AN INVESTIGATION OF COGNITIVE FACTORS IN DEPRESSION

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I declare that I am the author of this work, that unless otherwise stated all work was carried out by myself and that this work is original and has not been submitted in part or in full for any other degree.

Paul R. Gilbert

SUMMARY

This thesis set out to investigate cognitive variables in depression. Introductory chapters examine some of the central philosophical and empirical questions raised by psychological theories of depression. The three studies conducted investigate some of the questions still to be resolved.

The first study involves 72 subjects and investigates whether depressed patients differ from a psychiatric (anxious) control group and a non-psychiatric control group on the parameters of (a) perceptual and attitudinal sensitivity to positive and negative events, (b) questionnaire measures of certain cognitive and attitudinal variables. These measures were re-investigated in the depressed patients on their recovery.

The second study considers the question of vulnerability to low mood and analyses data from 150 fear of failure questionnaire non-psychiatric respondents. High scorers (n = 18) and low scorers (n = 16) were investigated on the same parameters as subjects in study one.

The third study examines more critically the discrimination of recovered depressed from improved anxious patients on various cognitive measures. Final subject selection was n = 15 for each group, drawn from an original patient sample pool of n > 100.

The results suggest that depressed subjects may be under-attentive to positive aspects in their environment rather than hypersensitive to negative events compared to control groups, but are not different from anxious patients on questionnaire measures of attitudes. The second finding appears true in both

the ill (study one) and recovered (study one and study three) conditions. There is also evidence that high fear of failure subjects may be more vulnerable to low mood than high hope of success (low fear of failure) subjects.

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CHAPTER ONE

INTRODUCTION

I. THE PROBLEM OF DEPRESSION

Depression has been referred to as the common cold of psychopathology. However, although this comparison conveys an idea of the frequency at which psychiatry encounters depressive illness, it fails to reflect the often serious and tragic nature of this disorder. This tragedy involves not only the patient himself but also the family who have to attempt to cope with a depressed member, especially when the depression is recurrent and/or severe.

For some individuals depressive illness ends fatally. It has been estimated (Stengel, 1970) that the number of people attempting suicide in a large metropolitan city such as London, with a population of eight million, is between 7,500 and 12,000 per annum. For England and Wales the figure is between 30,000 and 40,000 per annum. Many suicidal attempts are not successful and many attempts are not the result of a depressive illness. Nevertheless it is generally recognised that the most vulnerable individuals to death by suicide are those suffering from depression.

Some idea of the size of the problem that depression presents to psychiatry can be gained from examining recent hospital figures. In 1977 the total number of admissions to the Royal Edinburgh Hospital was 2,381, of which 581 or 24.4% were given a diagnosis of depression. The admission figures for 1975 published by the Scottish Home and Health Department (1978) show that of the 2,363 admissions to the Royal Edinburgh Hospital, 30% of the 1,318 women admitted and 12% of the

of the 1045 men admitted were diagnosed as suffering from a depressive illness. It is also interesting to note that for women depression was the most common single diagnosis, but for men the single most common diagnosis, 33% was alcoholism and alcoholic psychosis. However, it has been suggested that alcohol abuse may be an attempt by some individuals to control depression and anxiety (Goss and Morasko, 1970). Thus the excessive use of alcohol may mask a depressive illness in some individuals, in particular in males.

This problem requires further consideration, but is not the issue of this thesis. Nevertheless, the problem of distinguishing between psychiatric disorders has presented one of the most serious and troublesome areas for psychiatry. Not only the differentiation between disorders, but also the sub-divisions within particular disorders, especially depression, have been the source of many disputes. For example of the 581 patients admitted to the Royal Edinburgh Hospital in 1977, the following sub-classifications were made (Table 1.1). These classifications were based on the criteria laid down by 'A glossary of mental disorders' (1968), which is based on 'The International Statistical Classification of Diseases' (1965).

Table 1.1 Sub-classifications of patients admitted to the Royal Edinburgh Hospital (1977) suffering from depression

Sub-classification	Glossary classification number	Number admitted
Involutional melancholia	296.0	35
Manic-depressive psychosis, depressed type (includes endogenous depression)	296.2	334
Reactive depressive psychosis	298.0	39
Depressive neurosis	300.4	173
	Total	581

In addition to these classifications there is the problem of depression following childbirth as discussed in the Glossary. There is also a separate classification for personality disorders which contains the sub-classification of affective (cyclothmic) that includes individuals with persistent anomalies of mood - either a gloomy depressed attitude to life, or the opposite, namely a perpetual satisfaction with life, etc. Thus, as mentioned in the Glossary, the problem of classification is a large one and the classifications recommended are considered to be primarily for the aid of international comparisons and are not meant to "impose theoretical concepts on the user". In fact, theoretically there are many different nosological systems available (Kendell, 1976).

In the preface to his book 'The Role of Diagnosis in Psychiatry', Kendell (1975) argued that it is the failure to develop a satisfactory classificatory system that has posed the greatest stumbling block to fruitful research on the question of aetiology in psychiatric disorders. Eysenck (1970) has also pointed out that before aetiological questions can be answered, there must be some idea of what the disorder is, and how it can be recognised and differentiated from other disorders. Although some progress has been made on this issue, there are still many disputes and problems to be overcome.

II. THE APPROACH AND SCOPE OF THIS STUDY

Some of the reasons for the failure to develop an acceptable classificatory system lie in the historical and philosophical origins of psychiatry. Moreover many of the philosophical assumptions which are often at the heart of present day disputes rest on suspect philosophical analysis. Since, as Kendell (op. cit.)

points out, these issues have constituted a major obstacle to progress in aetiological research, especially in depression, they require some discussion and are thus examined in chapter two.

In the last twenty-five years, however, attention has turned to the mechanisms of depression, especially the biological mechanisms. This development has recently been combined with an essentially psychobiological approach to depression which has attempted to point out the multifarious nature of depression (e.g. Akiskal and McKinney, 1975). One of the major changes of emphasis that this sort of multicomponent approach has helped to bring about is to challenge the view that depressive illness is simply a disorder of mood which can be best understood and treated by biological alterations in the mood regulating areas of the brain. The multicomponent approach focusses more on the interactive nature of psychological and biological events and argues that it is in a better understanding of these interactions that depression will be better advanced at this stage, rather than in a more detailed analysis of individual systems, though such remain important.

In addition to this change in the philosophical assumptions of what depression actually is, other workers have pointed out the paradoxical dichotomy that appears to exist between explanations of normal mood and explanations of pathological mood. Since normal mood appears to change to a considerable degree with the psychological responses of the individual, it has been argued that pathological mood disturbances are amenable to the same explanatory framework.

To a large extent, the multicomponent approach to depression and the development of a psychological framework present exciting possibilities for the future, especially in efforts to combine them. The old assumptions that it is always a pathological mood

disturbance which produces the pathologically disturbed view of the world and the other negative and behavioural symptoms of depression now seem very untenable, though it still haunts many views of depression. These old assumptions about depression, what it is and how it is caused, have directed research in the past and indeed still do, but they should be looked at afresh and seen against the background of philosophical beliefs and assumptions which incorrectly argued in favour of the separateness of psychological and physiological processes, and the separateness of individual aspects of a person's psychological functioning.

These issues are examined in the introductory chapters and special emphasis in chapter three is given to the development of a multicomponent model of depression which pays particular attention to coping processes. Indeed, it is from the point of view of the determinants of coping behaviour that the psychological approach to depression is introduced. It is argued that the psychological factors which shape the appraisal of stress and the availability of coping options, can, through their effects on adaptive coping behaviour, influence biological response patterns to perceived stress. Having outlined this relationship, the last introductory chapter looks more closely at the purely psychological factors that have been implicated in depression. From the point of view that certain psychological factors can act as internal constraints on adaptive coping behaviour, these psychological factors may be seen as operating to increase the individual's vulnerability to depression.

This thesis examines the extent to which the psychological factors outlined by various theories of depression are prevalent in depressed and depression-prone individuals and the extent to which they are specific to depression. Moreover, a normal population is screened to test for vulnerability characteristics.

CHAPTER TWO

HISTORICAL AND PHILOSOPHICAL PROBLEMS IN THE STUDY OF DEPRESSIVE ILLNESS

Depressive illness was first recognised and observed over two and a half thousand years ago. In the fourth century B.C. Hippocrates labelled it melancholia. Hippocrates was a keen clinical observer and on the basis of observed symptomatology classified mental illness in terms of a number of disorders including epilepsy, mania, melancholia and paranoia (paranoia referred to a form of mental deterioration). He also recognised hysteria and phobic states (Zilboorg and Henry, 1941). Since Hippocrates, descriptions of depression (melancholia) have undergone very little change. Beck (1973) points out, for example, that Plutarch in the second century A.D. presented a clinical description of melancholia which would be easily recognisable today as depression. Yet in spite of this long history depressive illness presents psychiatry with one of its most confusing and controversial problems. There is no other single illness which has such a long history and yet which presents so many unresolved questions. Paradoxically the lack of common agreement concerning this condition cannot be attributed to a lack of effort or interest. Indeed, depressive illness has often been at the centre of some of the most heated and fiercely debated disputes in psychiatry. Rather it appears that many of these arguments reflect as much a basic and fundamental division in the philosophical concept of disease and its treatment, as a disagreement over scientific evidence.

I. THE ORIGINS OF MODERN PHILOSOPHICAL CONTROVERSIES

Schneck (1960) points out that Hippocrates was primarily concerned with the treatment and prognosis of illness rather than diagnosis. This orientation to medicine was in part due to the fact that Hippocrates was a keen physician and in Greek society the reputation of such a man depended upon the 'goodwill of his patients'. But Hippocrates's approach to medicine also reflected a basic philosophy. Hippocrates believed that the acquisition of knowledge depended upon careful observation made about individual events and objects. Such a philosophy emphasises the importance of observed variations in individual events rather than generalisations concerning specific classifications. Such an approach to medicine has sometimes been referred to as the quantitative approach, where illness is viewed as a quantitative observable deviation from normal.

Plato (427-347 B.C.), at one time a student of Hippocrates, rejected Hippocrates's philosophy. Although Plato thought highly of Hippocrates he opposed the notion that observation is the best method for the acquisition of knowledge. Observation, Plato argued, depends upon the use of the senses, yet the senses are easily misled and deceived. In many ways Plato followed Pythagoras (570-489 B.C.) and was more concerned with mathematics and what is often referred to as a 'doctrine of universals' (Kendell, 1975). Plato's influence on medicine which was to be considerable, was indirect. Plato was not regarded as a noted physician in the same way as Hippocrates. As a result, his approach to medicine did not depend upon the 'goodwill of his patients'. Consequently his orientation to medicine was determined

very much by his philosophical approach to events in general. In line with Plato's belief in a doctrine of universals, his influence on medicine was to draw attention to diagnosis of diseases, in which diseases were seen as discrete entities. This concept of disease argues that diseases can be viewed as qualitative deviations from normal, such that each disease has its own recognisable characteristics. In this approach the individual person is of little interest, instead attention is focussed on the individual's disease.

Kendell (1975) has pointed out that the influence of these two alternative (though not totally opposing) philosophical approaches to illness are reflected in the investigation of diseases in two early medical schools. On the one hand the school at Cos, following an essentially Hippocratic philosophy, meticulously studied the varied manifestations and natural history of diseases in the individual patient. Its rival Platonic school at Cnidus was more interested in the diagnosis and classification of individual diseases. It is reported that this school was able to recognise and classify a number of diseases of the bladder and the bile (Kendell, 1975).

Over the two and a half thousand years following Plato and Hippocrates, as is often the case with such long disputes, these two philosophical disputes became polarised, so that as Kendell (1975) observes:

"The essence of the dispute between the Platonic and Hippocratic traditions in psychiatry, as in medicine as a whole, has always centred around the concept of disease entities, the former maintaining that such things exist, and bent on identifying them, the latter regarding them as man-made

abstractions, at best an irrelevance, at worst a dangerous source of misconceptions distracting us from reality. The true relationship between these two contrasting attitudes, and the justification for each has often been obscured by the smoke of combat." (P. 63).

But the conclusion from a number of sources (Zilboorg and Henry, 1941; Ackerknecht, 1959; Schneck, 1960; Kendell, 1975) appears to point out that the present dichotomy and confusion governing these alternative approaches is the product of history rather than the original philosophies. As Kendell argues, the need for diagnosis remains in that it "serves the important negative function of excluding from consideration many other types of problem". Further, the need for diagnosis becomes greater as different methods of treatment are shown to be differentially effective with different types of patient. Such a position does not necessarily imply, however, that Plato's concept of illness, in terms of a qualitative change, is the only way around this problem. Hippocrates more ably recognised and distinguished between various manifestations of mental illness than Plato, yet stressed and maintained a philosophical approach based on the study of individual manifestations of illness. But it may be useful, as Kendell believes, to retain the importance of diagnostic procedures which have some basis in a classified system of illness, whilst not necessarily accepting the concept of diseases as distinct and separable entities.

II. SPECIAL PROBLEMS OF MENTAL ILLNESS

Hippocrates was a forceful advocate of the view that mental illness should be placed within the boundaries of medicine. He fiercely opposed attempts to retain

mental illness in the realms of theology and philosophy which was current practice at the time. His attempts to achieve a medical explanation for such illnesses are highlighted in his work on epilepsy, which, until Hippocrates, was considered an illness of the gods. Hippocrates is reported to have written:

> ". . . if you cut open the head you will find the brain humid, full of sweat and smelling badly. And in this way you may see that it is not a god which injures the body but a disease . . ." (Zilboorg and Henry, 1941, P. 44).

Not only was Hippocrates a firm believer in mental illness being considered as a medical problem, but it also seems that his view of mental illness was extremely flexible and enlightened even by modern standards. Zilboorg and Henry (op. cit.) claim that:

> ". . . Hippocrates, depending upon the case and occasion, favoured a purely anatomical view represented even in the twentieth century, which claims that a disease or injury to the brain is the sole cause of mental illness. Yet he also believed in the physiological theory, in our days represented by psychiatric endocrinology, which claims that certain body juices are responsible for madness. Finally, he was occasionally of the opinion that purely mental, emotional states may produce deep changes, even physiological changes, in an individual; " (P. 46).

The concept of multi-causality favoured (at least occasionally) by Hippocrates was only possible via his approach and concern with the individual and with the concept of illness as a quantitative deviation from normal. It is doubtful whether a purely (Platonic) qualitative approach lends itself to similar

flexibility. It would certainly seem on the surface that the multi-causal concept of illness does not fit with the concept of there being disease entities; since, as entities, each disease is considered to have its own (idiosyncratic) aetiology, course and outcome. As will be discussed later, the adoption of the diagnostic approach, especially through Kraepelin (based on a Platonic orientation to medicine) has seriously handicapped multi-causal theories of mental illness. Had Hippocrates's influence been more enduring and not opposed by Plato and subsequent religious dogma and superstition, it is possible that present day conceptualisations of mental illness would be more flexible. As it is, it was only in 1975 that Akiskal and McKinney published their important paper on the integration of ten models of depression and attempted to show that depressive illness could be viewed as a multi-component phenomenon.

Plato, disagreeing with Hippocrates, argued that there are two kinds of mental illness, one the result of disease, the other a gift of the gods. This retrograde step had a devastating effect on subsequent attempts to understand mental illness. In effect Plato contributed in returning mental illness back into the realms of theology and philosophy. Thus as Zilboorg and Henry (1941) argued:

> "As a result, the great effort made by Hippocrates seemed not a little in vain: by reintroducing the concept of divine, revelatory madness, Plato reasserted the "low" nature of ordinary madness - a conception which Hippocrates opposed as much as the conception of sacred madness. The medical point of view was thus considerably weakened by Plato; this is particularly true in view of the fact that Platonic thought exercised an enormous influence for several centuries, including the early part of the Christian era" (P. 53).

Plato's belief in a condition of sacred madness placed the mind and soul in a complex interrelationship. With the decline of the Greek and Roman era of medicine and the spread of organised, dogmatised religion, Plato's views of mentality became firmly incorporated within early Christian teachings. By its monopoly of what little education there was, the church shackled the development of medicine with its emphasis on endless interpretation of classical ideas. Genuine debate and the expansion of knowledge by further observation came to an end. The dogma of the early Christian church brought the further development of science in any form to a slow and hesitant crawl.

The problem of the mentally ill became the jurisdiction of the church, and as such soon became the subject of demonology. Although Plato's philosophical view of mental illness certainly contributed to this development, the demenological relationship between mind, soul, madness and demons can be found in many places in the Bible. It was common for healers, including Jesus Christ for example, to heal the mentally ill by the casting out of devils. It is however surprising how long this belief lingered on. Even today in some religious quarters, exorcism is used as a way of attempting to cure the mentally ill. So entrenched did the demonological approach become that it was not until 1567 when Johann Weyer (1515-1588) published a detailed treatise advocating that witches be treated as mentally ill, was the demonological view seriously challenged. But even as famous and as respected as Weyer was in his day, the demonological view lingered on. For example, Daniel Sennett (1572-1637), a noted clinician of his day, believed that cases of ecstatic mental illness were caused by the devil. Van Helmont (1577-1644), another highly respected clinician of his day, considered that disease

(especially mental illness) was brought by the devil as payment for sin. Thomas Willis (1621-1675), most noted of all, especially as a physician and neuroanatomist, believed in devils and in the harsh treatment of the mentally ill. He argued that it was only by torture and torment that the disorderly minds of the possessed and mad could be brought into a meek and ordered state. But the Renaissance slowly saw the barriers of fear and superstition broken. Two men especially stand out in the return to open scientific research in the study of man. The first of these was Leonardo da Vinci. Leonardo restated a typical Hippocratic orientation to science by declaring that "Science comes by observation not by authority" (Blakemore, 1976). His dissection of human corpses for the purpose of anatomical observation and drawing led him into direct conflict with the church and eventually he was forced to leave Rome. Nevertheless his drawings and approach to the study of the body had a considerable impact on the medical world and were used for some considerable time (e.g., especially the pictures of the baby in the mother's womb). Leonardo's example of observation by dissection was slowly copied. But there was an area of the body which the church remained adamant about in its condemnation of its dissection. This was the brain.

III. MIND BODY DUALISM

Plato placed the seat of the mind in the head (unlike Aristotle who placed it in the heart). But the problem of a relationship between mind and soul meant that to dissect the head (thus the brain) was to cause an interference with the soul. For some time there seemed little way round this problem, until Descartes offered a solution. Descartes proposed that the functions of the mind and

mechanisms of the body operate independently of each other. It was the existence of the mind that separated man from animals. Such being the case, the brain (as part of the body) could be dissected and investigated without fear of interfering with the soul and the order of God. As will be discussed later, this view of man has caused considerable problems to psychiatry and psychology, not least because the definition of the Cartesian mind remains ambiguous. Nevertheless, the Cartesian solution to the religious scientific conflict allowed for the physical investigation of the brain to begin in detail. As Blakemore (1976) points out:

> "Descartes was to make explicit the principle of dualism, that the bodily functions of the brain are entirely distinct from the mystical domain of the soul . . . it freed men, even devout men, to speculate about the working substance of the brain, without fear of treading in the footprints of God. It made possible the existence of a sincere belief in a rational soul and a materialistic attitude towards the mundane parts of the mind" (P. 597).

To be able to appreciate the impact and influence dualism was to have on subsequent medical thought, account should be taken of the social, religious and political background from which it grew. Descartes was not the sole innovator of a dualistic view of man, the principle of dualism existed in Hippocrates's era though in an unspecified form, and Plato apparently believed that the soul was temporarily imprisoned in the body from which death was the only release. However it was Descartes who made explicit the principles of dualism.

Descartes was a deeply religious man and also fearful of the church. Yet he also had a strong desire to forward knowledge, especially knowledge concerning man himself. It is possible that partially because of his religious beliefs, Descartes refused to accept a totally mechanistic view of man. To Descartes, dualism offerred a logical solution to the religious scientific conflict. As Blakemore has pointed out, Cartesian dualism offered an almost ideal solution which allowed even the most devout Christian to dissect and investigate the brain without fear of damnation. Thus dualism quickly established itself through the intellectual necessity to circumnavigate the conflict with religion and the renewed desire for objective knowledge of man himself, so much a mark of the Renaissance. But dualism remains now as then a position of compromise, the result of an emotional cultural conflict, rather than objective argument.

