.73-74.

SSCRs did not differentiate the three groups either before or after the pretreatment phase, and there were no significant changes within the groups. The results of the analysis are summarized in table nine, p.75.

HR decreased significantly following the pretreatment phase in all three groups as revealed by a two-way analysis of variance with one repeated measure which showed a significant treatments effect ($F = 24.40 > F.99(2,27) = 5.49$ - see table ten, p.75, for summary).

Anagram performance did not differ significantly between the three groups. Although subjects in Group 3 took longer to solve the anagrams and solved less anagrams than the subjects in Groups 1 and 2 (see table eleven, p.76), A Kruskal-Wallis one-way analysis of variance by ranks did not show these differences to be statistically significant (number of anagrams solved: $H = 2.16 < x^2.95 = 5.99$; mean time per anagram solved: $H = 2.42 < x^2.95 = 5.99$; number of anagrams passed: $H = 0.09 < x^2.95 = 5.99$; time out: $H = 2.26 < x^2.95 = 5.99$). The API values approximated a normal distribution ($Fmax = 1.91 < Fmax.95(3,9) = 5.34$) and an one-way analysis of variance showed no significant differences (see table twelve, p.76, for summary).

Seligman's criterion of three consecutive solutions in less than 15 seconds could not be applied because three subjects in Group 1, three in Group 2, and five in Group 3 did not reach it at all. Furthermore, three subjects in Group 1, five subjects in Group 2, and three subjects in Group 3 reached

the criterion at some stage and then slowed down later in the test.

Discussion

The absence of mood change contrasted with previous findings and was not expected since earlier piloting (see appendix 4) indicated that the 8SQ is sensitive enough to small, transient mood changes and also that the procedure used to induce helplessness was adequate in inducing a limited mood change. Yet, this finding is not unique. Failure to find any significant mood changes following pretreatment with inescapable aversive noise has been reported by Cole and Coyne (1977), and Miller and Seligman (1975) found no change in the depression scores of subjects after pretreatment with inescapable aversive noise, using the MAACL. On the other hand, mood changes, whenever found, were the result of inescapable aversive noise and not failure in a task. Also, these changes were rather shortlived (Gatchel, Paulus and Maples, 1975), and this is the case with mood induced by means of other mood induction procedures (Mukherji, Abramson and Martin, 1982).

It could well be that the limited mood change indicated in the pilot study, was an artifact of the procedure; only mood was assessed, before and after the task, and this prompted some of the subjects to ask: "are you trying to see if my mood has changed?". In the final experiment, the physiological recording has spaced the two mood assessments further and probably made the connection between the task and the 'expected' mood change less obvious, and the subjects less 'aware' of the purpose of the task. Form B could have been regarded by the subjects, in this case, as collection of additional data instead of different data. This was not the case in previous studies, (Miller and Seligman, 1975; Catchel, Paulus and Maples, 1975), where the same check

list was used twice, thus making the purpose of the repeated measure rather obvious to the subjects.

The absence of anagram performance deficits also contrasted with previous findings that failure in a task causes subsequent decrements in anagram performance. Again, this finding is not unique. Bihm, McWhirter and Kidda (1982) who also failed to find any anagram performance deficits following failure in a problem solving task, concluded that "with slight modifications in the experimental procedure, learned helplessness often proves to be an elusive phenomenon." (p.153). These modifications can be anything from modifying the scoring procedure of the anagram test (Tennen and Eller, 1977) to manipulating the stress associated with the failure in the pretreatment task. Samuel, Baynes and Sabeh (1978) found facilitation in anagram performance after failure in a relaxed environment - subjects were told that they were to do some "word puzzles" and to "relax and take it easy" - and permance deficits only after failure in a stressful environment - subjects were told to take the tests very seriously because previous research had showed them to be an indication of intelligence. Williams and Teasdale (1982) also manipulated task importance and obtained similar results. In the present study care was taken to eliminate, as far as possible, any clues concerning the possible significance of the task which was presented to the subjects as a necessary step in assessing the 'relationship between mood, performance and physiological responding'.

Nevertheless, casual observation gave the impression that subjects in Group 3 were affected by their failure enough to feel apologetic for their failure, blame the "stuffy" room for their poor performance, and even angrily express annoyance towards the experimenter for trying to make them feel bad.

There were also obvious signs of relief when they were later told about the deception. Similar behavioural effects have been noticed by the experimenter in the Bihm, McWhirter and Kidda (1982) study, although these manifestations of frustration and anger were not quantified. Perhaps a phenomenological analysis could provide information concerning the effects of failure such as is available for other mood induction procedures (Natale and Bolan, 1980; Goodwin and Williams, 1982).

It is probaly of some relevance that SCLs increased in Group 3 after the pretreatment phase, thus indicating an increase in arousal or anxiety; SCL has been reported to be responsive to stress (Miller, 1968). This increase would suggest that LH is similar physiologically to 'reactive' depression if only the subjects in Group 3 also exhibited the expected anagram performance deficits and mood deterioration.

Gatchel and Proctor (1976) and Gatchel, McKinney and Koebernick (1977) reported increased SCLs in their escapable groups (depressed and nondepressed in the 1977 study) and interpreted this as indicating greater task involvement and motivation. It should be pointed out, however, that their data were obtained by continuous monitoring throughout the experimental session and could be an artifact of hydration, the electrolyte used*, the motor activity of subjects - subjects seem to exhibit greater motor activity after failure - or a combination of these factors. For these reasons their results are not directly comparable with those of the present study in which all

* They used Johnson K-Y jelly while Neptic gel was used in the present study. Results obtained using different electrolytes are not comparable, in the absolute sense, and purpose-made gel (0.05M NaCl) has been recommended if quantitative comparisons are to be made between studies (Grey and Smith, 1984; see also Venables and Christie, 1980).