For all its practical value in the development of seventeenth century philosophy and medicine, dualism presents many philosophical difficulties to man's conception of himself. A major problem stems from the semantic interpretation of the concept of the mind. Descartes himself was not particularly clear as to what he meant by the term 'mind'. It is argued (Oatley, 1972) that Descartes' view of the mind represented a concept of the (religious/spiritual) soul allied with the faculty of reasoning. The inclusion of the faculty of reasoning in the concept of the mind is most probably due to the fact that Descartes regarded the ability of reasoning as a characteristic of man, not possessed by animals. He believed animals were completely explainable within a mechanistic approach which, in fact, was very similar in its concepts to later theories of reflexology. Consequently, Descartes is sometimes described as the father of reflexology.

Descartes' mechanistic theories of behaviour were influenced by the water gardens, popular in France at the time. A visitor walking through these gardens could step on a tile arranged to operate a series of valves. Opening of the valves caused water to run from a main reservoir along pipes and activate life size statues. What these statues actually did depended on the whims of the engineer who made them (Oatley, 1972). Descartes believed that external, environmental stimuli had a similar effect on the mechanisms of body as the visitor stepping on the tile in the water garden. Thus, as Oatley (op. cit.) points out, Descartes believed that:

> ". . . the sense organs were operated by stimuli in the environment (just as were the tiles in the garden paths) and an external event acting upon a sense organ pulled tiny strings which ran within the nerves up to the brain. In the brain, the heart had pumped up a head of fluid which was stored in reservoirs (the ventricles). The strings opened valves to let this fluid run down the appropriate motor nerves (which were thought of as tubes) to inflate whatever muscles were appropriate for responding to the stimulus that had occurred." (P. 42)

Descartes believed that in humans, though not in animals, the reasoning soul or intellect resided near the main reservoir, rather like the master engineer in the water gardens. In this way the soul kept an eye on things and occasionally opened a few valves of its own. Thus Descartes' "mind" (the reasoning and spiritual soul) was totally distinct from the workings of the body, in the same way as the engineer was totally distinct from the workings of his water gardens. Because of the mystical and divine qualities he invested in the mind and his strong religious beliefs, Descartes avoided detailed explanations of the mind. These he

left to the church. As a result the concept of the mind remained extremely vague.

Clearly, dualism could not remain in this ambiguous position indefinitely. The discovery of electrical nerve impulses and biochemical changes replaced Descartes' conception of valves and reservoirs. But the basic principle of behaviour as a system of reflexes not only remained but was to be considerably developed in the late nineteenth and early twentieth centuries by the behaviourists. Thus the bodily operations described by Descartes in the execution of behaviour were easily adaptable to modern discoveries. The concept of the mind, however, with its imprecise and partly religious definition, presented many more problems. As philosophy and early psychology began considering psychological events in man, it was more acceptable, especially in philosophy, to attribute these events to the mind rather than to biological mechanisms. Further, with the decline of the church's power over the development of science and in particular medicine, the need to retain the concept of a mystical soul as a main component of the mind steadily declined from being an undefined semi-religious concept, the mind became a descriptive term for psychological factors that were considered not translatable into biological terms. Many psychological events such as reasoning, conceptualising, thinking, experiencing, feeling, etc., along with such concepts as 'consciousness', became encompassed within the concept of the mind. Modern psychological arguments reflect this change to a mentalistic approach. For example, some modern concepts of the mind include the notion of complex information processing operations. Mandler's (1975) view of the mind is a typical example. Mandler

argues that the mind is a theoretical system composed of a number of mental events. He argues:

> "I wish to imply no more and no less than the theoretical system that structures readiness, attention and search for environmental inputs, transforms these inputs as a result of complex processing mechanisms that include self-correcting and self-instructing systems, and then structures its output to the environment in line with its interpretation of environmental and systematic requirements. It is in this sense that I wish to use the word "mental" and to call the theoretical system ascribed to the organism the Mind" (P. 3).

Quite absent from this conceptualisation of the mind is any reference to a mystical soul, or any religious concept. Here the mind is defined as a reference term for an assortment of mental events. Thus the concept of the mind has developed in two important ways since Descartes. First attempts have been made (e.g. Mandler, op. cit.) to give a precise and tangible definition of 'the mind'. Secondly the mind has lost its metaphysical qualities such as the mystical soul.

The mind body problem is still an immensely complex and often heated area of discourse. Although the religious components of the mind have been dropped in modern definitions, there remains a strong tendency to conceive of 'the mind' as more than a biological machine analysable in terms of neuronal functioning. This tendency is often expressed as a problem of irreducibility. In other words it is argued that the complexities of human experience cannot be reduced to biological mechanisms. It is difficult to estimate the extent to which this view reflects (as it did for Descartes) a noble and ego-centric view of man. Most

recently attempts have been made to offer 'higher order' explanations of the mind, that is explanations that go beyond both psychological and physical explanations and utilise theories from other disciplines, notably physics. Thus explanations of the mind have been offerred in terms of the hologram (see Brown, 1975) and quantum mechanics (see Watson, 1973). Whatever the outcome of future discussions concerning the mind body problem, the fact remains that it is an entrenched concept within psychiatric thinking and as such presents many problems for theories of mental illness.

IV. THE PROBLEM OF DUALISM FOR THE CONCEPTUALISATION OF MENTAL ILLNESS

One implication of the dualistic philosophy in psychiatry is to lend weight to the notion that psychological and physical events can enjoy relative freedom of each other, or to put the same position in a slightly different context, that psychological causes and physical causes of illness can operate independently. Further, that these different causes can give rise to different types of illness. There is little doubt that such an assumption is at the heart of the neurotic-psychotic distinction (Hill, 1968). The problem raised by such a distinction is that if these two types of illness are shown to lie on a continuum, what happens to the dualistic principle as it applies to mental illness? There seems no clear cut answer to this dilemma, unless the original philosophical position of dualism is reconsidered, or all mental illness is considered as a problem only of the mind or only of the body. Szaz (1974) has pushed this principle of dualism in psychiatry a long way when he states that there is no such 'thing' as mental illness.

"The notion of mental illness derives its main support from such phenomena as syphilis of the brain, or delirious conditions - intoxications for instance, in which persons may manifest certain disorders of thinking and behaviour. Correctly speaking these are diseases of the brain, not of the mind " (P. 13).

Szaz argues that mental illness refers only to 'illnesses' of the body, i.e. diseases of the brain. Other 'phenomena' labelled as 'mental illness' are problems of 'the mind' brought about by "the problems of living" and should not be thought of as illness in the same way as bodily illness. He argues that many of the observed symptoms of patients undergoing psychiatric aid are not reducible to biological mechanisms. For example, beliefs (e.g. delusions), Szaz argues, cannot be understood or explained in terms of defects or diseases of the nervous system. In presenting such an approach Szaz not only advances a very rigid form of dualism, but also appears to be arguing that the term 'mentally ill' should be reserved for those disorders of brain biology. This approach presents considerable problems for psychiatric thinking, since it implies that the mentally ill are not mentally 'ill' until a biological cause for their 'illness' is found.

Although Szaz's position is somewhat extreme, milder forms of this attitude are detectable in certain psychiatric theories, in the form of a belief in psychological and biological causal dichotomies. These philosophical problems generate considerable confusions and paradoxes in psychiatric thinking. Major confusions arise from the fact that philosophical disputes concerning, first, the concepts of disease (Platonic versus Hippocratic) and, second, the principles

of Cartesian dualism have taken the same stage and are often discussed within the same terms of reference. It was not until the twentieth century that these issues became problematic, but by then these disputes, originally totally different in context, had become so entwined that a clear identification of the issues involved became extremely difficult. Until the twentieth century psychiatry coped with these problems by various assumptions. For example, it was assumed (with a few rare exceptions) that mental illness represented a physical organic illness. With the growth of French psychiatry in the late nineteenth century and the development of the concept of neurosis, such assumptions became less and less acceptable.

V. THE BEGINNINGS OF A PURPOSEFUL SEARCH FOR A SYSTEM OF CLASSIFICATION

For the most part, early post-renaissance psychiatry ignored the mind body problem. On entering an age of new humanism, towards the mentally ill, the emphasis was Hippocratic and on caring and treatment. A notable example of the change that was brought to psychiatry at this time is portrayed in the works of Philippe Pinel (1745 - 1826).

Pinel's contribution to psychiatry was twofold. Firstly he was one of the first to instigate a vastly improved system of caring for the mentally ill. Second, true to the Hippocratic spirit of his age, he was one of the first to advance the idea of careful note taking on individual patients; a procedure to be taken up later in America, notably by Meyer and Hoche.

Following a personnel request to the new revolutionary central beaureau, Pinel obtained the rather sceptical permission to release from their chains all

but the most disturbed of patients in the Bicetre where Pinel was superintendent. Pinel introduced a far more humane approach to his patients, banishing whipping, blood letting, ducking and spinning which were all common practices at the time. Pinel began carefully studying the patients with whom he was confronted, particularly as individuals. Together with a Hippocratic approach to the study of mental illness, Pinel offerred a classificatory system of mental illness very much in the Hippocratic tradition. This classification consisted of : (modern classifications in brackets) : Mania - (included schizphrenia and hypomania) - Melancholia - (included genuine depression, depressed schizophrenia and paranoia) - Dementia - (included general paresis, paranoia, schizophrenia and neurosis) (Ackenknecht, 1959).

Like other classifications to come, Pinel's classification was based almost entirely on observed symptomatology. When Pinel devoted his professional life to the study of mental illness, knowledge was scant and vague and as a result his classificatory divisions were few and simple. During the rest of the 19th century following Pinel's death, many more physicians turned their attentions to the mentally ill. As far as developing classificatory nosologies, many followed the directions of David Skae (1814 - 1873) who argued that symptoms were observable, whilst causes could only be speculated. Mental illness could be 'arranged' in correct order only in accordance with symptoms - the only aspect of mental illness that was believed best understood. Consequently, as Zilboorg and Henry (1941) point out, classifications were

frequently no more than a list of outstanding symptoms which people were unable to explain. In line with developments in other branches of medicine the desire to understand mental illness in terms of natural divisions increased. But the emergence of a new Platonic orientation to medicine, with its emphasis on classification, brought with it less interest in the individual patient. For example, although Maudsley (1835 - 1919) advocated the careful taking of notes on individual patients, he developed a somewhat complex system of classification, and had far less feeling for the individual patient than that of some of his contemporaries, for example D.H. Tuke (1827 - 1895). D.H. Tuke's system was far more simple than Maudsley's and his interests lay firmly with the individual patient. Zilboorg and Henry (1941) comment that with rare exception, the tendency to develop classifications grew in inverse proportion to the psychiatrist's clinical interest in the individual patient. D.H. Tuke was one of the last truly Hippocratic psychiatrists. Spending his childhood in the grounds of the psychiatric hospital set up by his great grandfather, his interest was always in the patient as a human being. Although Tuke's influence on psychiatry cannot be outlined here, his impact on psychiatry was immense. Yet psychiatry was wanting to catch up with other branches of medicine. It was the latter half of the nineteenth century that witnessed the startling discovery of bacteria by Koch and Pasteur. It was discoveries of this kind that psychiatry wanted to emulate, but, if it was to do so, it was necessary to adopt the Platonic concept of disease entities and follow general medicine's example. Thus, as Kendell (1975) points out:

"As, one by one, each of the infections that had dominated the practice of medicine for centuries, tuberculosis, syphilis, typhoid, even cholera and malaria, was shown to be due to the presence of a specific micro-organism, the tacit assumption that all other diseases were also discrete entities, each with its own distinct cause and distinctive symptoms, became almost irresistible . . ." (P. 62).

In fact Psychiatry did not resist such a temptation for very long.

VI. THE SEARCH FOR DISEASE ENTITIES IN PSYCHIATRY

Emil Kraepelin (1855 - 1926) was born during this Platonic orientation in medicine. It was an orientation whose interest lay not so much in the patient as in the clinical phenomenon itself. For all Kraepelin's abilities, he reflected the spirit of his age in that his interest in the individual patient was, more often than not, notably absent. As Zilboorg and Henry (1941) put it, Kraepelin was "greatly interested in humanity but comparatively little in man". Kraepelin collected data from a number of sources and by careful analysis attempted to impose an ordered system of classification based on the assumption that mental illness could be categorised in terms of a distinct and finite number of disease entities.

The development of Kraepelin's system will not concern us here except to outline some of the problems that arose from Kraepelin's approach. The major distinction Kraepelin put forward for psychosis was in terms of dementia praecox and manic depressive insanity. Kraepelin argued that manic depressive insanity runs a cyclic course where the patient suffers from attacks of elation

and depression, often with returns to normal in between attacks. The prognosis was considered favourable, in that these patients recovered. Dementia praecox was characterised by symptoms of catatonia and hebephrenia, often accompanied by auditory hallucinations. Prognosis for such patients was not good, since it was thought that dementia praecox would eventually terminate in a state of dementia proper. Diagnosis was by prognosis, if the prognosis was correct then the diagnosis was correct. This unfortunately led to a rather deterministic attitude to treatment; if the patient suffered from manic depressive psychosis, the patient would recover, if however he suffered from dementia praecox, the individual would eventually deteriorate into dementia. It is a sad reflection that elements of this attitude linger on today.

Although ideas concerning dementia praecox (or schizophrenia as Bleuler renamed it later) had been forming for over forty years, Kraepelin's system did not receive uncontested support. Fifty years earlier Griesinger and also Zeller had scorned any attempt to sub-divide mental illness, asserting that although mental illness could have different manifestations it was essentially a unitary phenomenon. Fifty years after Kraepelin, Menninger (1963) offerred the same rebuke to the Kraepelinian system. Even in Kraepelin's own day there were many who did not accept the Kraepelinian classificatory system. Hoche argued that there was absolutely no evidence that these "well-formed, self-contained, disease entities" actually existed in any pure form (see Lewis, 1967). Hoche viewed Kraepelin's distinct disease entities as no more than convenient symptom clusters. There also appeared factual discrepancies in the Kraepelinian system. Sometime earlier Kahlbaum who

had contributed enormously to the development of dementia praecox being labelled as a disease entity, had reported cycles in catatonia. Three years before Kraepelin published 'Lehrbuch' in 1899, Mendel had argued that neurasthenia could also be cyclical in its course. Thus, as Zilboorg and Henry (1941) argue, these facts amongst others, had to be overlooked in order to preserve the sense of solidity and correctness in the new posological system. Consequently, Kraepelin's system, even from the time of its first emergence, showed certain characteristics of artificiality.

From a historical point of view it seems that the acceptance of the Kraepelinian system by psychiatry was a product of the age. First, as mentioned earlier, the concept of dementia praecox had been forming for over forty years, with notable aid from Kahlbaum. Second, it offered psychiatry a fairly concrete paradigm in the Kuhnian (1962) sense, which had great potential as an integrating framework. Third, the developments and achievements in general medicine placed considerable pressure on psychiatry to produce similar results. The Kraepelinian system seemed to answer the call on behalf of psychiatry.

In a sense Hippocrates won a great victory with the inauguration of the Kraepelinian system; mental illness was firmly in the hands of medicine. Yet paradoxically, in so doing, it had slipped the principles of Hippocrates and had taken up with Plato. Investigations into mental illness became more centred on the disease entity and its characteristics rather than on the individual patient. Schule's Hippocratic style warning to psychiatrists to remember that they were treating sick people, not merely sick brains, went unheeded. As

Zilboorg and Henry (1961) put it, "this principle clashed too much with the newly established harmonious relationship between medicine and psychiatry".

One of the obvious consequences of Kraepelin's classificatory system which became so dominant in European psychiatry was that it had a significant effect on the orientation of future research. This orientation was based on the philosophy of discontinuities between disorders. Depressive illness was most affected by this approach. Unfortunately Kraepelin has left many pitfalls for subsequent researchers. First, Kraepelin's category of manic depressive psychosis was a very inclusive category. Lewis (1967) points out that Hoche (1910) seized on this problem in his argument with Kraepelin over the existence of disease entities. Hoche argued that by Kraepelin's eighth edition (1909), Kraepelin had relegated 'melancholia' (involutional depression) from a disease to a clinical picture -

> "and that it no longer mattered whether there was mania or melancholia, occurrance once in a life or many times, at regular or irregular intervals, whether late or early with predominance of these or those symptoms, it was still manic depressive insanity."

But it was not only the inclusiveness of the manic depressive complex that presented problems for future research. Another major confusion arose from its use of terms and distinctions, such as exogenous/endogenous, reactive/psychotic, which crept into psychiatry at the turn of the century.

VII. THE ESTABLISHMENT OF THE EXOGENOUS/ENDOGENOUS POSITION

The exogenous/endogenous causal distinction was introduced into psychiatry

by Mobius (1893). The concept was borrowed directly from the botanist Caudelle (1813). This fact is of more than historic interest, however, since the botanist's exogenous/endogenous distinction rested totally on a physical discrimination. Similarly, the original psychiatric use of the exogenous/endogenous distinction was to discriminate between different "physical causes" of mental illness. Mobius labelled those illnesses considered to be due to degenerative or hereditary factors (i.e. internal causes) as endogenous disorders. Those illnesses considered to be due to bacterial, chemical, or other toxic agents (i.e. external causes) were labelled as exogenous disorders. As Beck (1973) points out, such definitions left no room for other causal factors, namely social or psychogenic.

Both Kraepelin and Bonhoeffer accepted Mobius's exogenous/endogenous distinction, although Kraepelin did not accept it as a classificatory system, since it was based totally on aetiological considerations. However, Bonhoeffer developed the distinction further and put forward his own concept of exogenous disorders. To Bonhoeffer (1909) (see Hirsch and Shepherd, 1974) exogenous reactions were also of a totally physical origin, they were "modes of response by the brain to injury" (see Lewis, 1971). If a direct relationship between a toxin or injury and mental illness could not be found, it was proposed that these toxins and/or injuries had produced intermediary products in the body, and it was these intermediary products which were responsible for the mental illness. Thus Bonhoeffer not only maintained the exogenous/endogenous discrimination as a purely physical distinction but also allowed for exogenous reactions to be of a psychotic magnitude. In contrast to some modern disriminations which on occasions have loosely equated exogenous with neurotic and endogenous

with psychotic, Bonhoeffer's use of these terms were clear. For Bonhoeffer exogenous and endogenous distinctions referred to differences in physical aetiologies, whereas the term psychotic referred to the severity of the illness, though definitions of psychosis remained vague. However, subsequent semantic confusions were to arise in the use of these terms. At the very time psychiatry was celebrating its strengthened union with medicine, with the delineation of the disease entities, dementia praecox and manic depressive insanity, work was being conducted (notably in France) which was to intrude into this neat nosological system and raise a host of thorny philosophical problems which presented many conceptual difficulties for Kraepelin's classifications.

VIII. PSYCHOGENESIS AS A PROBLEM FOR THE EXOGENOUS/ENDOGENOUS DICHOTOMY, AND THE PLATONIC CONCEPT OF DISEASE

Through the work of Charcot (1825-1893) and later Freud (1856-1939) the importance of psychological processes as the major aetiological factors in neurosis became established. But the actual concept of 'neurosis' was vague and its relationship to psychosis even vaguer. It appears that at the turn of the century, the only illness considered to be of true psychogenic origin was hysteria. For example in 1911 Bonhoeffer wrote an important paper entitled "How far should all psychogenic illnesses be regarded as hysterical?". In this paper Bonhoeffer stated,

" . . . many psychiatrists would today assume that an illness must be hysterical if in its origin and development it can be seen to depend to a marked

degree on psychological causes." (P. 54, see Hirsch and Shepherd, 1974).

Although this statement offers some insight into the current attitude toward psychological factors, it was not a position which Bonhoeffer advocates. In the same paper he put forward the view that psychogenesis could play a major role in the development of other disorders besides hysteria. With regard to depressive illness Bonhoeffer argued,

> "There is, however, a group of depressive illnesses in which the psychological impetus is of prime importance. We are indebted to Reiss for drawing our attention to this group, which he called "reactive depressions". These depressive states are constitutionally based: they occur in individuals who from youth onwards are inclined to take things badly, whose depressive reaction is generally severe and of more than average duration. Severe psychological upsets can be followed by depressive exacerbations of psychotic intensity which are clearly psychogenic since frequently, although not always, when the psychological cause is removed the depressive exacerbation likewise disappears" (P. 57, see Hirsch and Shepherd, 1974).

Bonhoeffer further argued that some forms of epilepsy, paranoia and mania could also be psychologically determined. Bonhoeffer believed that these illnesses could be ascertained by considering precipitating events. All such forms of illnesses Bonhoeffer labelled as reactive disorders [1].

Footnote: [1]

(1) The view that "these depressive states are constitutionally based" may imply that Bonhoeffer believed that 'reactivity' was an endogenous predisposition, thus placing reactivity within the concept of endogenous factors.

Kraepelin accepted Mobius's and Bonhoeffer's exogenous/endogenous aetiological distinction. He further accepted the possibility that psychogenic factors could play a role in certain mental disorders. However, unlike Bonhoeffer, Kraepelin placed psychogenic factors within the concept of exogenous causes, and did not distinguish them from physical exogenous causes, ignoring the concept of reactivity (see Lewis, 1971). This destroyed the purely physical characteristics of the original exogenous/endogenous distinction. In so doing, Kraepelin introduced the old philosophical problem over the existence of a dualism between mind and body into his own nosological system.

Before the absorption of psychogenesis into the exogenous concept of causality, exogenous illness was regarded as physically determined, e.g. through bacteria, toxin and/or injury. The inclusion of non-physical factors within the exogenous concept presented considerable problems since it implied that psychological, non physical, factors could interact with the (physical) disease process, but this position is essentially incompatible with the Platonic concept of a "disease entity". If mental illness is a disease entity, it is a disease in a bodily (physical) sense, brought about by either organic injury, toxins or other physical entities (Bonhoeffer's definition of exogenous), or through an inherent (endogenous) weakness in the physical (genetic) make up of the individual. A disease "entity" in this sense cannot be influenced by non-physical factors. If mental illness is a disease entity (or a number of disease entities) then it follows that mind and body should be regarded as similar (i.e. physical entities), in other words dissolving the dualisms between mind and body at least as far as mental illness is concerned. But if such is the case, if there are

no grounds for separating mind and body, if they are both to be regarded as physical entities, how can non-physical, psychological factors affect a physical mind and induce or influence a physical disease process? On the other hand, if the principle of a mind/body dualism is maintained, then the concept of mental illness as a disease entity begins to become strained, since it is not logically consistent to have a (physical) disease entity of a 'non physical' mind in the same way as a physical disease entity of a physical body. As mentioned earlier it has been this type of issue which has prompted Szaz to adopt his somewhat extreme position.

Yet attempts have been made to save the disease entity concept of mental illness by separating all illnesses of psychogenic origin from the endogenous (physical) illnesses. Thus, as Fish (1974) points out, Schneider has argued that neuroses, psychogenic reactions, personality developments and abnormal personalities are not illnesses in a sense of there being a morbid process in the nervous system, while the functional psychoses are illnesses in this sense. But this solution still leaves the problem of clearly specifying the dividing line between 'physical' mental illness and 'non physical' mental illness. It is the attempts to identify this dividing line that has been at the heart of many of the controversies regarding mental illness, especially those concerning a meaningful classification of depression.

IX. BASIC ISSUES IN MODERN CLASSIFICATION SYSTEMS OF DEPRESSION

The controversies which have developed in discussions on depressive illness have most often revolved around attempts to show clear differences between the

psychogenic depressions and the 'disease entity' depressions. Following the development of the Kraepelinian nosology much was written about the 'exogenous' disorders, with their identifiable external aetiological characteristics, but very little appeared concerning the endogenous disorders. The endogenous disorders remained "hypothetical, intangible, elusive predispositions, constitutional or hereditary forces which could be conjectured but not demonstrated " (see Lewis, 1971). Thus classification in terms of aetiology was dubious since endogenous disorders were by definition simply those disorders for which no precipitatory (or exogenous) factors could be found. Consequently the diagnosis of an endogenous disorder rested on the assumption that if no exogenous event presented itself as the cause of the illness, then the illness must be a product of a morbid disease process in the central nervous system. Partly due to this dilemma, various methods have been used to identify the natural boundaries between the sub types of depression. In general three alternative approaches have been used.

(1) The cross-sectional approach which seeks consistent groups of signs and symptoms (syndromes)

(2) The longitudinal, natural history approach which seeks interrelations between various factors such as family history, age of onset, duration, severity, periodicity and outcome.

(3) The treatment-response approach which seeks to identify sub groups by examining different responses to various forms of therapeutic intervention.

Unfortunately, the semantic definitions of the subgroups studied have presented many confusions. For example, it has been pointed out (Bowman and

Rose, 1951) that the distinction neurotic/psychotic usually refers to no more than vague differences in severity of illness. Further, for reasons outlined above, the exogenous/endogenous distinction used by Kraepelin presented logical problems (Lewis, 1971). Realising the confusion produced by the inclusion of psychogenesis in a definition of exogenecity, Ewald (1948) (see Lewis, 1971) argued for a trichotomy of exogenous, endogenous and psychogenic. However, while the logicality of these distinctions was accepted, it has not become an established classificatory system. To confuse matters further, many writers have used terms concerning aetiology and terms concerning severity synonymously. Thus, as Kendell points out, some authors, including himself, have

> ". . . regarded 'psychotic' and 'endogenous' as synonymous, and 'neurotic' and 'reactive' likewise, using either pair of terms to denote two contrasting syndromes - the one consisting of severe, unvarying depressions, often with an acute onset, and accompanied by retardation, guilt, diurnal variation of mood and severe insomnia and weight loss; the other consisting of milder illnesses, often accompanied by anxiety, prone to vary from day to day, with self pity and histrionic outbursts rather than guilt, and complaints of anorexia and weight loss" (1976). (P. 17)

On the other hand, some authors, notably those from North America, have been more specific in the use of these terms. For them, the distinction between neurotic and psychotic refers only to a consideration of severity, whilst the reactive (exogenous)/endogenous distinction refers only to aetiological considerations. According to this use of these psychiatric terms, reactive psychotic illness and neurotic endogenous illness would be quite meaningful clinical descriptions. Thus

considerable confusion has been generated over the differential use of certain psychiatric terms, often resulting in misconceptions regarding the type of patients who have been placed in one subgroup or another. Furthermore, although various studies have often used aetiological terms and distinctions as a description of their 'classified groups', these studies have not been directly concerned with aetiological considerations. Rather, as Becker (1974) points out,

> " . . . they are chiefly concerned with whether depression is unitary or binary, that is, a single syndrome or two syndromes (endogenous and reactive), and whether the syndrome or syndromes is/are categorical or dimensional, that is, whether they are discrete entities (categorical) or whether they are normally distributed and occur in varying combinations with each other or with other syndromes (dimensional) " (P. 38).

These disputes continue to reflect a failure to solve the Platonic (qualitative, categorical) versus the Hippocratic (quantitative, dimensional) concept of disease. Thus at the heart of these disputes remains the fundamental problems of the philosophical orientation to the concept of disease. Eysenck (1970) has argued that in the study of depressive illness, each of these two philosophical approaches has two basic positions. For the Platonic qualitative concept of a disease entity, depression can be either unitary categorical or binary categorical. For the Hippocratic concept of disease, depression can be either unitary dimensional or binary dimensional.

X. CONTROVERSIES OF EVIDENCE AND POSITION

The recent history of the search for a classification of depression has

provided advocates for each of the four positions. Although a detailed discussion of the relative merits and backgrounds behind various researchers' adoption of one position or another is not relevant to this thesis, a brief outline of some of the issues and problems should be examined in view of the model of depression which will be advocated later.

Eysenck (1970) has argued that Mapother and Lewis both held a unitary categorical view of depression. However, while Lewis was certainly against the sub-division of depression, the evidence is unclear as to whether Lewis believed that depression was a well delimited, clearly separable illness, distinct from all other illnesses. Yet a necessary condition for both the unitary and binary categorical view of depression, must be the demonstration that depression is a clearly distinguishable and separable 'disease'. Unfortunately, as Kendell (1975) is at pains to point out, there is no clear evidence for distinguishing affective illness from other disorders such as schizophrenia or anxiety. The common use of the term schizo-affective psychosis in part attests to this situation. In fact Kendell argues that there is, as yet, no clear evidence to 'establish the existence of any functional disease entity'.

However, Roth and his colleagues from Newcastle believe that they have provided the necessary evidence, not only for distinguishing between 'neurotic' and 'endogenous' depression (Kiloh and Garside, 1963, Carney et al., 1965) but also for distinguishing between depression and anxiety states (Gurney et al., 1972). Their position on depression is essentially the binary categorical position.

Attempts to replicate these findings have unfortunately not provided over-

whelming support of the binary categorical position. Kendell (1968, 1975, 1976) argues that factor analytic studies produce results which favour a dimensional rather than a categorical view of depression. Kendell (1968) is in favour of a unitary dimensional approach to depression and argues that a continuum model more accurately forecasts treatment and outcome than traditional categorical diagnosis. Eysenck (1970) is in agreement with Kendell on the need for a dimensional model of depression, but argues that two dimensions, neurotic and psychotic, are required (binary dimensional). Considerable information is lost, he believes, if these two separate dimensions are collapsed, apart from being an unjustifiable statistical procedure. Both Kendell (1968, 1975, 1976) and Eysenck (1970) (see also Becker, 1974) have criticised the Newcastle findings from a sampling and statistical point of view. It appears at present that the dimensional approach may be the more profitable approach, especially from a research point of view. As to whether one or two dimensions are required remains in dispute. As Kendell (1976) points out, essentially the same argument that Eysenck (1970) makes for having two dimensions instead of one, can be made for two, three or four dimensions and so on. The number of dimensions really depends on the purpose for which the classification is designed.

The controversies regarding the endogenous-reactive dichotomy continue. The preference for a dimensional model of depression reflects the return to an Hippocratic age. But what a dimensional model of illness implies for the mind/body problem is unclear. Attention has been focussed (e.g. Kendell, 1975) more on the implications of these philosophical controversies for medicine rather than psychology. Nevertheless

the mind/body problem still remains, even if it has been stowed away for the time being.

XI. ALTERNATIVE CLASSIFICATORY SYSTEMS

Not all new classification systems to appear in the last forty years have been directly concerned with Platonic versus Hippocratic or reactive versus endogenous issues. A recent and quickly established distinction is between unipolar and bipolar depressive illness. Perris (1966) using this distinction first introduced by Leonhard (1959) has shown that there are differences in age of onset (bipolar illness commences approximately 15 years earlier than unipolar illness), duration of illness (bipolar illness tends to be of shorter duration than unipolar illness) and personality difference (bipolar patients tended to be warmer, more energetic, and more extroverted on recovery than unipolar patients who tended to be anxious and tense and withdrawn). Blackburn (1972) has replicated these findings and also argues that bipolar depressives are more retarded than unipolar depressives, and that whilst ill unipolars are more extrapunitive than bipolars.

However, Kendell (1976) argues that no consistent differences in symptomatology have yet been demonstrated between unipolar and bipolar illness. The definitions of patients filling each group are unclear. Three consecutive unipolar depressive attacks without evidence of mania may not be sufficient to ensure that the patient will never develop mania in the future. It is also unclear as to what the status should be of the patient who has a brief attack of mania after treatment with the anti-depressant tricyclic compounds or electric convulsive therapy (E.C.T.). Further, there is as yet no clear evidence that unipolar and bipolar illness respond

differently to different treatment procedures (though this appears to be a promising area, but more evidence is required).

Most importantly, even if unipolar-bipolar does prove to be a useful distinction, the reactive-endogenous problem will still require major consideration, Is it possible that unipolar illness can be reactive or endogenous? If so, are they best represented by a categorical distinction or a dimensional system? Can bipolar illness also be reactive or endogenous? Although cases of reactive hypomania are rare they do occur (Ashcroft, 1977). Further, what is the relationship between unipolar and bipolar illness? Should they be regarded as separate disease entities (Blackburn, 1972) or as a dimensional nosology, where perhaps personality scores (for example extraversion-introversion) provide one of the axes. And lastly how far is the concept of unipolar bipolar illness only relevant to depression? Could schizophrenia also have unipolar (withdrawn, retarded) and bipolar (over-active, pressure of speech) components? Certainly some forms of mania are often misdiagnosed as schizophrenia, especially in America. The frequency of such misdiagnoses (see Beck, 1973) suggests that some forms of schizophrenia and some forms of mania may appear very close (Loudon, Blackburn and Ashworth, 1977).

These issues give rise to some important considerations. If the unipolar-bipolar distinction is shown to be highly correlated with differences in pre-morbid personality, then to what extent can one be sure that unipolar illness and bipolar illness represent separate 'disease entities' (Blackburn, 1972) rather than different expressions of the same morbid 'disease process'? In other words, even if it is assumed that depression is the result of a physical (e.g. biochemical) dysfunction,

then to what extent can cognitive and personality factors change the manifestation of this physical dysfunction? Interestingly Whybrow and Parlatore, (1973) point out that a number of studies have shown that cerebral spinal fluid (C.S.F.) levels of 5-hydroxyindoleacetic acid (5-HIAA) the metabolite of 5-hydroxy-tryptamine are low in both depression and mania. These findings should be taken to indicate that to view depression and mania as biological opposites may not be correct. In fact biochemical research has shown them to be more alike than different. Further consideration of such problems will be given in the next chapter.

Many other classificatory systems of depression exist (e.g. primary and secondary depression). Further discussion of such systems is not relevant to this study and excellent reviews already exist (Beck, 1967, 1973; Kendell, 1976; Mendels, 1970; Becker, 1974; Levitt and Lubin, 1975; Blackburn, 1972). In fact, as Kendell (1976) points out, the present day position sees an abundance of classifications where nearly every conceivable nosology, from one up to thirteen distinctions, have been put forward at one time or another. More worrying perhaps is that each has provided some more or less plausible evidence to support it. Thus, the search for the 'natural boundaries' between syndromes, types and sub-types of depression continues. But the major stumbling block for a classificatory system remains firmly rooted in the reactive-endogenous dichotomy. Kendell (1975) hints at the possibility that if psychotic illness can be clearly differentiated from neurotic illness, then it may be necessary to have a series of categorical sub-groups for psychoses (schizophrenia, depression, mania, dementia, etc.). This is especially true if a direct link between these psychoses and genetic

(endogenous) factors are demonstrated. Neurotic illness on the other hand would be best described by a dimensional system. However this view simply underlines the proposition that neurotic illness should not be viewed as a disease in the sense of there being a morbid process in the central nervous system, whereas functional psychoses can be viewed in this way. But this approach leads straight back to the trail in search of the natural boundaries between reactive and endogenous (neurotic and psychotic) illness, the existence of which Kendell (1968) himself was unable to find in depression. Furthermore such a position argues strongly for the claim that there is a (qualitative) difference between "non-physical" (psychogenic, neurotic and/or reactive) mental illness, and physical (endogenous and/or psychotic) mental illness, thus carrying with it the assumption of a boundary and distinction between mind and body, i.e. maintaining the concept of mind/body dualism. Other implications may also follow. Does Kendell's (1975) position, for example, imply that genetic or other physical factors only give rise to psychotic illnesses; or, that psychogenic or reactive factors only give rise to neurotic illnesses? The answers to these questions are unclear but oddly seem to carry an implicit affirmation if Kendell's view is accepted.

These are some of the special conceptual problems that mental illness presents to the researcher. They are problems that are so deeply rooted in outdated philosophical controversies, and present so many paradoxes and confusions that it may be better to abandon them altogether and start afresh. In other words, the concept of a mind/body dualism born out of a necessity to solve a religious/scientific dispute should be dropped, along with the almost

three thousand year old Platonic versus Hippocratic concept of disease, at least in its "either/or" form. To replace the former, a view of man as a multi-component response system can be put forward. In such an approach, man may be viewed as being composed of a number of component systems or levels including biochemical, physiological, behavioural and cognitive component systems. Each level or component system is separate and distinct from every other level or component system, and can be defined in terms of its fundamental structure and mode of operation. Yet each component system is delicately interrelated in terms of cause and effect with other component systems. It is this concept of a multi-component systems approach which will be taken up more fully in the next chapter.

XII. CONCLUSION

This chapter has not attempted to provide a detailed and comprehensive review of all the studies pertaining to the problems of classification. Rather it has attempted to highlight particular historical and philosophical factors that may account for some of the confusions that exist in this area.

As Kendell (1976) has argued, the history of the disputes over depressive illness reflects the history of disputes over mental illness in general. Depressive illness has often acted as a 'test case'. An examination of its long history reveals a number of unresolved philosophical issues which are evident in many of the assumptions concerning mental illness. The most important philosophical controversies revolve around the quantitative (Hippocratic) versus qualitative (Platonic) view of disease, and the justification or otherwise of regarding mind

and body as separate entities. It seems reasonable to conclude that through the integration of these two issues in the study of the phenomenology of mental illness, so many paradoxes and confusions have occurred, that it may be more profitable to adopt an alternative view of man and his illnesses rather than to seek their resolution. In this respect the most likely alternative view of mental illness could be provided by the multi-component (also referred to as the multi-dimensional and multi-disciplinary) approach. Although this approach is compatible with Hippocratic philosophy (see P. 10), it has most in common with the philosophical principles of the psychobiological school, founded by Adolf Meyer. It is to this alternative approach, which provides a more flexible and useful view of mental illness, that attention is turned in the next chapter. Moreover, the philosophy of psychobiology provides a more concrete base for the development of a multi-component model of depression.

CHAPTER THREE

A PHILOSOPHICAL, THEORETICAL AND EMPIRICAL DEVELOPMENT OF A MULTI-COMPONENT APPROACH TO DEPRESSION

I. INTRODUCTION

In the preceeding chapter it was argued that psychiatric thinking has encountered two major philosophical problems. These problems express themselves in the disputes concerning the neurotic/psychotic distinction, and the search for qualitative differences between psychiatric syndromes. In this chapter the development and philosophical orientation of pragmatic psychobiology, which offers a useful alternative methodology that overcomes some of the problems inherent in Cartesian dualism, will be examined. In very general terms, pragmatic psychobiology encompasses those approaches to psychopathology and psychology that adopt multi-disciplinary, multi-component or multi-level explanatory frameworks. Having traced the historical and philosophical perspectives of this approach, it will be shown that the study of normal emotion has benefitted enormously from such an orientation. It is argued that in the study of abnormal emotion, Akiskal and McKinney's (1973, 1975) model of depression constitutes the most advanced multi-disciplinary model available. As in the study of normal emotion, such a multi-disciplinary or pragmatic psychobiological approach offers enormous advantages to the conceptualisation of depression, yet at the same time avoiding any firm commitment to a specific disease entity view of depression. Within the same methodological tradition as Akiskal and McKinney, the second

half of the chapter attempts a slightly different interpretation of the data pertaining to depression. Special emphasis is placed on the concepts of stress evaluation and coping behaviour. The last part of the chapter attempts to draw the many aspects of depression that are discussed together, and demonstrate, with the use of a model, how distortions in stress evaluation and maladaptive coping behaviour can precipitate a chain of events, including biological change, that combine to produce a depressive illness.

II. A PSYCHOBIOLOGICAL VIEW OF MENTAL ILLNESS

A multi-component approach to illness has not, until comparatively recently, enjoyed any dominant position in the history of medicine. This is possibly because it has not been advanced in any great philosophical position, although Hippocrates very occasionally favoured such an approach (P. 10). Yet, as discussed in the last chapter, the history of medicine and especially psychiatry has reflected to a considerable degree the ebb and flow in popularity of certain of the major philosophical positions. In view of this it is probably fair to argue that the origins of the multi-factor or multi-component approach to mental illness lies in the recent past, as exemplified by the work of Adolf Meyer (1866-1950). Meyer's approach to psychiatry was aptly named the psychobiological approach, because it stressed a consideration of the interactions between psychological and biological events.

Swiss by birth, Meyer moved to America early in his career. Though European in origin, Meyer did not follow the European approach to mental illness for very long. Shortly after moving across the Atlantic, Meyer advanced his own classificatory system based upon various psychopathological reactions.

Kraepelin's nosology of disease entities, hailed in Europe as a great advance for psychiatry, was considered totally misleading and a hindrance to the treatment of the individual patient, by Meyer and his colleagues. Only through an understanding of the total personality in all its aspects, Meyer argued, could the individual patient be understood and treated.