*
It should be pointed out, however, that certain limitations in the present design might have contributed to the absence of significant findings in respect to anagram performance and mood change. First, the helplessness induction procedure was of rather short duration (10 minutes) and failure was limited to one task only. Had the subjects experienced repeated failure in two or three different tasks the helplessness effects whould have probably been evident in the subsequent anagram performance. Second, the 8SQ is not designed to detect small, transient mood changes - despite the limited evidence from the pilot study - and probably a visual analog scale whould have suited the purposes of the present study best. Also, even though an attempt was made to cut short the time of its completion by computerizing the procedure, the mean duration time for completing the questionnaire was twice as long (20 minutes) as the helplessness induction procedure thus requiring the subjects to spent more time overall describing their feelings than being in direct contact with the experimental task.

Finally, the helplessness induction procedure could have been designed as to create the impression that the pretreatment task was reflecting differences in intelligence between the subjects thus making the subjects more vulnerable to helplessness through manipulating their attributions.

physiological data were obtained during a relaxation period.

Since the number of SSCRs did not change in any of the groups following pretreatment, the significance of the finding that SCLs increased in Group 3 becomes even weaker. EDA in Group 3 should have either increased or decreased following failure and this was not the case since only SCL showed a significant change. The decrease of HR in all groups disconfirmed the hypothesis that HR whould show an increase in Group 3 following failure.

The results overall failed to demonstrate a positive difference between the three groups in the present study. Subjects in Group 3 did not differ, in respect to anagram performance, from subjects in Groups 1 and 2 so, it seems, helplessness has not been induced. The increased SCLs in Group 3, which might indicate some heightened state of arousal or anxiety, suggest that failure is not without consequences for the subjects but one can hardly call that 'depression' or even 'helplessness'. Also, the physilogical data did not point to the presence of a distinctly different physiological state in any of the three groups following pretreatment, the presence of elevated SCLs in Group 3 after pretreatment being rather limited evidence on which such an assertion could be safely made. *

Perhaps a replication of the present experiment with a larger, not strictly student, sample and employing aversive noise in the pretreatment phase might yield positive, more reliable results. In any case, despite previous claims, and in view of the present findings, the present writer doubts whether in the space of two hours, or less, that a typical LH experiment lasts, physiological changes would have taken place to such an extent as to reliably differentiate 'helpless' from control subjects.

Conclusions

The aim of the present study was to assess claims that LH in humans has certain distinct psychophysiological concomitants which differentiate it, as a state, from clinical depression, and normality.

Doubt has been expressed about the validity of these claims on the grounds that they were based on results from studies on analog populations and the experiment reported here was designed to overcome some of the drawbacks of these studies. Hypotheses concerning the EDA and HR of 'helpless' subjects were formulated, based on the current knowledge of the behavioural significance of these variables and the psychophysiology of clinical depression, in order to assess the analogy of the LH state with clinical depression at a psychophysiological level. None of these hypotheses were confirmed and, furthermore, the expected mood changes in the 'helpless' subjects and the anagram performance deficits did not take place.

The review of the literature, although limited, revealed a rather perplexed state of affairs that does not justify the acceptance of the LH model as a valid analog of clinical depression. The present writer, like Cogan and Frye (1981) before him, "seems inescapably drawn to the depressing conclusion that LH, in humans, is not a reliable enough phenomenon to be considered an analog of depression" (adapted from Cogan and Frye, 1981, p.288). Nevertheless, especially since the LH model has, by now, found its way into introductory textbooks (e.g. Coleman, 1976; Kleinmuntz, 1980), a re-examination of the model and some of its basic assumptions seems imperative.

CHAPTER FIVE

WHICH DEPRESSION IS LH THE MODEL OF?

The question concerning the analogy between the LH state and depression is still open. Problems with the LH phenomenon as such, its replicability and robustness, and its generality across situations have already been discussed (see chapters two and four). There are, however, some more fundamental issues worth reconsidering.

The LH model has been accepted by many, it seems, as a viable model of depression and the arguments have developed, over the years, around the similarities between helpless subjects - an 'analog' population themselves - and analog populations for most of the time. Most of the LH studies have been carried out using the human equivalent of the white rat, i.e. the college student. College students are not a representative sample of the population at large, let alone a clinical population. Suarez, Crowe and Adams (1975) found significant differences between college students and non-students in active and passive avoidance tasks, subjective ratings of aversive noise, and EDA, and questioned the utility of college student analogs in the study of depression. Weissman, Prusoff and Pincus (1975) found that "Normal depression is characterized by a relative absence of somatic complaints and somatic anxiety." (p.15). and that "behaviour, not mood, distinguishes clinical depression from normal unhappiness." (p.16).

In addition, the most common instrument used in the selection of subjects as depressed and nondepressed in LH studies was the BDI. This is an instrument designed to assess severity of depression already diagnosed as clinical

and not as a substitute for clinical diagnosis; in one study even a short form was used (Seligman et al., 1979). Hammen (1980) found that "the BDI indiscriminately identified students who displayed either stable or unstable depression and who subsequently revealed considerable variation in diagnosis and symptoms patterns." (p.128). Lewinsohn and Teri (1982) also found that "classification of subjects as depressed cannot be accomplished by reliance on self-report measures alone; almost half of those classified depressed by self-report were not judged clinically depressed on the basis of clinical interview." (p.591).

The conclusions drawn from such studies may support, at best, an analogy between LH and nonclinical depression in college students. It is rather risky to regard results from such studies as supporting an analogy between LH and clinical depression. Clinical depression, on the other hand, is not a unitary phenomenon (see chapters one and three). Seligman, nevertheless, has produced a comparative list of symptoms, causes, cure and prevention common to LH and depression in general (see table one, p.21).

Passivity comes first in his list of symptoms common to LH and depression. Passivity and motor retardation are seen in endogenous depressions only and this leaves a large proportion of the clinically depressed population (reactive or neurotic depressives) completely at odds with this assertion. Time course is another putative similarity between LH and depression. LH in humans is a rather shortlived phenomenon and, for ethical reasons, the LH effect cannot be enhanced by repeated exposure to uncontrollability, as it can be done with animals. In dogs, the alleged time course is one week long at the best of times (Seligman and Groves, 1970; Seligman, Rosellini and Kozak, 1975) and similar duration of the LH effect has been reported in rats

(Sherman, Sacquitne and Petty, 1982). Nevertheless, Hiroto and Seligman (1975) suggested that LH might be an induced 'trait', a suggestion that is not viable considering the transience of LH in animals and humans. Furthermore, the time course in clinical depression can be anything over three months (Bebbington, 1982) and in the case of neurotic depression this 'time course' might not even exist at all (Wolpe, 1979). The lack of aggression in helpless subjects does not line up with the reported increase in hostility scores in the MAACL (Miller and Seligman, 1975; Gatchel, Paulus and Maples, 1975) and introjected hostility is not a universal symptom in depressive states (Grinker et al., 1961; Kay et al., 1969; Paykel, 1971; Paykel et al., 1973). Weight loss, appetite loss, social and sexual deficits have yet to be demonstrated in human LH; demonstration of noradrenaline depletion and cholinergic activity is also lacking. Again, weight loss is not always found in depression and the role of neurotransmitters is far from clear.

When it comes to causes, Seligman is even more categorical. LH is caused by "learning that responding and reinforcement are independent." Similarly, depression is caused by the "belief that responding is useless." Rippere (1977) made the following comment on Seligman's rather casual list of 'symptoms', 'causes', 'cure', and 'prevention': "One can probably excuse the mixing up of subjective 'symptoms' with objective signs .... But to declare the 'belief that responding is useless' as 'cause' of depression is to blur even the more important distinction between empirical observation and speculative interpretation." (p.208). Seligman was adamant. In direct reply to these criticisms he insisted that "The learned helplessness model of depression is an attempt to delineate the underlying state of affairs which causes many depressions." (Seligman, 1977, p.210). This was a rather ambitious statement and the present writer has been unable to find any account by Seligman, or any other pro-

ponent of the LH theory for that matter, that explicitly delineates the 'underlying state of affairs' and integrates the diverse material in the field.

The problem with the original model is that it predicts patterns of symptoms that simply do not occur (Depue and Monroe, 1978; Costello, 1978; Buchwald, Coyne and Cole, 1978). One, in such a case, has to either accept a model based on animal and student analog studies or the plethora of reports indicating the non-unitary, multifaceted nature of the depressive disorders. And even if one accepts the hypothesis of depression being a final, common pathway that once attained becomes autonomous and assumes the dimensions of illness (Akiskal and McKinney, 1973 and 1975) the proposal of the 'belief that responding is useless' as the 'cause' of depression is still an over-simplification to say the least.

As far as cure is concerned, the LH model has either not generated much research or the results have been, overall, inconclusive and therefore not reported. At least this is what is reflected in the review by Blaney (1981) who noted that "there appears to be but one nonanalog study assessing an intervention that could be said to be uniquely a helplessness treatment." (p.26). Blaney was referring to the study by Glass (1978) who used a technique analogous to "dragging helpless dogs across the shuttle box" (p.23). There were three groups in the study. A helplessness treatment group, prompted to engage in behaviours within the repertoir of the subjects in it, so that they would learn that responding and outcome are related, a waiting list control group and a group that received support and clarification of feelings and engaged in self-monitoring. The helplessness group showed, overall, the most improvement but not all comparisons between the three groups were significant and, most important, the cognitions of helplessness failed to diffe-

rentiate the treatment groups, thus leaving the reasons for the greater improvement of the leplessness training group unclear.

The problem with the reformulated model

The main problem with the reformulated model is that it incorporates a lot of rather speculative concepts about the depressed persons' attributions concerning positive and negative outcomes (Lavelle, Metalsky and Coyne, 1979). The model predicts that LH ensues when the subjects make relatively internal, stable and global attributions for netative outcomes, whereas it was externality which was associated with succeptibility to helplessness in the early days (Hiroto, 1974; Seligman, 1975). The attributional processes predicted by the reformulated model have not always been verified; conflicting results have been reported by Alloy, Abramson and Viscusi (1981) and Mukherji, Abramson and Martin (1982). Problems with the LH procedure in inducing mood change in subjects, apparently, made the latter to adopt the Velten technique (Velten, 1968) to induce mood in their subjects in order to test the hypotheses generated by the reformulated LH model.

Once the predicted changes in the attributional patterns had not been found the authors stated that "It is possible that the depressive mood induction did change attributional patterns but that the depressed mood wore off before the subjects completed the Attributional Styles Scales, and consequently, changes in attributional patterns were reflected for the first, but not the last, items of the scale. A reanalysis of the data separating the first half of the positive and negative items on the scale from the second half provided no support for the hypothesis." (Mukherji, Abramson and Martin, 1982, p.19).

Furthermore, although the concept of locus of control is 20 years old (Rotter, 1966), its relationship to depression is not at all clear. The positive correlation often found between depression and scores on externality in the Rotter (1966), Internal-External scale disappears when the depressive element in the external statements is statistically controlled (Aiken and Baucom, 1982; Lamont, 1972a and 1972b).

LH fails to come close to depression even at the limited scope of a model for the simple reason that there is no such disorder as Seligman postulates, which has one cause and fits his rather loose discription. It seems, after all, that the model "has attracted considerable attention, perhaps as a result of the name 'learned helplessness' rather than as a result of scientific strength." (Gelder, Gath and Mayou, 1983, p.209).

Despite the criticism raised so far, the present writer is certain that, under certain experimental conditions subjects who have previously experienced failure in a task will show certain deficits, compared to subjects who have experienced success, in some later test. To doubt this is to doubt that failure has any effects whatsoever on the person who experiences it. But, to regard failure in a task, or even the perception of uncontrollability as <u>the cause</u> of one of the most diverse mental disorders simply because experimental evidence points to anagram performance deficits after failure in a task in a laboratory setting is dangerous.