To Meyer and the psychobiologists, causation was considered a continuous process, involving interactions between all the various aspects of the individual's life. As Zilboorg and Henry (1941) point out, Meyer:

> ". . . would incorporate all available data into the picture of the phenomena of pathological mental reactions. To him the total picture is first the summation, then a possible synthesis of all the forms and aspects of the life of the individual - organic, sociological, general cultural and purely psychological" (P. 503)

To select any one particular link in this continuous chain for special emphasis was, for Meyer, to distort the true nature of the illness.

Mayer-Gross et al. (1969) have argued that Meyer's psychobiological approach to mental illness was founded on two cardinal philosophical principles:

(1) The uniqueness of the individual patient, and

(2) The unity of mind and body and the necessary combination of both psychological and biological aspects in all cases of mental illness.

From a clinical point of view, it was the first of these principles that produced most criticism of the psychobiological approach. Because of Meyer's insistence that each patient be treated as unique, classifications based on hypothetical disease entities were regarded as confusing and damaging. Consequently treatments such as electric convulsive therapy (E.C.T.) and insulin

coma considered by European psychiatrists to be effective with certain types of patients having certain types of diseases, were largely ignored by the psychobiologists. According to Mayer-Gross et al. (1969), the only treatment that arose from the psychobiological school was a form of psychotherapy, sometimes referred to as 'personality analysis', which though interesting has not proved particularly useful in practice. These authors go on to argue:

> "It is also difficult to overlook the weakness of a system that ranges all types of reaction, neurotic, psychotic and organic, side by side, as if there were little to choose between them in depth of abnormality or in causative factors involved." (P. 15)

From a clinical point of view, many regarded the psychobiological approach as weak and unhelpful. As Mayer-Gross et al. (op. cit) pointed out, many psychiatrists believed that in opposing Kraepelin, Meyer went too far and "threw the baby out with the bathwater". But in a sense the supporters of the Kraepelinian system may also be guilty of the same error in their rejection of Meyer. While Meyer's insistence on the individualism of each patient has provoked justifiable criticism, it seems unfortunately the case that Meyer's concept of mind body unity and his belief in causation as a continuous process have byandlarge been passed over. It is only recently that these latter two principles have received further attention.

The growth of knowledge concerning the functioning of the nervous system has permitted a closer and more detailed examination of the correlation between mental events and biological functioning. The fruits of this research have lent considerable weight to Meyer's belief in the unification of mind and body. Such unification would seem central to Akiskal and McKinney's (1975) model of

depressive illness, and Lader (1972) has pointed out the necessity for such a view in both psychosomatic illness and emotional states. Even in general illness, the Meyerian view of mind body unity has received some support (Engel, 1972, 1977). For Meyer though, without the insight of modern research, the belief in the unity of mind and body was a philosophical belief rather than a view based on objective evidence. In this regard there is reason to be cautious of Meyer's resolution of the old Cartesian issue.

III. PHILOSOPHICAL PROBLEMS WITH THE UNITY OF MIND AND BODY

Lewis (1967) points out that the psychobiological approach presents us with a monistic view of man; that is man is seen as a psychosomatic unit. This psychosomatic unit is made up of "integrated parts describable in differing languages according to the hierarchical level of integration". Quoting from Meyer (1915), Lewis (op. cit.) adds:

> ". . . As psychobiologists we treat the functions of total organisms which blend, with more or less consciousness, in a manner constituting a special level of integration which has been especially and most characteristically enriched by the interindividual and social development of language . . . All that constitutes psychobiology to the physician is therefore physical as well as mental. We can further recognise an ultrabiological level of facts when we consider the products of such functioning as logic and mathematics or theory of relations . . . In this way we obtain an orderly perspective of the various sciences but eliminate the contrast between physical and mental." (P. 174)

Thus, for Meyer, mind body unity was established because of the "ultra-biological level of facts". In other words, the distinction between mind and body

dissolves as mind is engulfed within an integrated system based on the hierarchy of the biological sciences. For such a view mental activity is simply the product of integrated biological mechanisms. Lewis (op. cit.) clarifies this position when he points out that:

"Those who think in this way constantly invoke integration - 'all the facts of experience prompt us to see in mentation a biological function, and we are no longer surprised to find the product of integration so different from the nature and functions of all the component parts. All the apparent discontinuities . . . are shown to be a general feature of nature and of fact'." (P. 174-175)

It would therefore seem that for the psychobiologist mentation was simply the product of the integration of a number of systems which ultimately rested on biology. This is a solution that has not been uncontested. Lewis (1967) seriously doubts its validity. He agrees with Sherrington and others that integration is not a sufficient explanation for mental activity. Moreover Lewis (op. cit.) contests that on evolutionary grounds there is no parallelism in the development of mind and body. They have distinct evolutionary paths and histories. Lewis (op. cit.) goes on to argue that even psychobiology has not completely resolved the problem of dualism without some loss in philosophical consistency.

Clearly these complex philosophical issues require consideration, but there is reason to doubt whether pragmatic psychobiology should unduly hinder itself with them. Whether integration provides a sufficient explanation of mental activity need not necessarily be an important issue for the pragmatic psychobiologist. Given that research has often demonstrated interrelationships between psychological and biological events, it seems justifiable simply to avoid the mind

body issue. Lader (1972) argues that the adoption of a philosophy of psycho-physiological parallelism sidesteps the controversies of the mind body problem, though as mentioned before parallelism, along with integration, is not necessarily a consistent solution. Lewis (1967) has opposed such a solution on evolutionary grounds. More importantly perhaps, parallelism does not seem a particularly useful or necessary assumption. If any assumptions are required, it is logical to have as few as possible. Thus it seems that pragmatic psychobiology need only make the assumption that mental activity is able to operate as another type of response system within the hierarchy of component systems that make up the integrated psychosomatic unit or multi-component complex. The form such activity may take, be it cognitive appraisal or contingency learning, need not be relevant to the practical application of the psychobiological paradigm. The only assumption which is required is that mental activity be considered as having a specific locus of influence within the integrated framework of the multi-component complex. Further, this assumption seems necessary in view of the wealth of data that have demonstrated the effects of psychological manipulations on various biological parameters (for example Mason, 1968, 1972; Lazarus, 1966; Weiss, et al., 1970; Weiss et al., 1976; Schildkraut and Kety, 1967; Lange, 1971; Frazier et al., 1969; Lockhart, 1973).

Many models of depression, including the model that will be developed later, rest on this concept of psychological/biological interactionalism. It has been for this reason that the philosophy behind such an approach has been discussed. Before developing this model, the axiom and background on which the model is based require further explanation.

IV. A MULTI-COMPONENT APPROACH AS AN EXTENSION OF PSYCHOBIOLOGY

The last thirty years have seen a tremendous expansion in the technology and consequent knowledge in both the biological and psychological disciplines. Such an expansion has greatly facilitated the study of the relationship between biological and psychological events. Nearly all areas of research concerned with these issues have demonstrated that psychological and biological processes interact and feedback on each other to a highly significant degree. It is not relevant here to review or discuss the vast volume of such data but Mason's (1968) review paper on the functioning of the pituitary-adrenal cortical system is somewhat typical of the findings of such research. Mason concludes his paper by saying:

"There is no longer room for reasonable doubt as to the validity of the basic conclusion, that psychological stimuli are capable of influencing the level of pituitary-adrenal-cortical activity."

More forcefully Mason (op. cit.) argues,

"Psychological influences are among the most potent natural stimuli known to affect pituitary adrenal activity."

Similar contentions exist in many biological areas, supporting the view that a concept of mind body unity, regardless of philosophical problems, has considerable scientific validity.

Meyer's idea of the interaction between various aspects of the individual's life can today be better expressed as multi-component interactionalism. These various component systems might be genetic, biochemical, physiological, psychological and sociological, etc. With regard to terminology, multi-component or multi-disciplinary analysis is simply the descriptive term for those studies

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that attempt to trace the changeable and flexible relationships between various component systems.

There is, however, a degree of vagueness as to what actually constitutes an individual component system. Often, component systems are defined within the boundaries of particular disciplines, e.g. biochemistry, physiology and psychology, but, more precisely, component systems tend to be considered as separate entities if the processes and products of the component system can be defined and analysed in a particular and idiosyncratic manner. For example, biochemical processes produce various metabolites which are measurable from cerebro-spinal fluid (C.S.F.), blood and urine. When analysing this type of data, these metabolites are normally considered and understood to be the product of the biochemical system which operates according to certain biochemical laws, e.g. protein synthesis, neurotransmitter uptake mechanisms, etc. Similarly, expectancies, or coping processes, are considered to be the products of the psychological system which operates according to psychological laws, e.g. coding and decoding of information, construct formation, etc. It seems that the separateness of each system depends on the separateness and idiosyncratic nature of its output. However, these distinctions between component systems remain pragmatic rather than objective. Whether, in fact, systems should be considered as separate units in this manner is questionable. Furthermore, as each discipline expands and develops, various systems can be subdivided, possibly complicating rather than clarifying pragmatic psychobiology. Nevertheless, given the notion of loosely definable separate component systems, the study of the interaction between these systems has greatly facilitated research in a number of areas, especially in psychosomatic illness and in emotion, both normal and abnormal.

V. PROBLEMS IN THE STUDY OF EMOTION BEFORE MULTICOMPONENT THEORIES

Before discussing Akiskal and McKinney's (1975) multicomponent model of depression, some consideration should be given to the study of emotion. This is because explanations of emotion, like Akiskal and McKinney's model of depression, have demonstrated that it is the combination of events, biological and psychological, that determine final phenomenology. Also the problems that theorists of emotion have had to face are similar in kind (though not content) to the problems facing the researcher in depression.

The study of emotion, like the study of depression, has undergone a number of stages where first one component system was emphasised only to be subsequently replaced by another. The early theories of emotion stressed the peripheral physiological aspects of emotion. James (1884) argued that it was peripheral physiological changes that determined emotion. Lange (1922), through independent research, stressed the same idea but highlighted physiological changes in the cardiovascular system. To both these workers emotion was a secondary event consequent to bodily changes. But evidence accumulated to show that the James-Lange theory of emotion was inadequate. Sherrington (1900, from Tyrer, 1976) demonstrated that dogs could display some emotional behaviour after complete transection of the spinal cord in the cervical region. It was later observed (Prideaux, 1922, from Tyrer, 1976) that there is a delay in the physiological response to an emotional stimulus, often the subjective report preceeding any physiological change.

Over the past fifty years a variety of theories of emotion stressing different factors or different component systems have been put forward. Most notable of

the early theories was Cannon's thalamic theory which offered the first major alternative to the James-Lange theory. Although still a physiological theory of emotion Cannon shifted the focus of interest from peripheral physiology to a central brain structure - the thalamus. Later Bard added the hypothalamus and argued that emotional behaviour depended upon activity in the thalamus and hypothalamus. But as investigations of the limbic system proceeded during the 1930s and 1940s, it became increasingly clear that the Cannon-Bard theory was also inadequate and that many other structures in this part of the brain mediated emotional behaviour. For example, Kluver and Bucy (1937) demonstrated the importance of the amygdala complex in emotion, and septal lesions were found to produce a very easily enraged animal (from Milner, 1971). Such studies led Papez to propose that it is not one structure but an integrated circuit of structures (the Papez-Circuit) which regulates emotion.

Unfortunately those various efforts to trace specific physiological mechanisms, circuits and pathways, both peripherally and within the brain, as being the basis of emotion, have met with only limited success. It was neurophysiological research in another area of the brain that was to put emotion on the road to multi-component analysis.

VI. AROUSAL AND EMOTION

In 1949 Moruzzi and Magoun published their important findings showing that stimulation of part of the brain stem (the reticular formation) produced a high frequency low voltage electroencephalograph (E.E.G.) resembling that of an awake animal. These investigators interpreted their work as demonstrating that the reticular formation is responsible for maintaining arousal in a normal animal.

Following the publication of these findings, psychologists saw in the reticular formation the physiological system to explain many psychological events, from selective attention and consciousness to emotion. As Milner (1971) points out, many of these theories went far beyond the limits afforded by the available data. Nevertheless, for the study of emotion, the concept of arousal was to prove invaluable. In 1950, Lindsley et al. found that chronic lesions to the ascending pathways of the reticular formation (later named as the ascending reticular activating system) in cats produced emotional apathy and somnolence. Lindsley argued that the ascending reticular activating system (A.R.A.S.) was important for E.E.G. activation which roused the animal under suitable stimulus conditions and simultaneously activated lower brain stem centres responsible for bodily changes. Lindsley (1952, from Tyrer, 1976) also believed that arousal could be regarded as an unidimensional phenomenon which ranged from deep sleep associated with slow frequency high voltage (delta) waves, to extreme arousal or excitement associated with high frequency low voltage, asynchronous (beta) waves on the E.E.G. Several workers followed this view, emphasising the importance of central and peripheral arousal in states of emotion. As a result, the idea that arousal was essential for the expression and experience of emotion became a dominant theme in certain theories of emotion.

Unfortunately both the A.R.A.S. and the concept of arousal have not proved as straightforward or as enlightening as was first hoped. In 1962 Feldman and Waller demonstrated that the A.R.A.S. was only important for E.E.G. arousal and that behavioural arousal depends on higher brain areas, the most important being the hypothalamus. More confusion has arisen from clinical reports of patients with mesencephalic damage. For example Lhermitte et al. (1963) reported on a

patient who had suffered severe damage to the mesencephalic reticular formation. In spite of his comatose appearance, he was still able to answer quite complex questions by using his only remaining response, a slight flexion of the forearm. More recently Lacey (1967) has pointed out that there is a poor correlation between different autonomic measures in aroused states. Such data cast serious doubts on the usefulness of a unidimensional view of arousal.

In consequence to these and other problems, many researchers are now agreed that the concept of arousal is in need of considerable modification, and various attempts have been made to modify the concept of arousal. For example, Routtenberg (1968) has offered a two structure system of arousal. The first structure is the reticular formation responsible for maintaining tonic alertness and orientating the animal to incoming stimuli. The second structure (or system of structures) is the limbic system which is activated by specific negative or positive reinforcing events.

As yet it is not entirely clear what such modifications imply for a theory of emotion. The fact remains, however, that although the importance of arousal is firmly established in theories of emotion, emotion is not simply arousal. Many other factors of equal importance interact with arousal and it is these interactions that produce the different emotions. In 1962, Schachter and Singer argued that the most important of these 'additional factors' was cognitive labelling.

VII. COGNITIVE LABELLING; AN EARLY MULTI-COMPONENT MODEL OF EMOTION

According to Lykken (1968), Schachter and Singer's (1962) experiment developed from two observations made some time earlier. In 1924 Maranon drew attention to the fact that peripheral sympathetic arousal (produced by an injection of

adrenalin) yields an "as if state" of emotion. Individuals receiving such treatment often expressed their feeling state as "I feel as if I ought to be frightened but I am not". On the other hand it was later observed that individuals who are deprived of peripheral sympathetic arousal by high spinal lesions appear to have a distorted and shallow subjective experience of emotion. They commonly express their feeling state as "I know I am angry but I don't really feel angry". (from Lykken, 1968).

Schachter and Singer (op. cit.) and Schachter (1964) regard these two sets of introspections as opposite sides of the same coin. In the first instance when individuals experience peripheral sympathetic arousal without cognitive labelling, no true emotion develops. In the second instance cognitive labelling in the absence of peripheral sympathetic arousal produces emotion which is weak and shallow. In their classic experiment (Schachter and Singer, 1962), these workers demonstrated that emotional experinece and expression depends jointly on peripheral sympathetic arousal and cognitive labelling; more specifically, that the intensity of the emotion depends on physiological arousal, but the type of emotion experienced and expressed depends on cognitive labelling.

Schachter and Singer's (1962) findings again highlight problems inherent in Cartesian dualism, since their multi-component model of emotion involves the interaction of both mind and body (psychological and physiological) events. Their work also present problems for the concept of mind body parallelism. These problems arise because according to this model, emotion cannot be ascribed to one event in the body being paralleled or reflected in the mind (or vice versa). Instead, the complexities of emotion are the products of interactions between totally different component systems. Although Schachter and Singer's work offers

a challenge to the philosophical debates of the mind, since 1962 a number of weaknesses in their original model have emerged (for a discussion of these see Lange, 1971). The most serious weakness is the failure of the model to explain adequately how, under normal circumstances, the individual becomes physiologically aroused such that cognitive labelling of this arousal becomes necessary. In an effort to overcome these problems, theories of emotion have had to extend the number of components required for an adequate theory of emotion. Thus, for example, Lader's (1972) model includes components such as C.N.S. arousal, peripheral arousal, genetic disposition, internal drives, cognitive appraisal, cognitive labelling and verbal report. These more complex models need not be discussed here, except to mention that there is growing evidence to suggest that cognitive appraisal (Lazarus, 1966) may be an important determining factor of initial physiological arousal.

VIII. AKISKAL AND McKINNEY'S MULTI-COMPONENT MODEL OF DEPRESSION

The purpose of briefly examining the developments of present day theories of emotion, from their unicomponent (physiological) beginnings to the more modern multi-component interactional models, has been to demonstrate the importance and necessity of avoiding one level of unicomponent explanations of phenomena which are inherently multi-factor or multi-component. Moreover, the problems and refinements made in the research and theories of emotion may have much to offer by way of methodological pointers for the study of pathological emotional states such as depression. As Akiskal and McKinney (1975) point out:

> ". . . depressive phenomena are neither inherently psychological (reactive) nor organic (endogenous). Like other emotional states, they should be defined at psychological (verbal-cognitive-experimental), somatic (physiological-

biochemical) and psychomotor (motor behavioural) levels. As the final common pathway of various processes that impinge on the functioning of the organism, melancholia can be described in many frames of reference, both psychological and biological, and at multiple levels of sophistication. One of the major challenges for modern psychiatry is to build conceptual bridges between these various frames of reference." (P. 288-289).

Moreover, if Schachter and Singer's (1962) model and later models of emotion do challenge the principle of Cartesian dualism, then it is arguable whether there are adequate grounds for maintaining such dualism in the study of pathological emotional states. This question is especially important when it is observed that on phenomenological grounds the boundaries dictated by Cartesian dualism are difficult if not impossible to find (chapter two).

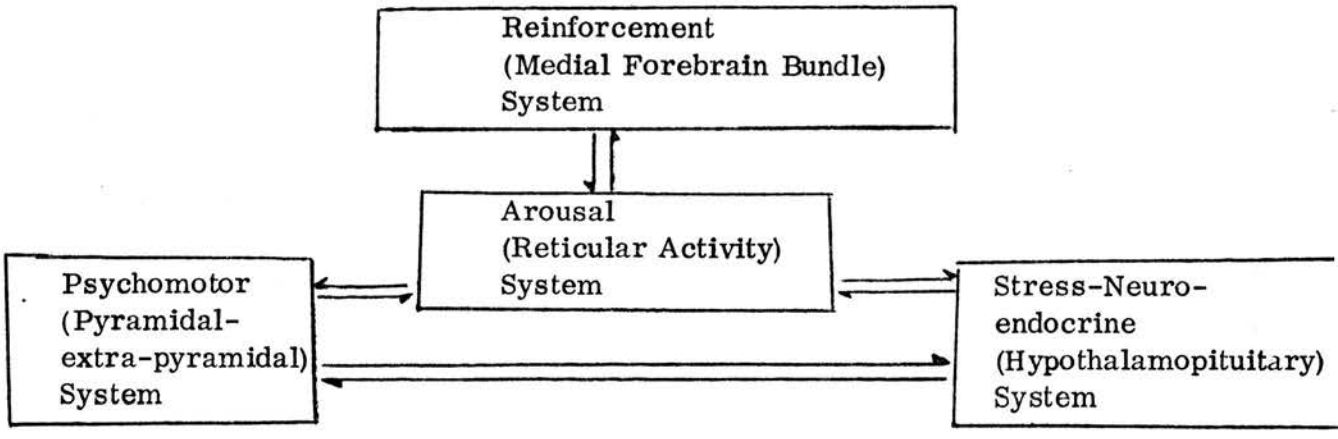
Akiskal and McKinney's (1975) model of depression represents the most comprehensive and conceptually useful model so far advanced. The model gains additional strength from its ability to partially circumvent the old Cartesian and Platonic versus Hippocratic philosophical controversies. In the first place the model demonstrates that cognitive and behavioural approaches to this disorder need not be seen as acting competitively or in opposition to the biological approaches. The principle of integrated component interaction allows for changes in one component system or level to have effects on other levels or component systems. Thus, cognitive events may produce physiological changes and vice versa. With respect to the problem of a mind-body discontinuity, this model has adopted a pragmatic psychobiological solution. The model has no difficulty in avoiding this old philosophical problem because purely on the basis

of objective evidence, which is the foundation of their approach, mind and body are shown to share a complex but well integrated relationship. In other words, Akiskal and McKinney's (op. cit.) model has the implicit assumption, necessary on the basis of evidence, that psychological processes (many of which share the characteristics attributed to the mind, e.g. the power of reasoning or information processing (Mandler, 1975)) act as a particular response system integrated within a multi-component complex.