Furthermore, Mayr (1961) noted that "causality in biological systems is not predictive, or at best it only statistically predictive" (p.1056). The validity of the experimental method has been repeatedly questioned when applied to human research (Cameron, 1963; Smith, 1970; Bowers, 1973; Phares and

Lamiell, 1977). It would seem imperative that an alternative way of approaching the human subject is adopted, even if that means 'going back' to case studies and 'subjective' phenomenological descriptions of the human condition.

As a general conclusion the present writer feels compelled to quote, yet again, Phares and Lamiell (1977, p.115) who wrote that "So much research these days either involves routinized administration of questionnaires to groups or else unimaginative laboratory studies. Small wonder that many students now prefer to analyze data rather than run subjects. Somehow we have to get back to research that involves something other than fleeting contacts with subjects, deductions from super-ANOVA tables to the exclusion of experience with people, and the unrealities of the laboratory that may engage hierarchies of needs and congitions wholly different from what we are trying to capture," and add that these comments are, sadly, still valid today.

APPENDIX 1

Description of the mood states measured by the Eight State Questionnaire

Anxiety Subject describes himself as worried, easily rattled, tense, emotionally upset, easily angered, high strung, easily annoyed. (More common frailties admitted, greater tending to agree, less confident of skill in untried performance, fewer questionable reading preferences, higher susceptibility to embarrassment, lower accuracy in checking numbers.)*

Stress Subject describes himself as feeling a lot of pressure, unable to take time off and relax, constantly on the go, feeling hectic, experiencing great strain, unhappy with own performance, experiencing lots of demands. (Low motor perceptual rigidity, better at memorizing meaningless material, high ratio of threatening objects seen in unstructured drawings.)*

Depression Subject describes himself as unhappy, disagreeable, pessimistic, in poor spirits, disapointed. (Poorer at memorizing meaningful material, low ratio of fluency regarding self relative to other topics.)*

Regression Subject describes himself as confused, unorganized, unable to concentrate, experiencing difficulty coping, acting impulsively. (Greater suggestibility, lower ratio of accuracy to speed, lower accuracy in spatial judgement, poorer two-hand coordination, higher score on neurotic symptom checklist, lower speed of Gestalt closure.)*

Fatigue Subject describes himself as exhausted, no energy, sluggish,

tired, needing rest, weary, below par in performace. (Greater variability in accuracy, rapid reversible perspective.)*

Guilt Subject describes himself as regretful, concerned about own misdeeds, experiencing difficulties sleeping, unkind, dissatisfied with self.

Extraversion Subject describes himself as sociable, outgoing, adventuresome, talkative, enthusiastic. (Greater number of objects perceived in unstructured drawings, less tendency to agree, less authority submission, more confident assumption of skill in untried performance.)*

Arousal Subject describes himself as alert, keyed up, excited, stimulated, keen and sharp senses.

* Behavioural correlates in objective test domain.

Adapted from the Manual for the Eight State Questionnaire [8SQ] Forms A and B, Institute for Personality and Ability Testing, 1975.

APPENDIX 2

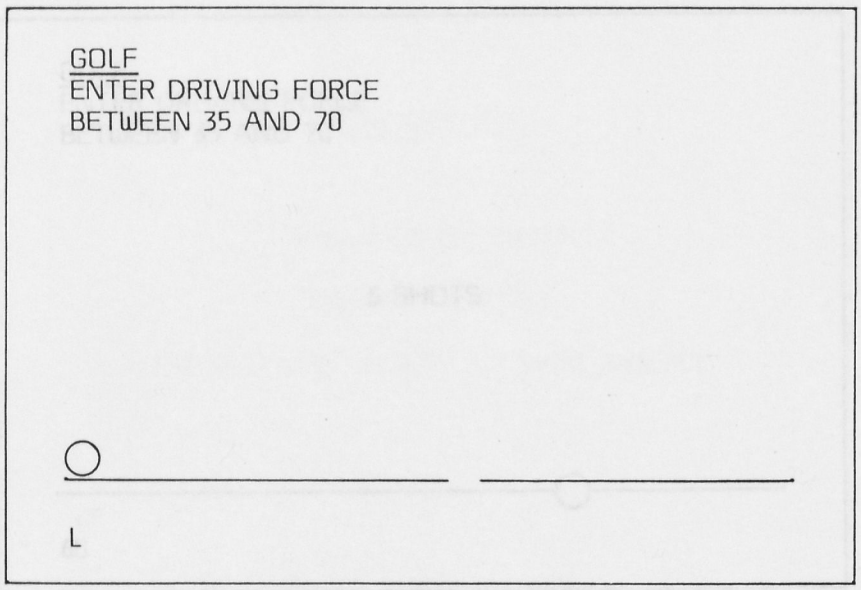

figure 2a

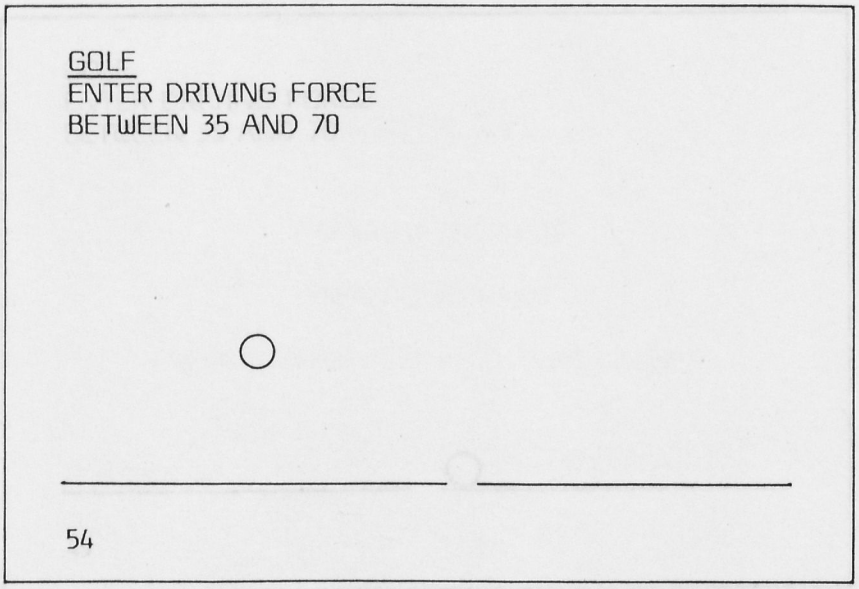

figure 2b

APPENDIX 2

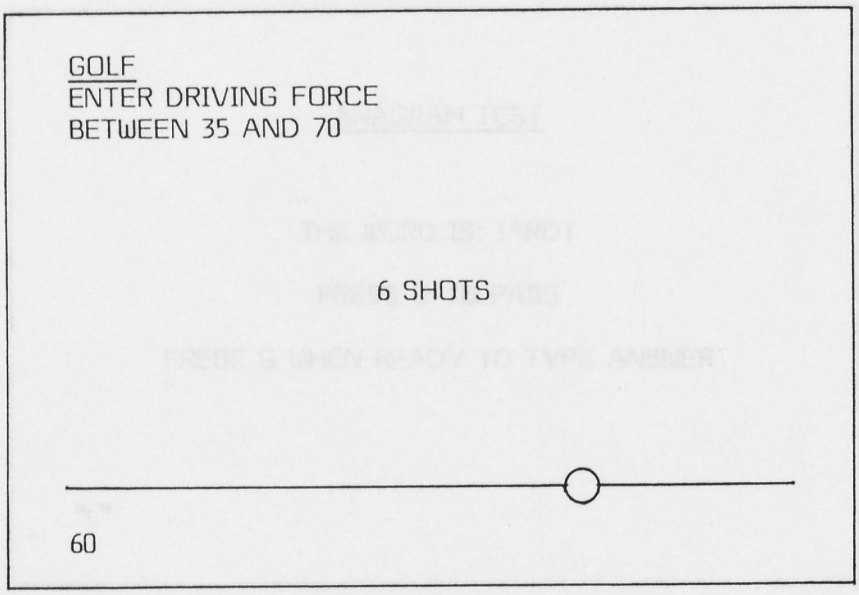

figure 2c

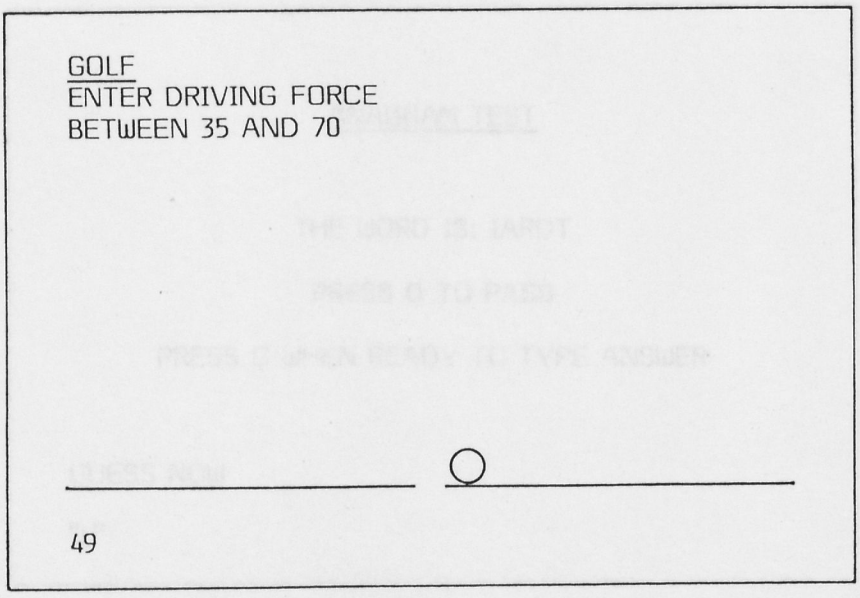

figure 2d

APPENDIX 3

ANAGRAM TEST

THE WORD IS: IARDT

PRESS O TO PASS

PRESS G WHEN READY TO TYPE ANSWER

"L"

figure 3a

ANAGRAM TEST

THE WORD IS: IARDT

PRESS O TO PASS

PRESS G WHEN READY TO TYPE ANSWER

GUESS NOW

"L"

figure 3b

APPENDIX 3

TRIAD IS CORRECT

figure 3c

XXXXX IS WRONG

figure 3d

APPENDIX 3

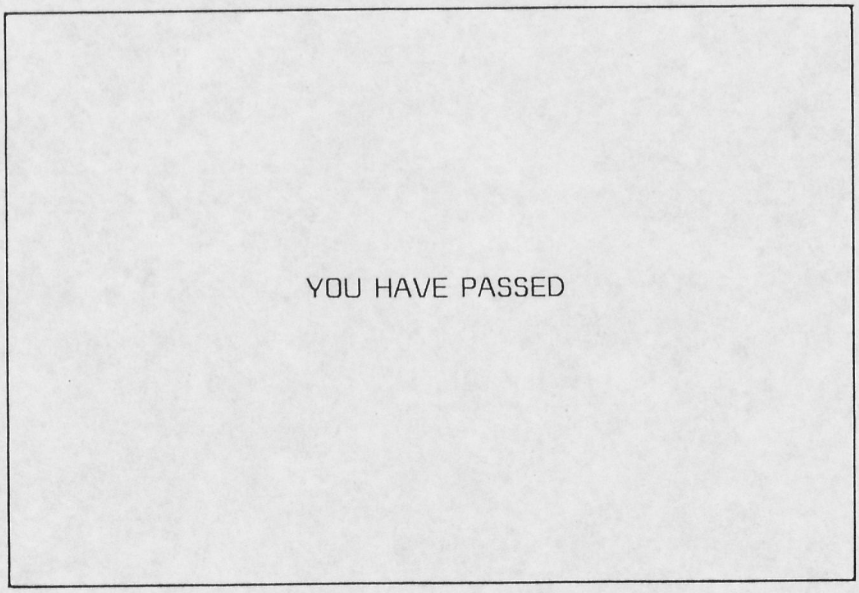

figure 3e

APPENDIX 4

FAILURE IN A TASK AS A MOOD INDUCTION PROCEDURE: A PILOT STUDY

Previous studies (Gatchel, Paulus and Maples, 1975; Miller and Seligman, 1975) have indicated that mood changes take place after the pretreatment phase in LH experiments with humans. In both studies mentioned above the MAACL was used. In the present study, however, it was thought more appropriate for the 8SQ to be used for reasons discussed in the Materials section (see chapter four, p.59-60). Since no reference to any relevant studies using the same instrument has been located, a pilot study was conducted employing three different tasks to induce a sense of failure or success in subjects whose mood was measured before and after completion of the task by means of the 8SQ.