Second, the model allows us to handle the apparently different symptomatology of severe psychotic depression, as opposed to mild neurotic depression, without recource to a philosophy of discontinuities or Platonic disease entities. Instead, an examination of the various component systems that interact in the production of depression allows for the possibility that one system can become so distorted or dysfunctional (e.g. biochemical pathways of the medial forebrain bundle) that it behaves autonomously. This could result in a breakdown in the feedback between integrated systems, allowing the disordered system to dictate events in other systems and thus substantially influence the course and symptomatology of the illness. Akiskal and McKinney believe that the tendency for a physiological system to become autonomous may depend on genetic factors, i.e. the individual has a genetic vulnerability in the negative feedback mechanisms of a particular system. The evidence on this issue is plausible but on theoretical grounds there is no reason to suppose that the only way one system may become autonomous is through genetic vulnerability.

At the cornerstone of Akiskal and McKinney's (1975) model of depression is the view that a number of different events, psychological, social, physiological and biochemical, can all influence the biological integrity of the reinforcement

structures in the diencephalon. Such is the case, they argued, because three neurophysiological systems, the stress (hypothalamopituitary) system, the arousal (reticular activating) system and the psychomotor (pyramidal-extra-pyramidal) system "are intimately related to the reinforcement (medial forebrain bundle (M. F. B.) and periventricular system (P. V. S.)) mechanisms". Diagrammatically Akiskal and McKinney (op. cit.) show the relationship between these systems in the following way:



The disruption in the integrity of the diencephalic mechanisms of reinforcement occurs through a disturbance in the negative feedback mechanisms which maintain these systems in homeostasis. Different events act on different neurophysiological systems changing the relationship of the affected system within the integrated complex. This change produces additional changes in the other systems, culminating in a disruption of the biochemical pathways of the reinforcement system. Thus it is the change in the homeostatic feedback mechanisms between the arousal, psychomotor and stress systems that induce disturbance in the activity of the reinforcement system. Akiskal and McKinney (1975) outline the dynamics of this model in the following way:

"Melancholic and manic behaviours, according to our scheme, result from a failure in the homeostatic mechanisms that maintain these systems in negative feedback - when physiochemical alterations in these systems produce increasing levels of positive feedback in the reinforcement system. Stress or frustration beyond the coping ability of the individual - together with their psychic (anxiety, hopelessness) and neuroendocrine (increased cortisol and sodium retention) correlates - are expected to produce heightened arousal and could disrupt the functional integrity of the reinforcement system. The resultant decline in negative and psychomotor functions and the perception of oneself as losing control in an impending state of decomposition serve as novel sources of stress with additional increments in arousal and additional decrements in coping mechanisms. Thus a vicious cycle of more arousal, more hopelessness, and more evidence of purposeless psychomotor activity. In a system's scheme like this, the controversy whether altered catecholamine metabolism is a cause or effect of depression can be easily resolved. Lowering of norepinephrine in the reinforcement system would contribute to a functional impairment whether it is primary or secondary. In other words, norepinephrine depletion can be an effect that, in its own right, can then serve as a cause in the pathogenetic chains of events". (P. 299)

Akiskal and McKinney (1975) expand their model by arguing that apart from possible genetic factors, other non-biological factors, for example psychosocial stressors (adult object loss, and chronic frustration) and developmental predispositions (early object loss and learned helplessness) can increase the probability of a disruption in the negative feedback mechanism outlined above.

In this way these authors describe how particular events allied with specific vulnerabilities can combine to produce the chain of events that result in a depressive illness.

IX. LIMITATIONS OF AKISKAL AND McKINNEY'S MODEL OF DEPRESSION

Akiskal and McKinney's (1973, 1975) model of depression provides a useful framework which succeeds in integrating a wealth of data. However, there are certain limitations to this model. Firstly the thumbnail sketch of Beck's model of depression is grossly inadequate for a multi-component theory of depression which purports to give equal weight to biological and psychological factors in depression. Such brevity has resulted in the authors arguing that:

> "According to this viewpoint, hopelessness and helplessness represent the central features of human depression and reflect a peculiar "cognitive triad" of negative conception of the self, negative interpretations of one's experience and a negative view of the future." (P. 292)

Although Beck (1967) does point out that depressives have a negative view of the future (hopelessness), there are other more central factors in the theory, discussion of which will be given later. Moreover, as Beck and his colleagues have found (Minkoff et al., 1973), hopelessness is significantly and positively correlated with suicidal intent regardless of diagnosis.

Secondly, the concept of helplessness which these authors derive from a theory developed by Seligman (1975) is given an indirect influence on biochemical systems. Although the dynamics of the learned helplessness theory will be

discussed later, it can be pointed out here that rather than acting as a novel source of stress or psychic anxiety, a perception of helplessness, according to the theory, produces a direct influence on mood. Indeed it is the perception of being helpless in the face of trauma that changes a potentially anxious situation into a depression inducing one. In addition, Weiss et al. (1970) and Weiss et al. (1976) have shown that the induction of learned helplessness in animals produces central and direct changes in catecholamine metabolism. These changes do not seem specific to any particular brain area but seem to occur throughout the noradrenergic pathways. So replicable are these changes that these metabolic events themselves have been suggested as the responsible factor for the learning deficits that follow exposure to inescapable trauma.

The third weakness in this model seems the most important, however. It will be recalled that Akiskal and McKinney (1975) argue that the whole sequence of events leading to depression can commence with 'stress or frustration beyond the coping ability of the organism". Indeed the role of coping mechanisms appears more than once in their integrated model. However, Akiskal and McKinney offer no discussion of the factors that determine coping mechanisms although a number of questions arise, such as why do some individuals cope with certain events (such as the break-up of a marriage) but others do not? What are the determinants of adaptive coping behaviour? And indeed why do some individuals find some events extremely stressful where others do not? None of the questions are considered by Akiskal and McKinney and yet if coping mechanisms and stress evaluations are the factors that can ignite the spiral of events outlined by Akiskal and McKinney, then these are central concerns. It is on these issues that cognitive psychology has most to offer.

X. THE ROLE OF COPING MECHANISMS IN A MULTI-COMPONENT ANALYSIS OF DEPRESSION

The concepts of stress and coping are difficult terms to define. As Whybrow and Parlatore (1973) point out, from the second world war onwards, research on stress has shown it to be as much a function of the individual as of the environment. Lazarus (1966) has offered some interesting insights into social and cultural determinants of stress. Lazarus (op. cit.) points out that the individual's behavioural and emotional reactions to a noxious event depends as much upon his evaluation and perception of the event, together with his perceived ability for reducing the threat, as it does upon the event itself.

In order to integrate the cognitive processes that determine and influence stress perception and coping evaluation into a multi-component framework of depression, some clarity should be given as to what biological processes coping mechanisms may interact with. More especially it needs to be shown how cognitive events which interfere with adaptive coping behaviour can disturb those biological systems which have been considered central to an understanding of depression. A useful starting point then is to consider some of the biological theories of depression and later to try and show how a detailed consideration of coping mechanisms can shed light on the question of how these biological systems become disturbed.

The most commonly known and important biological theories of depression are concerned with the functioning of the central neurotransmitters, noradrenaline, dopamine, 5-hydroxytryptamine and acetylcholine. It is the role of these neurotransmitters (with special emphasis on noradrenaline and acetylcholine) which will be discussed here. A review of other biological aspects of depression can be found in Whybrow and Mendels (1969) and Lader (1975).

XI. THE BIOGENIC AMINE HYPOTHESIS OF DEPRESSION

During the late 1950s and early 1960s there was a gradual accumulation of evidence which suggested a link between mood disorder (mania and depression) and the availability of the catecholamines, noradrenaline (NA) and dopamine (DA) and the indole-amine 5-Hydroxytryptamine (5-HT) in the central nervous system. The evidence was largely indirect and rested on observations from the use of the drugs reserpine and iproniazid (Schildkraut, 1965, Bunney and Davis, 1965, Coppen, 1967 and Iverson and Iverson, 1975 provide excellent reviews of this early work). Reserpine used in the treatment of hypertension was reported to produce a disorder resembling endogenous depression in 10 - 15% of patients so treated. Reserpine depletes the brain of NA, DA and 5-HT. Conversely iproniazid, used in the treatment of tuberculosis, was reported to have mood elevating properties. This drug increases the availability of NA, DA and 5-HT in the central nervous system (C.N.S.). The original catecholamine hypothesis of depression thus postulated that depression was associated with central catecholamine depletion and mania with central catecholamine elevation. More recently Prange et al. (1974) have suggested that a reduction in 5-HT may be common to both depression and mania, with the catecholamines determining the type of mood disorder.

The development of the anti-depressant drugs both reflected and helped develop this basic hypothesis. The two major groups of anti-depressant drugs, the monoamine oxidase inhibitors (MAOIs) and the tricyclic compounds, both change catecholamine and indole-amine synaptic mechanisms (Coppen, 1967; Iverson and Iverson, 1975). Theoretically, however, a rise in the storage level of these amines as produced by MAOI treatment does not necessarily

imply that in response to nerve activity more transmitter is automatically released, though this remains a plausible explanation (Iverson and Iverson, 1975).

XII. BIOGENIC AMINES AND REINFORCEMENT

With the development of histochemical fluorescence, adapted for use in the C.N.S. by Dahlström and Fuxe (1964), it became possible to map these various neurotransmitter pathways in the brain. A major observation from the use of this technique showed that the reward areas (Olds and Milner, 1954), especially in the region of the lateral hypothalamus, coincided with the major bulk of noradrenaline tracts. The 'reward areas' received their name following the finding that direct electrical stimulation of various parts of the limbic system and extrapyramidal system were found to be innately rewarding (Olds and Milner, 1954; Milner, 1971), the best result being obtained from the medial forebrain bundle (M.F.B.). In contrast to these areas is the periventricular system which appears to be the anatomical substrate of punishment.

Over the past twenty years, research has demonstrated that the functional integrity of the reward system depends on appropriate noradrenergic and possibly dopaminergic activity. A number of reviews of this work already exist and need not be further discussed here (Synder, 1975; Stein, 1968; Stein et al., 1972; Lipton, 1972; Crow, 1973).

XIII. THE ACETYLCHOLINE HYPOTHESIS OF DEPRESSION

The monoamines have enjoyed a monopoly of the research conducted in neurotransmitter changes in depression. Recently, however, some inroads

into this monopoly have been made by Janowsky et al. (1972). They proposed that under normal circumstances an equilibrium is maintained between the activity of acetylcholine systems and noradrenergic systems. In depression there is a disruption in this equilibrium in favour of cholinergic dominance, while in mania the disruption shifts to a noradrenergic dominance. This hypothesis derives some support from experimental evidence which has demonstrated an antagonistic relationship between NA and acetylcholine (Ach) in certain brain areas (Glisson et al., 1972, 1974; Karczman, 1974). Indeed some neurochemical research has attempted to map antagonistic interactions between neurotransmitter systems in some brain areas (e.g. Perez De La Mora and Fuxe, 1977). Much of this work has been reviewed elsewhere (Roth and Bunney, 1976; Karczman, 1974, 1976) and cannot be further taken up here. Suffice it to say here that there is a growing number of studies which provide evidence for the view that neurotransmitter activity depends on a complex and antagonistic process of interaction, and that different areas of the brain function according to the outcome of these complex interactions. Such data adds theoretical feasibility to Janowsky's et al. (1972) view of depression and mania.

Clinically, Janowsky et al. (1973) have demonstrated that manic symptoms could be alleviated by the use of drugs, e.g. physostigmine, whose main mode of action is to facilitate acetylcholine activity. With regard to depression, Gershon and Shaw (1961) reported an increased incidence of depression in patients who had been poisoned by organophosphate cholinesterase inhibitor insecticides. Further evidence of the role of acetylcholine in depression comes from observations that many of the anti-Parkinsonian agents, which are used for their atropine-like effects, have mood elevating properties (Janowsky et al., 1972; Loudon, 1977).

In the same vein, it is also interesting to note an observation by Hollister et al. (1964) who used atropine as a placebo control in a comparison study of amitriptyline and imipramine. They reported that although each drug was superior to the control treatment in some respect, most differences were small and inconsistent. In a later study Hollister et al. (1966) found that a group of patients given a period of treatment with atropine as a preliminary screening procedure resulted in the loss of half the potential sample. Clearly the use of atropine as a placebo can be criticised (see Becker, P.49-50). But the important issue here is that from the point of view of Janowsky's et al. hypothesis these observations with the use of atropine in depression adds some additional support to their theory.

XIV. LIMITATIONS OF NEUROCHEMICAL THEORIES OF DEPRESSION

While the advent of the anti-depressant drugs have constituted a significant advance in the treatment of depression, neurochemical theories of depression are significantly limited as explanations of depression in their own right. In the first instance human subjects receiving biogenic amine depleting drugs have shown only a slight tendency to develop true depressive disorders. A good review of both animal and human studies involving reserpine (which depletes the brain of NA, DA and 5-HT) and alpha-methyl-para-tryosine (which depletes the brain of NA and DA but not 5-HT) and parachloro phenylalanine (which depletes the brain of 5-HT but not NA or DA) has been presented by Mendels and Frazer, (1974). They concluded that there was little evidence to support the claim that biogenic amine depletion is a primary causal event in depression. They did, however, propose the view that depletion of the biogenic amines at specific sites

in the brain may act as a secondary factor in depression, a view also taken by Akiskal and McKinney (1975).

More recent work has shown that complete destruction of NA pathways in newborn rats by subcutaneous injection of 6-hydroxy-dopamine, produces no significant behavioural deficits (as measured on two-way active avoidance, activity, reactivity and discrimination learning) in adult life, compared to normal controls (Isaacson et al., 1977).

The evidence for changes in acetylcholine activity as a primary causal event in depression is also weak. Davis et al. (1976) examined affective and cognitive changes in a group of normal subjects given an intravenous administration of physostigmine. They reported that there was no significant difference in affect between this group and a saline control group. However, two subjects who received physostigmine did report depressed mood: the first, a woman with a past history of premenstrual depression and the second, a subject who admitted to marihuana use. However physostigmine does produce side effects, sometimes referred to as the physostigmine syndrome, which include lethargy, loss of energy and irritability, all of which are commonly observed in depression. However, physostigmine does not in general seem to exert any direct influence on mood (Weiss et al., 1976; Davis et al., 1976). Indeed, as Mendels and Frazer point out, although direct alterations (via drugs) in neurotransmitter systems can produce symptoms which resemble those found in depression, true depressions following exposure to such drugs are rare. The more the consequent symptoms resemble true depression, the more likely is the individual to have a history of psychiatric disturbance.

These considerations argue strongly in favour of there being some other significant factors, apart from changes in internal neurochemistry, which need to be present in the aetiology of depression. These other factors may be responsible for inducing neurochemical changes. As Schidkraut (1965) was aware some thirteen years ago, it is in the psychological functioning of the individual that these 'other factors' may exist. He points out:

> "Such hypothetical changes in catecholamine metabolism may be conceptualised to be part of the pathophysiology of depression, although not necessarily of primary aetiological significance. Since the importance of psychological factors in the aetiology of at least some depressions is well established, investigations of the possible effects of psychological factors on catecholamine metabolism seems a most promising area for future research. (P. 517)

In fact, the investigation of such psychological-biochemical interactions as may exist in depression has proved extremely difficult (Schidkraut and Kety, 1967). Nevertheless it is this area of investigation that presents the greatest challenge for psychiatry and promises to offer the most insight into depression. The next few sections attempt to take up this challenge in the light of new experimental work, and to examine some of the areas of psychology that might aid this search.

XV. NEUROCHEMICAL CHANGES AS SECONDARY EVENTS

The research on the biochemical correlates of depression seems to present the psychologist with two possible biological events that need to be explained, (1) a change in catecholamine metabolism such that noradrenergic activity decreases, (2) a change in acetylcholine metabolism such that acetylcholine

activity increases. In fact, although these may appear as separate events, because of the way neurotransmitter systems seem organised in the C.N.S., i.e. in terms of mutual antagonism, they may share common aetiologies. In other words, if it is shown that certain psychological events increase acetylcholine activity, then it is possible that the same events may also produce reductions in noradrenergic activity. Indeed, independent research examining (1) the role of acetylcholine in active avoidance and (2) the role of noradrenergic activity in active avoidance, strongly supports this proposition.

It is important to examine briefly some of this research because it provides a necessary link through which maladaptive coping behaviour can be shown to exert a disturbing influence on internal neurochemistry. Moreover, since it is the relationship between stress evaluation, coping mechanisms and the neurochemical correlates of depression that is a major aim here, this work will furnish valuable insights.

XVI. ACETYLCHOLINE ACTIVITY AND THE HIPPOCAMPUS

The role of acetylcholine in active avoidance learning has been of interest to psychologists for two main reasons:

(1) Neurochemically, the septum and hippocampal areas appear to be a focal point of the limbic acetylcholine system (Lewis and Shute, 1967). Moreover, in the hippocampus itself, acetylcholine appears to be the activating or excitatory neurotransmitter. However, noradrenaline terminals are also present (though more diffused) in hippocampus and these appear to inhibit hippocampal activity (Strom-Matheisen and Fronnum, 1972). Thus the functioning of the hippocampus may well depend on the acetylcholine

noradrenaline ratio. Research has shown that drugs which block acetylcholine activity (e.g. atrophine) produce similar behavioural deficits as lesions to septum and hippocampal areas (Carlton, 1969; Hington and Aprison, 1976). (2) Behaviourally, the septum and hippocampus appear to be the most important brain areas regulating inhibitory behaviour. Lesions to these areas result in an animal that is deficient in the acquisition of passive avoidance, but facilitated in the acquisition of active avoidance. In general, destruction of parts of these areas can result in an animal that has difficulty in withholding or inhibiting certain behavioural responses (Milner, 1971; Carlton, 1969; Gray, 1971; Altman et al., 1973). Gray has referred to these areas of the brain as the septo-hippocampal stop mechanism which is responsible for bringing punished behaviour and unrewarded behaviour to a halt. Altman et al. (op. cit.) argues that:

> ". . . the hippocampus may be thought of as a focal point of a septal and a limbic loop exercising facilitatory influence on a central nervous braking system." (P. 578)

These authors argue that the concept of braking rather than inhibition is a better term to describe hippocampal function, because the term inhibition implies an inactivated state, whereas the term braking implies an unabated state of arousal which does not produce any effective behaviour as long as the powerful brakes (controlled by the hippocampus) are applied. Drug studies (Carlton, 1969; Hington and Aprison, 1976) have shown the importance of acetylcholine in the operation of this hippocampal braking system. Moreover, rats that have been bred to have high levels of brain acetylcholine have

been found to be significantly more inhibited in their behaviour, and show deficits in the acquisition of active avoidance learning compared to controls (Burxton et al., 1975). Such results show that animals with high levels of brain acetylcholine have a greater tendency to inhibit or 'brake' certain behavioural responses.

In view of these data, it can be speculatively argued that psychological events which produce an aroused but inhibited animal may activate the hippocampal braking system which is mediated by an increase in acetylcholine activity. If such a view is valid, it leads to the question of what psychological events might produce an aroused but behaviourally inhibited animal. It is in the area of active avoidance learning that some of these psychological factors can be found.

XVII. THE HIPPOCAMPUS, ACETYLCHOLINE ACTIVITY, AND COPING BEHAVIOUR

The experimental manipulation which most commonly results in an aroused but inhibited animal is active avoidance learning. The reason for this lies very much in the demand characteristics of the experiment. Carlton (1969) explains why:

"Consider the kind of experiment in which the experimenter arranges things so that the animal can avoid shock. But it is a rare rat that knows how to avoid when the experiment starts; accordingly we may find substantial shock induced suppression of behaviour, suppression of that very behaviour that we want the animal to emit. From the rat's point of view, there is no way to cope with shock until the appropriate means of coping is learned. And the suppression consequent to the lack of coping behaviour may preclude learning how to cope. Accordingly, we expect many animals to fail to learn in many kinds of avoidance

situations, as in fact they do. Those situations in which poor learning (or coping) occurs are those that reduce the chance of the animal's "catching on" to the programme contingency." (P. 308-309).

Carlton (op. cit.) argues that the suppression (or inhibition) of behaviour is due to some inhibitory hippocampal mechanism which 'switches in' in the presence of a traumatic event. However, coping behaviour (active avoidance learning) can be facilitated by giving the animal acetylcholine blocking drugs, suggesting that it is acetylcholine which is responsible for behavioural inhibition. The proposition that follows from this is that situations which expose an animal to an aversive event with reduced means of coping should result in an aroused but inhibited animal; that is an animal deficient in learning and executing the required avoidance response. This inhibition in behaviour is mediated by an increase in acetylcholine activity in the septo-hippocampal braking system. Preventing an animal from acquiring adaptive coping behaviour when exposed to a stressful event should increase the probability of activating inhibitory centres (the septo-hippocampal system) resulting in an aroused but passive animal.

XVIII. LEARNED HELPLESSNESS

The observation that rats appear to become helpless or give up when exposed to inescapable trauma was first reported by Mowrer and Viek (1948). In view of the approach that has been taken here, this helplessness in inescapable traumatic situations could be explained in terms of the activation of the hippocampal braking system, i.e. in the absence of adaptive coping behaviour, powerful brakes are applied to behaviour, such that the animal reduces its own inputs (Gray, 1971).

In 1967 Seligman and Maier (1967) and Overmier and Seligman (1967) observed the phenomenon of helplessness in their active avoidance learning experiments. Although the original hypothesis is not clear, it seems that these researchers were examining the role of fear on avoidance conditioning. However, they found that their fear induction procedure, instead of producing invigorated avoidance learning, actually seemed to reduce responding. In their procedure dogs were placed in a Pavlovian harness and exposed to inescapable shocks, contingent on a warning signal. All responses during this session were ineffective in avoiding shock. Following this procedure the dogs were released from the harness and a normal active avoidance learning trial commenced. In this case, following the onset of the warning signal, the dogs were required to jump a hurdle to avoid the contingent shock. However, instead of learning the required avoidance response, many of the dogs simply howled and only slightly moved around the chamber. As the number of trials increased, the activity of these animals progressively decreased until they were generally immobile throughout most of the trials.

There is an immediate tendency to interpret these results in terms of the septo-hippocampal (acetylcholine) braking system. However, this is not the mode of explanation favoured by Seligman and his colleagues. They explained their findings as follows: animals exposed to inescapable,uncontrollable shock learn that their responses are independent of outcome. They learn that responding is pointless. This learning Seligman calls Learned Helplessness. This belief (Seligman, 1975, allows his animals beliefs) carries over into the active avoidance task, resulting in an animal who makes little attempt to learn the

programmed contingency. The phenomenon of learned helplessness can, it has been argued, be produced in a variety of species (Seligman et al., 1971) and underlies certain types of human psychopathology (Seligman, 1974, 1975). The area of psychopathology that has received most attention from Seligman is depression. Seligman (1974, 1975) argues that like the dogs in the pavlovian harness the depression-prone individual believes that his responses are independent of outcomes. He believes he is helpless in controlling his reinforcements. This belief in helplessness has the associated effect of depression. However, as in many animal models of depression, phylogenetic discontinuities make animal observations extremely tenuous as explanations of human behaviour. Moreover there is some reason to doubt Seligman's explanation of the behaviour of his dogs. As Carlton (1969) has pointed out, behavioural inhibition is sometimes the first response observed in active avoidance learning. On occasions this tendency to suppress or inhibit ongoing behaviour can be strong enough to interfere completely with appropriate avoidance learning. For Seligman to be correct, then presumably all failures in active avoidance learning which result in a passive animal are due to learned helplessness and not some neurochemical correlate. This would include those animals which appear helpless from the very first trial.

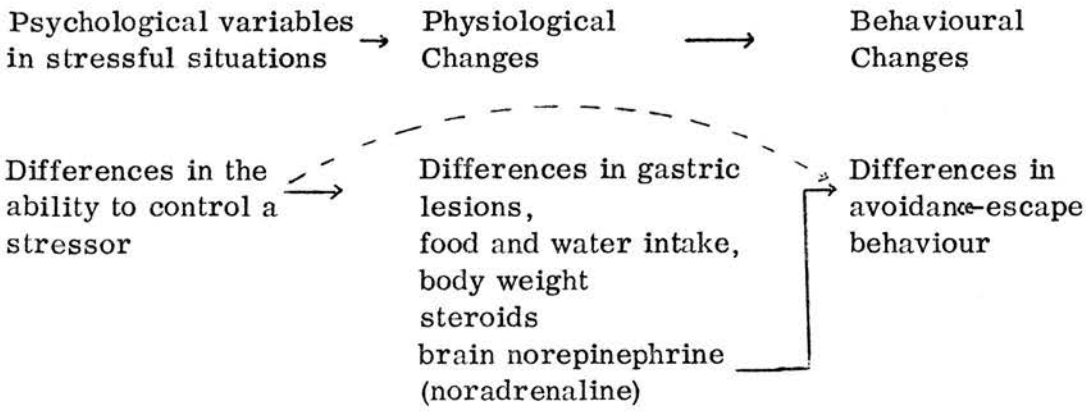
However, Seligman's work has generated some interesting work. It will be recalled that earlier it was argued that reduction in noradrenergic activity is considered by some an important neurochemical correlate of depression. Thus explanations of depression in terms of maladaptive coping behaviour should attempt to account for this neurochemical event. It has been neurochemical work stimulated

by Seligman's observations that have furnished the best insights into the role of coping behaviour on neurochemistry, especially catecholamine metabolism.

XIX. THE ROLE OF NORADRENALINE ON COPING BEHAVIOUR

One group of workers who have not accepted Seligman's learning explanation of learned helplessness is Weiss and his colleagues in New York. Weiss et al. (1976) reviewed a series of twelve experiments conducted by them over the last eight years. What these experiments purport to show is that the phenomenon of learned helplessness is mediated by physiological changes.

In the original learned helplessness experiments, Overmier and Seligman (1967) reported that the learning deficits resulting from exposure to inescapable shock dissipated over 48 hours or so. Miller and Weiss (1969) have argued that this dissipation effect is not at all common in most learning situations. Such an effect, they argued, suggests the interpretation that physiological variables mediate learned helplessness. Weiss et al. (1976) have proposed the following model to explain the development of learned helplessness.



This model proposed that the failure to learn adaptive coping behaviour

following exposure to inescapable shock is due to biochemical disruption which impedes certain adaptive responses. In an early experiment, Weiss et al. (1970) found that animals who received inescapable shock had lower levels of brain noradrenaline than animals who received exactly the same shocks while performing an avoidance escape response. A number of subsequent studies (Weiss et al., 1976) demonstrated this to be a consistent result and these researchers went on to investigate the neurochemical mechanisms that might underly such changes. They also demonstrated that the administration of an MAOI (in this case pargyline) could protect against the development of learned helplessness (see also Weiss et al., 1975).

The hypothesis proposed by Weiss and his colleagues is that brain noradrenaline is important for the execution of certain motor activities. Stress beyond the coping ability of the animal produces central changes in catecholamine metabolism, notably noradrenaline depletion. This depletion in central noradrenaline produces a deficiency in the ability of the animal to perform the required motor responses necessary for active avoidance.

Whether the motor-activation deficit hypothesis as proposed by Weiss et al. (op. cit.) is accepted as an explanation of learned helplessness is something of a secondary issue. The most important point here is that Weiss and his colleagues have demonstrated that severely limiting an animal's ability to cope with stress results in substantial metabolic changes.

Thus manipulations of active avoidance learning procedures seem to produce two types of data:

(1) A reduced ability to cope with stress can result in the animal inhibiting

its behaviour. This inhibition of behaviour may reflect the activation of the hippocampal braking system which is mediated by acetylcholine. Acetylcholine blocking drugs will facilitate the acquisition of adaptive coping behaviour. Moreover, Seligman (1975) reports a study in which atropine injected directly into the septum of cats, dissipated learning deficits produced by inescapable shock.

(2) A reduced ability to cope with stress results in a disruption in catecholamine metabolism. Such a disruption may produce an animal unable to perform the necessary motor responses required for adaptive responding because of a depletion of noradrenaline. A change in catecholamine metabolism will effect the functioning of areas of the brain in which noradrenaline acts as the excitatory transmitter. In accordance with early neurochemical theories of depression, the most important brain area is the medial forebrain bundle.

As mentioned earlier, because of the way neurotransmitter systems appear to be organised, these two neurochemical changes may occur to the same set of psychological events. Indeed the evidence of neurochemical changes in active avoidance learning supports this view. Thus Janowsky et al. 's (1972) view of depression would seem to have greater validity than single neurochemical system theories. As Weiss et al. (1976) and Reiser (1976) point out, the catecholamines constitute one physiological system amongst many. Uncontrollable aversive events may well be expected to produce changes in other physiological systems such as the neuroendocrine systems. The interaction between the various biological systems in response to 'stress beyond one's coping ability' is an area

for future research.

XX. IMPLICATIONS OF THE NEUROCHEMICAL MEDIATORS OF COPING BEHAVIOUR

In regard to a multi-component and multi-disciplinary approach to depression, the data that has been discussed in the last few sections carry some important implications. As Reiser (1976) in discussing Weiss et al. (1976) points out:

> "These data imply that if you can cope with stress or loss adequately, you can avoid physiologic turbulence and consequences and be less susceptible to successful invasion by pathogenic factors, be they infectious, neoplastic, climatic, or whatever." (P. 178)

Moreover with regard to depression, Klerman (1974) has argued:

> "It seems that the most significant factor accounting for the occurrence of depression is the predisposition or vulnerability of the individual. Thus we have to investigate why certain individuals are able to cope with stressful events while other individuals fail in their coping efforts or even develop clinical symptoms in the absence of apparent life stresses." (P. 142)

Although Klerman stresses different predisposing factors for the failure of certain coping behaviours than those expressed here, the idea that the determinants of coping behaviour are one of the most important issues in depression is a view he strongly advances. In this regard the evidence presented in the last few sections have highlighted two main issues central to such a viewpoint.

(1) The role of coping behaviour in depression requires further clarification. In this regard Akiskal and McKinney's (1975) model of depression must be seen as deficient, in that it fails to examine adequately the determinants of

coping behaviour.

(2) A consideration of coping behaviour based on new evidence demonstrates that the failure to cope adaptively with stress can produce many physiological changes, including those considered to occur in depression. The most important observation being that those neurochemical changes believed to occur in depression can be externally (the difficulty of an avoidance contingency) determined. Thus the psychological determinants of stress evaluation and coping behaviour seem essential issues in a multi-component theory of depression.

XXI. SUMMARY OF THE ARGUMENT SO FAR

The dis cussion so far has tried to develop a logical progression in the examination of the data pertaining to depression. First, the internal biological correlates of depression were examined. Second, possible external determinants for these neurochemical changes were discussed. The most salient determinant of these changes seems to be whether or not the individual can cope with stress. In the next few sections the psychological determinants of coping will be examined. Before discussing these psychological factors, a summary of the complex problems covered so far seems helpful.

(1) The catecholamine hypothesis maintains that there is a link between mood disorders and the availability of the monoamines (NA, DA and 5-HT) in the C.N.S.

(2) An alternative neurochemical theory of depression has argued that it is the relationship between the activity of noradrenaline (and possibly the other monoamines) and acetylcholine that determines mood disorder.

(3) Drug studies have tended to show that purely neurochemical theories of depression are inadequate as primary aetiological explanations of depression. However, neurochemical disturbances may play an important secondary role, maintaining certain depressions once they have started, and possibly producing certain symptoms.

(4) Research using active avoidance learning procedures has shown that interference in the acquisition of adaptive coping responses can lead to neurochemical changes which once activated can maintain maladaptive behaviour. Such research can account for both (a) changes in acetylcholine metabolism such that acetylcholine activity increases (the hippocampal braking system activation hypothesis), (b) changes in catecholamine metabolism such that noradrenergic activity decreases (the motor activation deficit hypothesis). In view of the way the neurotransmitter systems seem organised, (i.e. in terms of mutual antagonism), these data seem consistent. Thus the idea of considering the acetylcholine/noradrenaline ratio in hippocampus and other brain areas seems a useful neurochemical procedure.

(5) Since the proposal of a coping behaviour ⟶ neurochemical change ⟶ behavioural change equation seems valid, the analysis of the role of psychological factors (e.g. appraisal, expectancies and beliefs) as determinants of coping behaviour require consideration. The proposal that follows from these data is that the depression-prone individual may significantly limit his own coping behaviour due to maladaptive cognitive structures and thinking patterns. Such internally generated constraints on the acquisition of adaptive coping behaviour may result in a significant

vulnerability to neurochemical disturbance and depression.

XXII. STRESS EVALUATION, COPING AND COGNITION

Cognitive factors place considerable limits on the usefulness of animal models of depression. Animals are limited in their ability to utilise many cognitive processes in coping and stress evaluation which humans obviously do. As Foulds (1976) eloquently points out: "the rat we may presume fears only what will be done to it not what it will do". Moreover, as Weiss et al. (1976) argue, Seligman (1975) considers that learned helplessness and depression are dependent on the 'perception' of a loss of control. This emphasis makes the hypothesis almost untestable in non-verbal animals. In any event the extent to which animals are concerned about their self-esteem or inadequacies in their own behaviour is undoubtedly limited. As a result, progress in the investigation of the important psychological determinants of coping behaviour and stress evaluation can only be advanced with human experimentation.

It is the cognitive evaluative elements in the stress ⟶ coping situation that presents the fundamental extension to the coping behaviour ⟶ neurochemical change ⟶ behavioural change equation derived from animal experimentation. A useful framework for investigating this extension has been provided by Lazarus and his colleagues at Berkeley. In 1966 Lazarus argued that the psychological mediators in the stress evaluation ⟶ coping situation operated as a sequence of appraisals. These appraisals consist of primary appraisal, secondary appraisal and reappraisal. It is the first two of these and their relationship to depression that is the main concern here.

XXIII. PRIMARY APPRAISAL AND DEPRESSION

Primary appraisal consists of the cognitive searching operations that produce an evaluation of the meaningfulness of an event. In general terms, this implies an appraisal operation that answers the question, how good, bad or irrelevant is this event. Such appraisal can lead to an event being evaluated as either (a) irrelevant, (b) benign - positive, or (c) stressful. It is the factors that can lead to an event being appraised as stressful that are the main concern here. Lazarus and Launier (1978) have argued that a stressful appraisal may be of three types: (1) Harm - Loss, (2) Threat, (3) Challenge. It is the first of these that has most relevance to depression. Harm - Loss implies an evaluation that some damage has been done to self or social esteem and future goals and reinforcements.

The validity of examining the various evaluations of harm - loss arises by virtue of the fact that Beck (1963, 1967, 1974, 1976) has presented a theory of emotional disorders, which proposes that the depression-prone individual makes systematic errors in his/her evaluations of harm - loss. Beck (1974, 1976) argues that it is the appraisal of loss that is 'the clue' to understanding depression. According to Beck the individual appraises his world and future in terms of significant losses (e.g. friends and support) and he appraises himself as a loser. In this context harm - loss evaluations refer to loss of self-esteem, of friends, support, money, employment, prestige, etc. Beck argues that the depressed individual has an exaggerated appraisal of loss because of systematic errors and distortions that exist in the individual's thinking processes. These include:

1. Selective abstraction — focussing on a detail out of context, "my friend ignored me thus he does not like me" (his friend may have been busy at the time)

2.	Arbitrary inference	drawing a negative conclusion in the absence of supporting evidence. If the therapist is late for his consultation the patient infers that the therapist does not like him and is purposely trying to avoid him.
3.	Overgeneralisation	making gross generalisations on the basis of a single incident. Confronting a minor isolated failure produces the appraisal that everything is going wrong.
4.	Magnification and minimisation	belittling the good events that happen and exaggerating the bad events; what Ellis calls catastrophising
5.	Personalisation	relating negative events to self when there is no basis for this. At the delusional level the patient may believe he is the cause of unrelated world disasters.

A. Lazarus (1971) has argued that there are two other systematic errors made by the depression-prone individual; oversocialisation which is a failure to recognise the arbitrariness of social norms, or failing to learn to compromise, and dichotomous reasoning, thinking in terms of polar opposites, right/wrong, good/bad, etc.

These systematic errors in the appraisal and evaluation of the good, bad or irrelevant characteristics of the environment, result in the over-appraisal of stress (in terms of harm-loss). Beck (op. cit.) argues that these appraisals go beyond the limits offered by a realistic interpretation of the environment and in this sense are pathological. A further examination of the psychological

antecedents that produce these systematic errors will be taken up in the next chapter. Suffice it to say here that all the distorted thinking patterns outlined above can loosely be seen as producing a negative primary appraisal in terms of harm-loss.

The observations of Beck (op. cit.) have significant implications for the stress ⟶ coping relationship. They demonstrate that the first element of this relationship - stress evaluation - may be distorted in the depression-prone individual. Stress beyond the coping ability of the individual may partially occur because of incorrect stress appraisal, which magnifies the aversity of the environment beyond that which actually exists.

Some indirect experimental evidence may support this view. For example, Lewinsohn et al. (1973) found that depressed students were more responsive (as measured by GSR) to an aversive stimulus than non-depressed controls.

Unfortunately direct experimental investigations bearing on the issue of primary appraisal in depression are scant. However, the above brief discussion suggests that it is an important area for future research.

XXIV. SECONDARY APPRAISAL AND DEPRESSION

Lazarus and Launier (in press) assert that ". . . the essential difference between secondary and primary appraisal lies in what is being evaluated. Secondary appraisal deals with coping resources and options."

These coping resources and options are the determinants of the ability of the individual to reduce the outcome of a (primary appraised) stressful event. As Mahoney (1974) points out:

". . . an individual's ability to function adaptively in both stressful and non-

stressful situations is dramatically influenced by his response repertoire. If he has acquired the component skills and integrative capacities to "cope", satisfactory adjustment is a much more likely occurrence." (P. 195)

Furthermore, as described earlier, coping with stressful events adequately can protect the individual from consequent physiological disturbance.

In depression, special emphasis has been placed on the individual's 'perception' of his own coping effectiveness in controlling stressful events. As mentioned earlier, Seligman (1974, 1975) proposes that it is a perception of response ineffectiveness which leads to a belief in helplessness and depression. He argues that:

"When a traumatic event first occurs, it causes a heightened state of emotionality that can loosely be called fear. This state continues until one of two things happens: if the subject learns that he can control the trauma, fear is reduced and may disappear altogether, or if the subject finally learns he cannot control the trauma, fear will decrease and be replaced with depression." (P. 53-54)

Although Seligman has popularised this view and some indirect support has been provided for it (Miller and Seligman, 1973, 1975, 1976; Gatchel et al., 1975; Miller et al., 1975, Klein and Seligman, 1976), a very similar conclusion was reached by Lazarus (1966) some time earlier. Lazarus wrote:

"In short, real hopelessness is here proposed as a condition of inaction in the face of threat. That is not to say there aren't other factors that may be involved, such as the suddenness of the condition or the social structure of the situation, but that a crucial antecedent of inaction and apathy is that there is no hope that an active response can be viable. The affect associated with such a condition of

hopelessness is depression. Fear and anger will occur only when the individual is not totally resigned to the hopelessness of his plight. The presence of these affects signifies the presence of action tendencies of flight or attack, however aborted, fleeting, or inhibited they may be." (P. 263)

These two similar expositions of causality in depression emphasise the importance of the appraisal of coping ability by the depression-prone individual. There is an indirect contention however, more obvious in Seligman's (1975) hypothesis than Lazarus's (1966), that depression either in cause and/or nature displaces other affects. This should be seen as an important weakness in these models. Naditch et al. (1975) have shown that depression and anxiety are positively correlated and Miller et al. (1975) found it is almost impossible to find depressed students who were not also anxious. Even behaviourally retarded depressed individuals often complain of racing and anxious thoughts. They may, for example, express deep anxieties about their future, illness, themselves or families, and also their perceived lack of coping abilities. Indeed the perceived deficits in their abilities together with a belief in personal inadequacies can be the source of much distress and anxiety. One view of retarded depression is that a contributing factor to this condition is a pathological preoccupation with these distressing thoughts (either real or imagined) to the exclusion of other environmental influences (Ashcroft, 1977). Moreover, as Akiskal and McKinney point out, depression constitutes a state of central hyperarousal rather than underarousal. So there is a real danger for theories of depression in postulating some sort of 'switching device' between one affect and another. While depression may be a probable diagnosis reached on the basis of observed behavioural,

vegetative, cognitive and mood symptoms, other affects such as anger, hostility and anxiety can be present to a significant degree (Klerman, 1974). The many cases of a 'mixed affective' diagnosis and the various nosologies that include sub-divisions of aggressive-depressed or anxious-depressed casts doubt on the notion that one affect can displace another (Solomon and Corbit, 1974) at least in pathological conditions such as depression.