Subjects(N)	Control	Experimental
SKEET	9	11
GOLF	6	10
ANAGRAMS	8	8

Method

Subjects were recruited by class anouncements at Sunderland Polytechnic and participation was voluntary. Allocation to groups was random. Subjects were told that they were to participate in a study concerned with the relationship between mood and performance.

Materials Mood was assessed by the 8SQ. Three taks in all were employed: two games, Skeet and Golf and a series of 20 five letter Anagrams. The Skeet game (see figure 4a) was rather simple and required no special skills from the subjects. The apparatus was housed in a box with an inclined top, measuring 15x22cm approximately. When the subject released the skeet, by pressing the Skeet Release button, the red light emitting diodes (LEDs) lit up

in sequence so as to simulate a target moving from left to right. The object of the game was to hit the target a its final potition - right hand red LED - by pressing the Fire button. When the attempt was successful the green LED on the far right would, momentarily, light up. The score indicator would light up after eight consecutive shots and display the score. The subjects had to keep a note of the score and reset the game, by pressing the reset button, until they completed ten games, i.e. 80 shots in all. A switch hidden from the subjects' view was used to select a normal game or an 'always miss' game.

The Golf, which was eventually chosen for the present study, is described in the Materials section (see chapter four, p.61-63).

Finally, 20 five letter Anagrams that had no solution (such as EVOLE, WOBAT, ICILE, LAKEY and FRENY) were used for the failure in the anagrams group. The 20 solvable anagrams and the procedure for administering them is described in the Materials section (see chapter four, p.63-64).

Procedure Upon arrival the subjects were shown the experimental room and were asked to complete Form A of the 8SQ. When Form A was completed the requirements of the task were explained and the subject was asked to proceed. In the case of Skeet the time required to complete the 80 shots was more or less the same between the success and failure groups because of the nature of the game. In the case of Golf it has been estimated in previous mock trials that 10 minutes were adequate for most subject to achieve 8 or more hits and thus give them a sense of success. In the failure condition the session was terminated by the experimenter after 10 minutes had elapsed. Finally, the time taken to complete the anagrams was deter-

mined by the subjects' speed in the success condition, and by the built in time limit in the failure condition, i.e. a maximum of 35 minutes.

When the task was over the subjects were asked to complete Form B, and after its completion they were debriefed, thanked for their participation and left.

Results

Simple t-tests (related samples, Robson, 1973) were carried out to assess the significance of the mood changes. Out of the eight different states, Guilt was significantly increased in the unsolvable Skeet group after failure ($t = 2.57 > 2.26$, $p < 0.05$), and Arousal decreased but this difference did not reach statistical significance ($t = 2.02 < 2.26$, $p > 0.05$). The Golf results showed a significantly increased Regression ($t = 2.67 > 2.26$, $p < 0.05$), and significantly decreased Arousal ($t = 3.14 > 2.26$, $p < 0.05$) in the unsolvable group. Finally, in the unsolvable Anagrams group, only Arousal was significantly decreased ($t = 3.06 > 2.36$, $p < 0.05$). No mood changes were observed in the solvable Skeet, Golf and Anagrams groups.

Discussion

The above results, despite the limited changes found, were considered a positive indication that the 8SQ is sensitive enough in detecting small, transient mood changes. The demonstration of gross mood swings was not considered imperative at this point since the evidence of mood changes in LH experiments has been equivocal. Miller and Seligman (1975), for instance, did not find any significant changes in depression scores in helpless subjects

and Cole and Coyne (1977) did not find any mood changes at all, following pretreatment with inescapable noise. In any case, the decreased Arousal scores in the unsolvable Golf and Anagrams groups were considered as indicative of reduced task involvement and therefore consistent with the LH theory.

The Golf game was chosen for being more consistent than Skeet in inducing mood change and also, since solvable anagrams would have to be used in the testing phase, the option of using solvable and unsolvable anagrams in the pretreatment phase as well was dropped in order to avoid any undue generalization effects.