Nevertheless the notions of helplessness or hopelessness which constitute a particular type of secondary appraisal have furnished useful insights into depression. These concepts have been either directly and/or indirectly involved in many of the psychological theories of depression (Beck, 1967, Seligman, 1975, Mandler, 1976, Melges and Bowlby, 1969, Bibring, 1953). The question of why certain individuals appraise their behaviour in such negative ways will be taken up in the next chapter.

XXV. INTERNAL CUES

In anxiety research some attention has been given to the interaction between the perception of internally generated cues (e.g. cues signalling peripheral arousal) and the appraisal of coping skills (Goldfried, 1971, Mahoney, 1974, Meichenbaum, 1977). The argument is that cues signalling peripheral arousal are interpreted by some individuals as evidence of an inability to cope with certain situations. Such appraisal leads to more arousal (with more internal cues) and a further devaluation in the individual's appraisal of his own coping skills. Depressed individuals may also be very attentive to changes in their bodily functioning. They may, for example, be aware of an increased level of fatigue, irritability, sleep disturbance, or loss of sexual interest. Statements

such as 'I feel I could cope with my life better if only I could sleep better', or 'If only I didn't feel so tired' attest to this situation. The extent to which these internal cues may undermine appraisal of effective coping behaviour seems a useful area for future research.

XXVI. REAPPRAISAL

Although Lazarus (1966) has used the terms primary and secondary appraisal to refer to different evaluative processes, they do not occur in any temporal order. For example, an appraised ability to be able to cope effectively with certain situations may greatly influence the primary appraisal of the event. Moreover, memories of past successes or failures in coping behaviour may also significantly influence how stressful an event is evaluated to be. This probability that memories of past coping attempts can influence the appraisal of stress has some bearing on depression. Lloyd and Lishman (1975) and Lishman (1972) reported an increase in the speed of recall of unpleasant memories which correlated with depth of depression. The implication of such work is that the depressed individual might devalue his own coping skills by distorting his recall of negative memories (possibly past failures) and thus increasing his appraisal of stress.

Lazarus and Averill (1972) have argued that it is reappraisal based on primary and secondary appraisal that determines emotional reactions and behaviour. These constructs of appraisal have provided a useful framework which enables a consideration of coping behaviour within a multi-component analysis. There are, of course, many other important factors that require consideration, most notably how and why depressed individuals distort primary and

secondary appraisal. These will be taken up in the next chapter. In the meantime, the discussion so far does allow for a useful integrated framework to be proposed.

XXVII. AN INTEGRATED MODEL OF DEPRESSION

In this section an attempt is made to bring together the many aspects of depression that have been discussed. A useful method for proceeding with such an analysis is to try to develop an integrated framework capable of demonstrating the interactions between the various factors and components that bear on depression.

At the beginning of the chapter a case was put forward outlining the possible benefits of examining depression in terms of a multi-component analysis. It was proposed that as in the study of normal emotion, depression requires such a multi-component integrated analysis to advance insights and point the road ahead more clearly. Having examined some of the neurochemical theories of depression, it was pointed out that such neurochemical changes as might occur in depression can be induced (in animals) by external manipulations of coping behaviour. In the last few sections the particularly human characteristics of stress evaluation and coping behaviour were examined. It was proposed that distortions in these psychological processes could significantly interfere with the acquisition of adaptive responding and bring about a degree of physiological vulnerability. Having briefly discussed these various areas, it is possible to integrate these physiological and psychological events within a multi-component framework. The dynamics and interactions of this model can be seen as a sequence of integrated events:

(1) The environment is evaluated (primary appraisal) as having significant stress (especially in terms of harm-loss) properties. A host of factors may be responsible for such appraisal. These will include internal factors, shaped by the past history of the individual (e.g. irrational beliefs and/or memories of past coping failures), and will also include the cognitive distortions outlined by Beck.

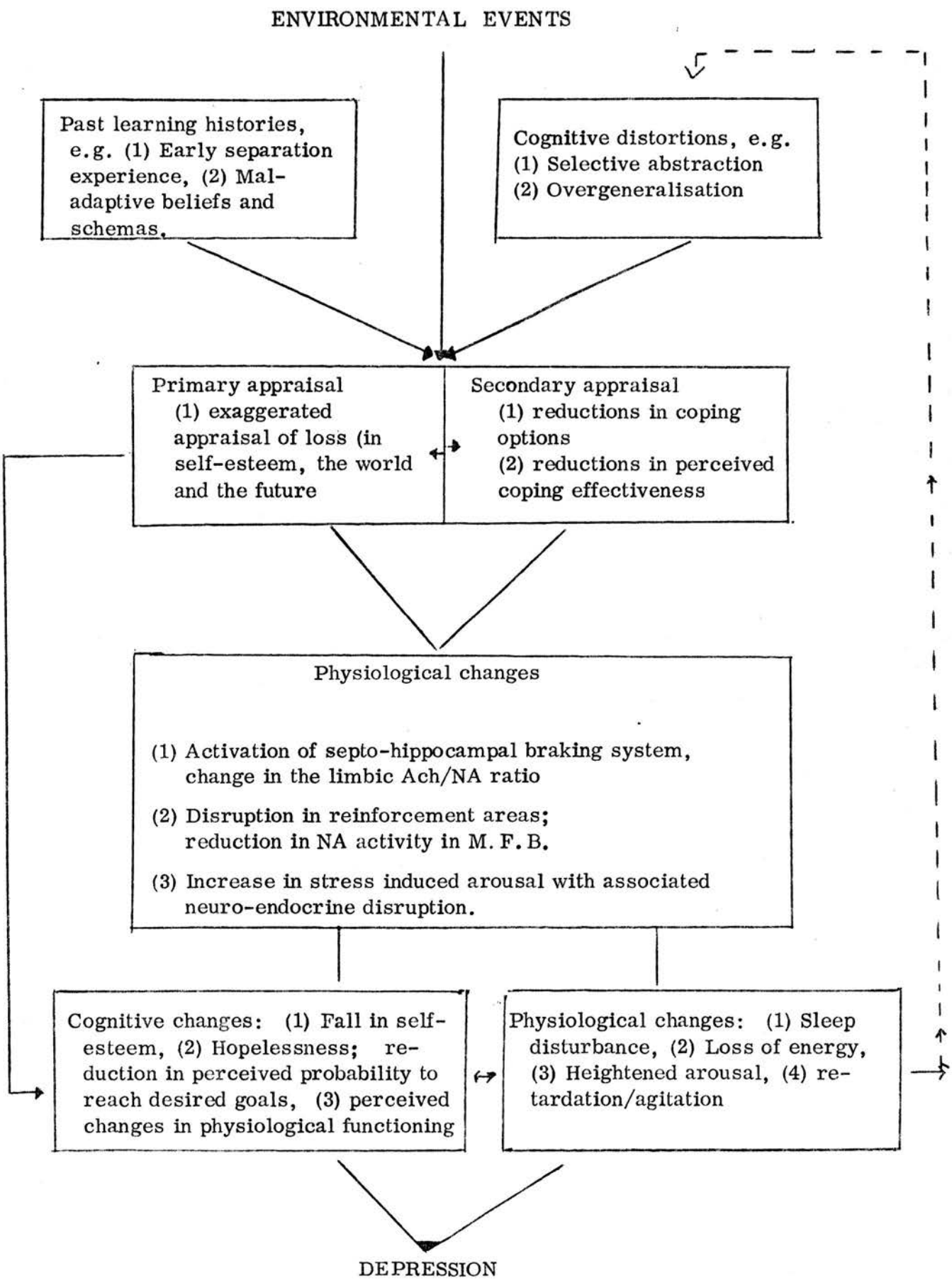
(2) From similar reasoning (e.g. past learning history and cognitive distortions), the depression-prone individual may inappropriately evaluate the effectiveness of his own coping behaviour in reducing or compensating for the loss.

(3) These distorted appraisals produce an exaggerated sense of loss. This appraisal of loss is centred on those evaluations which are concerned with the world, the self and the future. The greater the distortions in primary and secondary appraisal, the greater the fall in self esteem, and the more the individual believes that goals now and in the future are unobtainable.

(4) These negative cognitive appraisals act as internal constraints on the individual's ability to develop adaptive coping behaviour, in the face of an appraisal of stress (loss). The greater the appraisal of loss and the more the individual believes he is helpless to make up the loss, the greater is the individual's vulnerability to certain physiological disturbance.

(5) These cognitive constraints maintain the individual in a distressed but non-coping state. As a result there is an increase in the probability of certain neurochemical changes taking place. The probability that neuro-chemical changes may occur may depend on the degree of distortion, the

AN INTERACTIONAL MODEL OF DEPRESSION



severity of the appraised event or genetic factors, possibly it is a combination of all three.

(6) The failure to develop adaptive coping behaviour for the relief of this distress, can result in two neurochemical changes:

(a) The activation of the hippocampal braking system, signalling an increase in acetylcholine activity. Activation of this system will further reduce the probability of adaptive coping behaviour developing. In severe depression this system might account for the depth of retardation of some patients.

(b) A change in catecholamine metabolism resulting in a reduction in noradrenergic activity. Such reductions may produce important changes on other neurotransmitter systems (e.g. acetylcholine). However, in accordance with the neurochemical theories of depression, such changes as do occur in the catecholamine system, may exert the most disruptive effect within the medial forebrain bundle, which according to the theory reduces the sensitivity and responsiveness of the individual to positive reinforcement.

The combination of these two neurochemical events, together with other physiological changes, e.g. in the neuroendocrine system, can be expected to cause a change in bodily functioning, for example disruptions in sleep patterns or a lowering in the fatigue threshold. The more physiological disruption that occurs, the greater the change in bodily functioning, and the greater the severity of the 'physiological symptoms' (sleep loss, etc.). A further proposal here is that the greater the disruption in the neurochemical systems, the greater the probability of

of depression being of a psychotic magnitude. In addition, as the individual becomes aware of a decline in his functioning, this may act as further negative information leading to further distortions in primary and secondary appraisal.

It is impossible to say at what level or at what stage different component systems become activated. However it is unlikely that the events outlined above will occur in any set temporal order. It is probably more realistic to argue that the different events (psychological and physiological) overlap to a considerable degree. It may be that genetic factors determine the ease with which internal physiological processes can become disrupted. Thus, for some individuals very significant stressors and very negative appraisals may be required before any serious internal disruption occurs. For others the metabolic mechanisms may be more delicately balanced and comparatively minor stressors may produce serious internal disequilibrium.

If a coping approach to depression does prove useful, then research in the future will need to determine whether the degree of cognitive distortion is sufficient to explain the depth of depression or whether internal genetic factors are required for an adequate explanation. In any event, whether coping behaviour fails because internal physiological processes respond quickly to a stress, before the individual has a chance to learn and use adaptive coping behaviour, or whether it fails because of psychological factors, the fact remains, as Klerman (1974) pointed out, that the determinants of coping behaviour are a most important issue in the study of depression.

XXIX. CONCLUSION

This chapter has tried to avoid considering depression within a framework that hides certain philosophical inconsistencies. Instead it has drawn attention to the philosophical and theoretical benefits derivable from a pragmatic psychobiological approach to depression. An examination of Akiskal and McKinney's (1973, 1975) model of depression was presented as an excellent example of this type of approach to depression. However their model was considered deficient in its analysis of the role and determinants of coping behaviour. Consequently the latter part of this chapter has focussed specifically on this issue. It has attempted to show (1) the interaction between neurochemical events and maladaptive (inhibited) coping behaviour and (2) the psychological factors that may act as internal constraints on the acquisition of adaptive coping behaviour. Finally, an integrated model was presented to trace the sequence of events that might occur in depression. As to the temporal ordering of events, however, very little information can be given.

The development of multi-component analysis to psychopathology does seem to be pointing a road ahead. As Lange (1971) points out:

> "We will need to confine ourselves to measurable behaviors in all systems, and discover the laws that determine their interaction. The data suggests that we must deal with each behavior system in its own terms. Treatment programmes will have to be tailored to each behavior, in the light of what we know about its educability . . . In short, psychotherapy should be a vigorous multi-system training program, tailored to the unique behavioral topography presented by the patient. Fortunately, it is not infrequent that successes in

the control of one system seem to precipitate broad change throughout the individual's behavioral repertoire. It may come about through a new insightful conceptualization, or relief from aversive autonomic feedback, or the generation of a new, active coping behavior." (P. 109)

The role of psychology, especially cognitive psychology, has much to offer this approach. Although physiological treatments will continue to bring benefit to those burdened with emotional disorders, as Park and Imboden (1970) point out, drugs do not "re-educate, train or rehabilitate". In this regard, cognitive-behavioural therapy has a distinct advantage, especially in aiding the acquisition of adaptive coping behaviour with a consequent reduction in depressive vulnerability.

CHAPTER FOUR

COGNITIVE FACTORS IN DEPRESSION

I. INTRODUCTION

In the previous chapter a multi-component approach to depression was presented which proposed that certain of the neurochemical events believed to occur in depression may result from a failure to develop adaptive coping behaviour. It was argued that distortions in stress evaluation and the appraisal of coping effectiveness could act as internal constraints in the depression-prone individual which significantly limited the probability of successful adaptive coping behaviour developing. The reason that the depression-prone individual cognitively distorts primary and secondary appraisal processes and thus becomes vulnerable to a depressive illness was not directly examined. However, if the importance of coping behaviour in depression is accepted, then the answer to this question is of fundamental importance. This chapter examines some of the psychological theories of depression that may shed an important light on this issue.

II. BASIC ASSUMPTIONS IN THEORIES OF DEPRESSION

Psychiatry has tended to consider man's mental life capable of considerable dissection. As Zilboorg and Henry (1941) point out:

"What formal psychiatry chose to consider as emotions were not emotions at all but verbal and muscular manifestations. Emotions were considered a separate functional department of man's body structure; they were subject

independently to abnormal variations and were therefore thought to cause "affective psychoses" - disturbances of the so-called "affective field" or of "the emotional level", which could be considered apart from other faculties such as thinking and imagination. Such a departmentalisation of the human personality made it easy to give one's methods of psychological investigation the appearance of scientific work. Each part could be studied separately and "objectively", that is to say, only from the standpoint of what it looks like from the outside and not how it works from the inside". (P. 446)

The neurochemical theories of depression reflect this belief in a dichotomy between thinking and feeling. According to such theories, depression can be understood in terms of a biological disturbance in the mood regulating areas of the brain. However, the study of normal emotion (chapter three) has shown that a distinction between thinking processes and emotion processes is completely invalid. Emotional experience and behaviour, to a large degree, depend on thinking processes.

It is in direct opposition to the separate and autonomous view of emotion that Beck has proposed his cognitive theory of depression. As Beck (1974b) points out, it is often considered that:

". . . for normal subjects, the conceptualisation of a situation determines the affective state, but in psychopathology the affective state determines the cognition." (P. 128)

This complete reversal of cause and effect between normal and pathological affect is, in Beck's view, erroneous and constitutes a major source of confusion. The difference between normal and abnormal affective reaction, Beck (op. cit.)

argues:

". . . lies in the degree of correspondence between the conceptualisation and the veridical stimulus configuration. In psychopathological states perservative faulty conceptualisations lead to excessive or inappropriate affective disturbance.

The typical conceptualisations leading to specific affects appear to be the same in both "normal" and "abnormal" responses. In abnormal conditions, however, conceptualisations are determined to a greater extent by internal processes which distort the stimulus situation." (P. 128-129)

Thus Beck puts forward two related hypotheses: (1) normal and abnormal affective states exist on a continuum and (2) the factors (conceptualisations) that determine normal emotion also determine abnormal emotion. The degree of affective disturbance is determined by the degree of distorted conceptualisation. With regard to these two hypotheses, Beck's model of depression is consistent with other cognitive views of emotion. For example, Lazarus and Averill (1972) point out that:

"If cognitive processes mediate between a situation and the emotional response, then every emotion must be understood in terms of a particular kind of appraisal. That is, for any given emotion such as anxiety, anger, depression, joy, love, etc. the underlying appraisal must be specified." (P. 243)

Beck's cognitive theory of depression is entirely consistent with this approach to emotion. Moreover Beck specifies the appraisal responsible for depression as the appraisal of loss. Beck (1974b) argues that a lowering of mood occurs when the individual appraises or evaluates that a reduction in his domain has occurred:

"whatever the actual stimulus situation, the individual (a) must perceive an event as a loss, and (b) the object or attribute that has been lost or downgraded must have some positive value." (P. 131)

In the depressed individual the appraisal of loss pervades evaluations concerning the self, the world, and the future. These negative appraisals Beck calls the negative (or primary) cognitive triad.

III. THE NEGATIVE COGNITIVE TRIAD

Beck (1967) argues that the disturbances in depression result from the activation of a set of three cognitive patterns that lead the individual to appraise himself, his world and his future in a negative (loss) and idiosyncratic way.

Negative view of the world: This component of the triad reflects the depressed individual's appraisal of his interaction with the environment. He sees his interaction as filled with obstacles and traumatic situations, all of which tend to defeat and detract from him in some important manner.

Negative view of the self: This second component of the triad reflects the depressed individual's view of himself. He regards himself as deficient, inadequate or unworthy in some important way. Most especially he regards himself as a 'loser'.

Negative view of the future: This component of the triad reflects the depressed individual's appraisal of the future.

> He believes that the world will continue to thwart his plans, and that he will continue to be inadequate and unworthy. Moreover he expects to feel depressed and 'bad' in the future. As things are now so they will continue to be in the future. The depressed patient can see no easing of his problems and troubles in the future.

These powerful negative attitudes and beliefs all contain significant loss properties. In Beck's (1967) model of depression it is the activation of this negative cognitive triad which is the primary cause of depression. As this negative cognitive triad comes to dominate the thought process, the individual distorts reality to make his interpretations and appraisals of the environment congruent with these dominant negative attitudes and beliefs. These cognitive distortions, in the form of selective abstraction, arbitrary inference, over-generalisation, etc. as discussed in the previous chapter, further increase the appraisal of loss and lead to a further lowering of mood. Moreover these cognitive distortions act as confirmatory evidence to the individual's negative beliefs and attitudes about the world, the self and the future. As the individual confirms his negative beliefs and attitudes via his cognitive distortions, the negative beliefs and attitudes become more dominant and powerful, leading to further cognitive distortions and a further deterioration in mood. Beck and Wynnewood (1964) argue that this sequence of events constitutes the main premise of their explanation of depression. In this early paper on the theory and therapy of depression they

argued that:

"The main thesis to be developed is that certain idiosyncratic cognitive structures (schemas) become prepotent during depression, dominate the thought processes, and lead to cognitive distortions." (P. 561)

The idiosyncratic cognitive structures (schemas) mentioned here refer to the beliefs and attitudes the individual holds concerning the world, the self and the future. More will be said of this concept of a schema later. For the moment, however, it can be seen that for Beck the predisposition to, and aetiology of, depression reside in factors that predispose the individual to suffer from the activation of the negative cognitive triad. Consequently an essential ingredient of Beck's cognitive theory of depression is concerned with the internal cognitive processes that predispose the individual to the emergence of a negative cognitive triad in certain situations.

IV. THE ROLE OF SCHEMAS

An important explanatory concept invoked by Beck is the concept of a schema. As Beck and Wynnewood (1964) and Beck (1967) point out, the concept of the schema has been developed and used by earlier writers, for example, Kelly (1955), Bruner et al. (1956), English and English (1958), Harvey et al. (1961), Miller et al. (1960). Although the definitions of schema vary slightly among these writers, the central idea of a schema is that it acts (exists) as an interposing cognitive structure which mediates between an external stimulus and the screening, coding, and evaluation of the stimulus properties. In many ways it is similar to the notion of the executive in cognitive theories of action (Neisser, 1967).