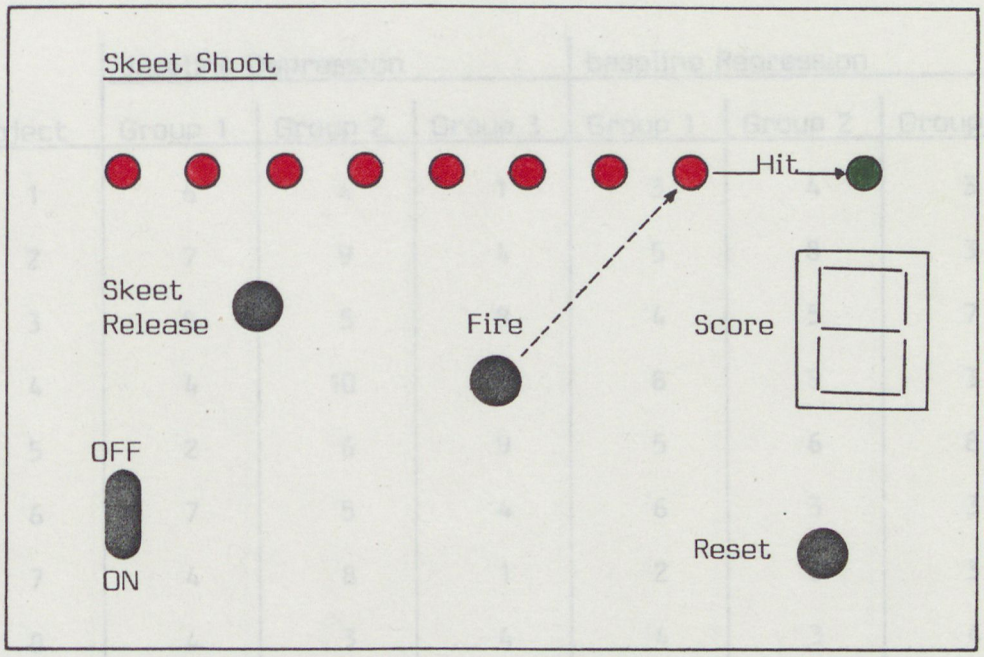

figure 4a

APPENDIX 5 Raw data

	baseline Anxiety			baseline Stress		
Subject	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3
1	6	2	3	7	5	5
2	5	8	1	6	9	1
3	5	4	6	4	6	4
4	9	7	5	9	9	2
5	5	5	10	3	4	10
6	7	6	3	9	5	1
7	1	5	4	1	3	2
8	3	5	2	5	8	1
9	6	1	7	2	1	7
10	4	2	8	1	4	6

	baseline Depression			baseline Regression		
Subject	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3
1	6	4	1	3	4	3
2	7	9	4	5	8	3
3	5	5	8	4	5	7
4	4	10	4	8	9	3
5	2	6	9	5	6	8
6	7	5	4	6	3	3
7	4	8	1	2	8	3
8	4	3	4	4	3	4
9	4	3	8	8	3	6
10	4	4	8	3	2	9

	baseline Fatigue			baseline Guilt		
Subject	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3
1	4	4	2	4	5	4
2	8	7	3	6	10	3
3	2	2	9	6	4	10
4	3	10	1	6	7	4
5	1	8	6	3	4	7
6	7	1	1	6	5	4
7	1	9	2	1	6	5
8	4	3	5	5	2	3
9	6	3	7	5	5	7
10	5	3	9	5	2	8

	baseline Extraversion			baseline Arousal		
Subject	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3
1	5	6	9	6	7	9
2	3	2	8	6	4	7
3	8	7	1	9	7	3
4	9	2	6	8	3	7
5	8	5	7	8	4	6
6	4	7	6	4	8	7
7	7	1	8	6	1	7
8	7	10	5	6	9	6
9	4	8	4	6	7	6
10	5	5	4	8	8	2

Subject	baseline SCL (µmhos)			baseline logSCL (µmhos)		
	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3
1	90.9	32.3	10.4	1.959	1.509	1.017
2	6.8	18.5	20.0	0.833	1.267	1.301
3	32.3	20.0	9.0	1.059	1.301	0.954
4	35.7	6.8	22.2	1.553	0.833	1.346
5	12.3	100.0	47.6	1.090	2.000	1.678
6	23.8	26.3	19.6	1.377	1.420	1.292
7	45.5	14.2	22.2	1.568	1.152	1.346
8	25.6	25.6	1.8	1.408	1.408	0.255
9	18.7	22.7	26.3	1.272	1.356	1.420
10	16.0	35.7	19.0	1.204	1.553	1.279

Subject	baseline Heart Rate (bpm)		
	Group 1	Group 2	Group 3
1	88	53	70
2	66	84	66
3	63	63	76
4	71	80	76
5	68	64	70
6	53	70	54
7	84	62	70
8	75	80	82
9	71	56	82
10	79	80	57

APPENDIX 5

Experimental Data

Subject	Anxiety					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	5	5	1	4	5	3
2	7	7	10	10	3	3
3	3	4	5	4	3	1
4	8	6	3	4	1	3
5	2	2	3	4	5	7
6	5	3	8	6	5	5
7	1	4	7	7	4	6
8	7	8	1	1	1	3
9	8	8	1	1	5	2
10	3	3	3	3	3	4

Subject	Stress					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	7	6	4	4	5	5
2	7	8	10	10	3	3
3	1	3	5	5	5	4
4	6	4	5	5	1	1
5	3	5	3	1	6	8
6	5	4	7	5	2	4
7	1	1	4	6	6	2
8	9	7	2	3	2	5
9	6	7	1	1	9	6
10	2	1	1	3	2	4

Experimental Data

Subject	Depression					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	5	3	6	6	7	3
2	7	10	10	10	3	3
3	3	4	5	3	4	8
4	4	5	4	3	1	4
5	3	2	5	5	8	6
6	3	5	7	5	7	6
7	5	4	10	6	7	3
8	10	8	3	3	3	3
9	8	6	2	1	8	4
10	4	4	3	3	4	5

Subject	Regression					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	5	7	4	6	6	5
2	6	9	9	10	3	4
3	4	3	6	4	3	4
4	8	5	4	6	4	3
5	3	3	6	5	8	6
6	3	6	6	4	5	5
7	3	1	10	10	3	2
8	7	8	2	2	4	5
9	8	9	3	1	4	6
10	4	5	3	1	3	7

Experimental Data

Subject	Fatigue					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	3	6	7	5	8	7
2	8	6	9	9	3	3
3	2	3	1	5	3	3
4	6	3	6	6	2	1
5	3	2	3	5	9	9
6	6	5	6	5	6	6
7	2	2	10	10	5	8
8	7	8	3	3	5	4
9	6	6	3	2	7	4
10	4	5	4	3	4	5

Subject	Guilt					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	5	4	5	4	5	5
2	8	10	10	10	5	3
3	4	3	4	4	8	6
4	7	5	4	6	1	2
5	2	3	2	5	7	10
6	5	4	5	4	5	6
7	1	4	7	9	3	5
8	8	8	1	3	4	5
9	8	8	1	1	7	5
10	4	6	1	2	4	4

Experimental Data

Subject	Extraversion					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	6	7	4	6	3	5
2	4	2	1	1	8	7
3	9	8	9	7	5	5
4	8	6	4	3	9	8
5	9	7	6	6	2	4
6	8	7	4	8	4	1
7	3	8	2	1	4	8
8	1	4	8	8	5	5
9	2	3	9	8	5	6
10	6	5	7	6	8	8

Subject	Arousal					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	4	5	4	5	3	3
2	6	3	5	2	5	6
3	7	10	8	7	6	8
4	6	5	7	5	9	7
5	8	9	7	7	3	1
6	7	7	4	7	6	5
7	6	8	1	2	6	6
8	3	2	8	8	6	5
9	5	4	8	10	3	5
10	6	6	8	9	5	6

Experimental Data

Subject	Skin Conductance Level (µmhos)					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	35.9	46.1	9.7	11.0	2.7	5.7
2	12.7	9.7	13.9	17.2	21.3	21.3
3	28.0	30.7	4.2	7.1	4.9	8.1
4	19.8	14.7	11.6	11.5	5.2	5.2
5	1.9	2.2	9.4	4.9	22.1	29.1
6	15.9	12.5	19.8	21.4	2.3	7.3
7	31.3	24.2	4.6	6.6	9.0	21.6
8	15.9	3.0	22.6	11.5	2.0	3.4
9	7.0	12.2	21.8	19.7	13.6	19.4
10	10.3	16.5	25.5	25.0	9.8	9.8

Subject	LogSkin Conductance Level (µmhos)					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	1.555	1.664	0.897	1.041	0.431	0.756
2	1.104	0.897	1.143	1.236	1.328	1.328
3	1.447	1.487	0.623	0.851	0.690	0.908
4	1.297	1.167	1.064	1.061	0.716	0.716
5	0.279	0.342	0.973	0.690	1.344	1.464
6	1.201	1.097	1.297	1.330	0.362	0.863
7	1.496	1.384	0.663	0.820	0.954	1.334
8	1.201	0.477	1.354	1.061	0.301	0.531
9	0.845	1.086	1.338	1.294	1.134	1.288
10	1.013	1.217	1.407	1.398	0.991	0.991

Experimental Data

Subject	Spontaneous Skin Conductance Responses					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	0	0	5	0	21	26
2	4	0	2	10	48	33
3	4	0	0	5	2	0
4	0	3	32	27	0	0
5	0	2	0	2	0	0
6	23	18	0	0	1	12
7	0	0	7	2	0	14
8	7	3	1	0	0	0
9	0	1	0	0	0	1
10	2	6	0	1	10	16

Subject	Spontaneous Skin Conductance Responses - log(X+1)					
	Group 1		Group 2		Group 3	
	before	after	before	after	before	after
1	0.00	0.00	0.78	0.00	1.34	1.43
2	0.70	0.00	0.48	1.04	1.69	1.53
3	0.70	0.00	0.00	0.78	0.48	0.00
4	0.00	0.60	1.52	1.45	0.00	0.00
5	0.00	0.48	0.00	0.48	0.00	0.00
6	1.38	1.28	0.00	0.00	0.30	1.11
7	0.00	0.00	0.90	0.48	0.00	1.18
8	0.90	0.60	0.30	0.00	0.00	0.00
9	0.00	0.30	0.00	0.00	0.00	0.30
10	0.48	0.85	0.00	0.30	1.04	1.23

APPENDIX 5

Experimental Data

Subject	Heart Rate (bpm) Group 1 before	Group 1 after	Group 2 before	Group 2 after	Group 3 before	Group 3 after
1	76	73	77	70	60	55
2	63	66	89	87	67	64
3	66	61	58	52	70	67
4	64	60	71	64	73	73
5	71	73	62	58	64	61
6	57	55	63	67	60	57
7	87	87	60	56	64	68
8	78	77	77	74	77	73
9	71	66	65	59	82	81
10	73	68	71	67	60	52

Subject	Number of Anagrams Solved Group 1	Group 2	Group 3	Mean Time per Anagram Solved* Group 1	Group 2	Group 3
1	8	18	20	32.43	17.47	20.32
2	17	20	6	35.44	15.00	51.40
3	20	19	18	13.37	13.11	19.35
4	11	19	20	50.00	14.17	9.05
5	19	19	17	13.17	11.78	15.88
6	20	14	8	15.47	32.54	61.00
7	11	10	20	33.3	40.78	21.63
8	20	19	6	21.79	11.44	41.00
9	19	18	5	17.22	31.94	30.00
10	20	18	10	13.53	18.65	20.22

* (seconds)

Experimental Data

Subject	Number of Anagrams Passed			Time Out		
	Group 1	Group 2	Group 3	Group 1	Group 2	Group 3
1	11	1	0	1	1	0
2	1	0	1	2	0	13
3	0	0	1	0	1	1
4	1	0	0	8	1	0
5	0	0	0	1	1	3
6	0	2	0	0	4	12
7	6	5	0	3	5	0
8	0	0	3	0	1	11
9	0	1	4	1	1	11
10	0	0	1	0	2	9

Subject	Anagram Performance Index*		
	Group 1	Group 2	Group 3
1	0.180	0.924	0.984
2	0.442	1.333	0.092
3	1.496	1.347	0.843
4	0.186	1.252	2.210
5	1.341	1.487	0.900
6	1.293	0.363	0.110
7	0.260	0.197	0.925
8	0.918	1.527	0.109
9	1.043	0.530	0.111
10	1.478	0.872	0.310

* Anagram Performance Index= Number of Anagrams Solved/ Mean Time per Anagram Solved + Number of Anagrams Passed + Time Out

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"Modern Science. The scientific revolution associated with the name of Galileo freed thinking of superstition and academic tradition and won attention to the observation of nature. But it failed to modify and extend its method to social and moral matters, and indeed science has gotten further and further from ordinary experience. With the dominance of science and applied science in our times, the result has been a specialist class of scientists and technicians, the increasing ineptitude of the average person, a disastrous dichotomy of "neutral" facts versus "arbitary" values, and a superstition of scientism that has put people out of touch with nature, and also has aroused a growing hostility to science."

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