Beck (1974b) argues that:

"The sequence between the initial registration of a stimulus and final closure may be formulated in terms of continuous matching of incoming perceptual data against an individual's conceptual categories (schemas) . . . In psychopathological conditions, in which certain types of schemas are dominant, the adjustment between external stimulus and internal category may be poor; then the conceptualisation of a stimulus situation will be determined more by the invoked schema than by the configuration of the external stimulus." (P. 129)

The schemas that are dominant in the depressed individual are those that relate to the self, the world and the future. In the depressed individual these schema are negatively orientated leading to cognitive distortions and an exaggerated appraisal of loss.

V. PREDISPOSITION IN BECK'S THEORY OF DEPRESSION

The predisposition to depression in Beck's model is viewed as a predisposition to use negative schema relating to the world, the self and the future (negative cognitive triad) under certain situations. On this issue of predisposition Beck (1967) argues:

"The vulnerability of the depression prone person is attributable to the constellation of enduring negative attitudes about himself, about the world and about his future. Even though these attitudes (or concepts) may not be prominent or even discernible at a given time, they persist in a latent state like an explosive charge ready to be detonated by an appropriate set of conditions. Once activated, these concepts dominate the person's thinking and lead to the typical depressive symptomatology." (P. 277)

Thus Beck answers the question of predisposition by arguing that the negative cognitive triad already exists in the depression-prone individual in a latent state. The development of this negative cognitive triad, the place of origin as it were, Beck argues, can be found in the early learning history of the individual. Beck (1974), comments:

"In the course of this development, the depression-prone person may become sensitised by certain unfavourable types of life situations such as the loss of a parent or chronic rejection by his peers. Other unfavourable conditions of a more insidious nature may similarly produce vulnerability to depression. These traumatic experiences predispose the individual to overreact to analogous conditions later in life. He has a tendency to make extreme, absolute judgements when such situations occur." (P. 7)

Beck (1967) also argues that the depression-prone individual attaches negative attitudes to certain attributes such as "it's terrible to be stupid", or it's disgusting to be weak". The tendency to label the self as having these "bad attributes" again appears to occur in the early learning history of the individual. Beck believes that failure may be labelled as evidence of being inept or inadequate. For example,

". . . a child who gets the notion that he is inept, as a result of either a failure or of being called inept by someone else, may interpret subsequent experiences according to this notion. Each time thereafter that he encounters difficulties in manual tasks he may have a tendency to judge himself as inept. Each negative judgement tends to reinforce the negative concept or self image. Thus a cycle is set up: each negative judgement fortifies the negative self-image which in turn facilitates a negative interpretation of subsequent

experiences which further consolidates the negative self-concept. Unless this negative image is extinguished, it becomes structuralised, i.e. it becomes a permanent formation in the cognitive organisation. Once a concept is structuralised, it remains permanently with the individual, even though it may be dormant; it becomes a cognitive structure, or schema." (Beck, 1967, P. 275-276).

Thus Beck's (1963, 1967, 1974, Beck and Wynnewood, 1964) model of depression argues that the depression-prone individual early in his life develops particular negative cognitive schemas relating to the world, the self and the future. Although these negative schemas may not be discernible at any given time, they are easily invoked by life events which are similar to those that were responsible for their formation. Thus, for example, the individual who has been labelled as inept or inadequate by others in failing at a certain task, will tend to respond to failure with this concept (of being inadequate or inept) when he confronts failure in the future. The result of this is the activation of the negative view of the self. As this view dominates the individual's cognitive processes, he will interpret all failures, trivial or otherwise, as evidence that he is inadequate. This shift towards a negative appraisal of other events outside the invoking situation is a cognitive distortion which results from the activation of the negative view of the self. The cognitive distortion, however, confirms the correctness of the invoked negative schema and leads to a further increase in its dominance in cognitive processing. The more dominant the negative cognitive schema becomes, the more cognitive distortions occur and the greater the disturbance of affect and depth of depression.

VI. RULES AND BELIEFS IN DEPRESSION

Beck has argued that schemas consist of beliefs, attitudes and assumptions which govern the interpretation of experience. Until recently there was some confusion as to the relationship between negative schema and the negative cognitive triad. However, in Beck's (1976) more recent work this relationship has been more clearly outlined. Beck (1976) has introduced the concept of rules and premises which govern the interpretation of experience. Beck relates his concepts of rules to the concepts of other writers by saying:

> " Inasmuch as other writers have used terms such as attitudes, ideas, concepts and constructs to refer to what we have called rules, those terms will be used interchangeably . . . irrespective of terminology used, many therapists have reported that helping the patient to modify his maladaptive ideas or to substitute more realistic attitudes has led to the disappearance of crippling anxieties, phobias and depressions." (P. 246-247)

For depression, Beck (1976) has listed a number of rules and attitudes which predispose individuals to excessive sadness or depression. Examples of these include

(1) In order to be happy, I have to be successful in whatever I undertake.

(2) If I'm not on top, I'm a flop.

(3) My value as a person depends on what others think of me.

In the discussion of the rules employed by the depressed individual, Beck argues that it is these rules that lead to a vulnerability to depression. Thus it is the use of these rules in the interpretation of experience that lead to negative cognitive triad and the other symptoms of depression. Moreover the key to understanding

the problem of predisposition to depression can be found in the early acquisition of these unrealistic rules in the individual's cognitive organisational processes. In other words, people become predisposed to depression because early in life the individual acquires certain unrealistic rules and beliefs, and continues to use these rules and beliefs later in life to organise and interpret his experience.

As Beck, (1976) points out, these rules are likely to lead to misery because they are absolute. It is impossible for one individual to be a success all the time, or for everyone to be thought of highly all the time. However, because of their absolute qualities any failure for events to comply to these rules results in feelings of rejection, unworthiness and failure.

This is a useful and illuminating development of Beck's theory because it becomes possible to look more closely at possible predisposing factors in depression. One interesting method for such investigations is to screen individuals for the existence of such unrealistic beliefs and to compare them in parameters of mood, anxiety and negative thinking with individuals who do not appear to hold such beliefs. If Beck is correct then those with a tendency to hold unrealistic beliefs or rules should also have a tendency to have lower mood, high anxiety and negative thinking. This research examines this possibility. However, before this is possible some of the rules as discussed by Beck need to be systematised. The later part of the chapter offers one approach that might help in this problem.

VII. THE PROBLEM OF FLEXIBILITY

Beck has presented a very interesting and useful theory of depression. It is a broad and flexible theory which encompasses many different factors in its explanatory framework. Particular attention is paid to attitudes, rules and

beliefs, attributions and the appraisal of events. One of these factors, attributions, not so far mentioned, is in fact a central concept in Beck's theory of depression. For example Beck (1974, 1976) argues that depressives are inclined to blame themselves for aversive events, often when there is no reasonable grounds for doing so. Beck (op. cit.) argues that when an aversive event occurs the individual wonders what it tells him about himself. The tendency to extract negative information about the self from such events is a common feature of the depressed individual. As Blaney (1977) points out, this aspect of Beck's theory provides a subtle distinction between his theory and Seligman's (1975) learned helplessness theory of depression. Seligman believes that aversive events are perceived by the depressed individual as being beyond his control (external attribution), Beck on the other hand argues that the depressed individual attributes the aversive event as being his fault, due to his own inadequacy (internal attribution). As will be mentioned later, however, Seligman, in a recent reformulation, seems to have moved nearer to Beck's internal attribution idea.

However, the broadness and flexibility of Beck's theory does produce certain problems. As Blaney (1977) points out:

> ". . . this very flexibility may render it immune to disconfirmation, or nearly so, and thus unsatisfactory as a scientific theory. What is needed is a specification of what role what cognitions have in what stage of the development or maintenance of what kinds of depressive conditions. Though Beck has not presented a theory of the needed specificity, his writings contain much material that could be expected to contribute to such a theory." (P. 212-213)

The next few sections attempt to present a slightly different orientation from

Beck's theory, which may go some way to answering Blaney's criticisms. Such an orientation attempts to specify and systematise the cognitive processes that Beck attempts to change in therapy. This may allow for the theory to develop insight into what cognitions at what stages are important.

VIII. THERAPEUTIC INTERVENTIONS: CHANGING COGNITIVE PROCESSES

The therapeutic procedures of Beck's theory share much in common with Rational Emotive Therapy (Ellis, 1962, 1970). Ellis argues that the emotionally disturbed individual is burdened by a core of irrational beliefs and ideas. Corrective therapy should aim to instate more rational beliefs, although there is some doubt as to whether rationality in itself is sufficient for producing emotional change (Mahoney, 1974, Blackburn, 1978). However, the actual dynamics of R.E.T. and Beck's therapy do share much in common. Both, unfortunately, are difficult to grasp in view of their loose and anecdotal presentation (Hammen and Krantz, 1976). Nevertheless, from a number of sources (Beck, 1963, 1967, 1970, 1973, 1974, 1976, Beck and Wynnewood, 1964, Ellis, 1970, Mahoney, 1974, Blackburn, 1978) it is possible to systematise Beck's main therapeutic aims. These involve trying to change the following cognitive events in the depressed individual.

(1) Event evaluation — Helping the patient to evaluate correctly the aversiveness (or positiveness) of an event. This appears to be directed at the various cognitive distortions, such as selective abstraction, arbitrary inference, etc. which distort evaluative decisions. These

distortions, produced by the dominance of a negative cognitive triad belief structure, lead to an exaggerated appraisal of loss.

(2) Attributions: Helping the patient to make realistic attributions for causality. In the face of a traumatic event the patient is helped to make alternative attributions for the event. This appears to be aimed at breaking the tendency for depressives to internalise, i.e. blame the self for mistakes and aversive events, e.g. "It's my fault things are as bad as they are".

(3) Irrational or stereotyped attitudes, beliefs and rules: Helping the patient change unrealistic beliefs or attitudes, e.g. "I need to be liked by everyone", or "Only successful people are loved and respected". In Ellis's terms, it involves helping the patient realise that to be fallible is not bad and should not be despised or dreaded. These negative beliefs are reflected in themes and the patient's automatic thoughts.

To a considerable degree these three cognitive events interact with each other. For example, a particular attribution may influence the evaluation of the aversiveness of an event. Nevertheless, these three therapeutic interventions

do seem capable of being reasonably separated.

With regard to the first of the interventions outlined above, event evaluation, some time was spent in the last chapter considering how depressives mis-evaluate their environmental situation. It was shown that this mis-evaluation can significantly impair adaptive coping behaviour and produce physiological vulnerability. However, attributions and attitudes also have important influences on event evaluation and coping behaviour and it is to these two factors that attention is now turned.

IX. ATTRIBUTION THEORY

Attribution theory rests on the philosophical position of Hume and Kant which asserts that causes cannot be observed but are constructed by the perceiver to render the environment more meaningful. In this sense they are psychologically determined. Attribution theorists have addressed themselves to the question of how causes are attributed to different events. The general events most studied by attributional theorists have been success and failure in achievement related tasks (Weiner, 1972).

Weiner and his colleagues (Weiner, 1972, 1974, 1977, Weiner et al., 1972) have argued that causal perceptions require two dimensions of explanation. One of these dimensions comes from the social learning theory of Rotter (Rotter, 1966, Rotter et al., 1962). This dimension is the locus of control or internal external dimension. According to this theory, causality is attributed either to internal factors (self) or external factors (the world). Those that adhere to the locus of control dimension of causality argue that expectancy and the persistence of behaviour depends on whether the individual perceives the event as being internally controlled (by self) or externally controlled (by others or the world).

Perceptions of internal control produce high persistence, perceptions of external control produce low persistence.

Attribution theory on the other hand argues that it is a stable/unstable dimension that best explains expectancy change and behavioural persistence. If the individual perceives the cause of success or failure as due to stable factors, then the same outcome can be expected in the future and persistence is determined accordingly. If the person perceives the causes of success or failure as due to unstable factors, then persistence may be high in the case of failure (tries harder, effort attribution) or low in the face of success (e.g. slot machine, luck attribution). The stable factors most investigated by attribution theorists have been ability and task difficulty. The unstable factors are effort and luck. The interaction between these two dimensions of perceived causality can be shown in the following way (Weiner, 1972, Weiner et al., 1972):

	Internal	External
Stable	Ability (a)	Task difficulty (b)
Unstable	Effort (d)	Luck (c)

These two dimensions have often been confounded in research. As Weiner (1972) points out:

"In experiments conducted by the Rotter research group, ability and luck

instructions are varied between experimental conditions. Thus the effects of internal, stable attributions are compared with the consequences of external, unstable attributions. The two dimensions of causality are confounded." (P. 397)

The predictions of the two theories are different. From the point of view of the relevance to depression it is the prediction of an attribution in quadrant (a) in response to failure that is important. According to social learning theory the perception that the event is internally controlled should generate high persistence. Attribution theory, however, would argue that since failure is attributed to a stable factor, the same outcome (failure) can be expected in the future, thus persistence is low. Although these issues are not straightforward, there is some evidence that the stable/unstable dimension is a better predictor of behaviour persistence and expectancy change (Weiner, 1972, Weiner et al., 1976, Dweck, 1975).

Caution should be exercised in the consideration that these two dimensions are independent. For example, effort probably depends on the perception that the individual does have the basic ability but needs to try harder. If the individual perceives he does not have any ability then there is really not much point in trying, (indeed to what extent is effort simply a measure of behavioural persistence?). Similarly for luck and task difficulty: luck attributions are more likely if the task is first perceived as being either unpredictable or difficult. Outcomes of tasks judged to be predictable and simple probably do not lend themselves to luck attributions. The interactions of these various attributions require further research. Such possible interactions between attributions would seem to carry

significant implications for research using the Rotter (1966) internal-external scale. For example, an internal attribution of low ability may produce a high external score (I cannot control my environment because I do not have the ability). These factors complicate interpretations of research using the internal-external scale (Lefcourt, 1976).

X. ATTRIBUTION THEORY IN DEPRESSION

Weiner et al. (1978) have shown some evidence that different attributions in success and failure situations can produce different affective reactions. These affective reactions are far more varied than the pride-shame affective reactions originally described by Atkinson (1964). As an introductory explorative method of examining different affective reactions associated with different attributions for success and failure, Weiner et al. (op. cit.) presented a brief story which gave a cause for success or failure to students, and asked them to rate the expected affective reaction. While accepting the limitations of such initial experimentation some of the findings have a significant implication for depression. Most noted in this regard were the affective reactions given to failure, according to different attributions. Weiner (op. cit.) found that the most dominant affective reaction given to lack of ability attribution was incompetence and inadequacy. Stable effort (as opposed to unstable effort) seemed closely related to depression with the affective reactions of hopeless, depressed and disheartened. Similar negative affects were associated with internal attributions to personality factors in the failure conditions.

It is too early yet to be able to draw any detailed conclusions from this work and much more research is required. Yet this does seem to be a useful and promising area. This is especially so since Beck (1967, 1974, 1976) has argued

that depression-prone individuals tend to make internal attributions for adverse events.

> "As the traumatized person reflects about adverse events (such as separation, rejection, defeat, not achieving his expectations), he wonders what it tells him about himself. The tendency to extract personally relevant meanings from unpleasant situations is particularly characteristic of the depression-prone individual. Moreover, a special impetus to ascribing negative meanings to a loss is produced by the tendency to find some personal explanation for important life events. Usually such determinations of causality are very simplistic and may be quite erroneous." (Beck, 1974, P. 9)

Thus, as Beck points out "the depression-prone individual is likely to assign the cause of an adverse event to some shortcoming in himself." If Weiner et al.'s (19[illegible] results are shown to have validity, then this would seem an important development for cognitive theories of depression, since attributions for failure may not only lead to reductions of self-esteem but also to a lowering of mood.

Unfortunately, recent research which has implications for this attributional approach in depression has not primarily addressed itself to Beck's model of depression. In general, the examination of attributional processes in depression have developed from Seligman's (1974, 1975) learned helplessness theory of depression (Klein et al., 1976, Dweck, 1975, Roth and Kubal, 1975, Weiner, 1977). In fact much of the work conducted on the learned helplessness model of depression is open to an attribution interpretation. Douglas and Anisman (1975) found that in the typical experimental manipulation of learned helplessness (Hiroto and Seligman, 1975), perceived difficulty was an important determinant of learned helplessness.

These researchers argued that under conditions where subjects expected to succeed, but failed, subsequent performance was disrupted. However, when there was low expectation to succeed, failure did not necessarily disrupt subsequent performance. The possible involvement of expectancies of success and failure in learned helplessness conditions carries important implications for attribution theory. Indeed Douglas and Anisman (op. cit.) hint at the possibility that the attribution of incompetence from failure at a simple task might be an important factor responsible for subsequent performance disruption.

Another study which lends itself to an attributional interpretation has been conducted by Roth and Kubal (1975). They found that manipulating the perceived importance of a task (importance was manipulated by presenting the typical learned helplessness training task as being either a good predictor of academic success or merely a puzzle) had significant effects on subsequent performance. The high importance condition produced the greatest amount of subsequent disruption. Again these authors consider that different attributions were important factors in determining subsequent performance deficits.

The clearest demonstration of the role of attributions on both depression and learned helplessness has been given by Klein et al. (1976). In this study the subjects consisted of two groups, depressed and non-depressed students. After being exposed to a discrimination task adapted from Hiroto and Seligman (1975), subjects were examined on their ability to solve anagrams. However, each group was split into three sub-groups, the first receiving internal attribution training, the second receiving external attributional training and the third receiving no attributional training. This was achieved by presenting a histogram showing the percentages of previous subjects' scores on the anagrams. In the internal

attribution condition, the subjects saw a histogram which showed that 55% of subjects managed all four anagrams correctly, 30% managed three, etc. In the external attribution condition the histogram showed that no-one achieved four correct answers, 1% solved three, 2% solved two, etc. The no attribution group were not presented with any histogram.

The results demonstrated that different attributions for failure resulted in different performance deficits in the depressed (though not the non-depressed) group. External attribution instructions resulted in better anagram performance than internal or no attribution instructions in the depressed group. Klein et al. discuss this finding in the light of Beck's (1967) theory of depression and argue that negative self attributions are probably important factors mediating learned helplessness in depressed individuals. Work by Tennen and Eller (1977) has also shown the importance of attributions on learned helplessness. Attributions of low ability produced learned helplessness, but attributions to task difficulty actually increased performance at a different task.

As Klein et al. (1976) point out, there are large gaps in this area. For example, there is little information about the tendency to generalise an attribution made in one situation to other situations. And there is no study which clearly examines the interaction of depression and causal attributions. However, in view of these findings it appears that Seligman and his colleagues are attempting to reformulate the learned helplessness model of depression. For example, Weiner (1977) has pieced together some paragraphs from an unpublished manuscript by Abrahamson et al. (1978) which apparently presents a reformulated model of the learned helplessness theory of depression in terms of attribution theory. Quoting from Weiner's text on this paper:

"Our reformulated hypothesis makes a major new set of predictions. The helpless individual first finds out that certain outcomes and responses are independent, then he makes an attribution about the causes. This attribution determines the chronicity, generality and intensity of the deficits. Depressed people seem to make more global, stable and possibly internal attributions about the cause of their helplessness and as a consequence show more general, chronic and intense deficits than non depressed people.

Depression occurs when an individual expects that the probability of a highly preferred outcome is low and he expects that he is helpless to increase it. If the attributions for the present state of affairs are to stable and global factors, the future will look dark to the individual. He expects that he will find himself helpless again and again to increase the low probability of a variety of important events. This is what is usually meant by 'hopeless'. Another implication of the formulation is that individuals will show the greatest loss of self esteem when they make internal global and stable attributions for the future." (P. 29-30)

In this reformulation, it would appear that attributional processes are the mechanisms which cause the behavioural state of learned helplessness to produce low mood. There is some doubt as to how far learned helplessness can withstand this change in emphasis, since the most salient perceptions of causality have always been external perceptions (Seligman, 1974, 1975).

However, as mentioned earlier, internal stable attributions can produce an individual who believes he has no control over his environment (due to lack of ability). Consequently Seligman's model could be developed into a two stage theory of depression; first the development of learned helplessness (external

attribution; depression follows when the individual blames himself for his helplessness. It is interesting to speculate whether such a formulation might make the learned helplessness theory a special case that could be incorporated within Beck's theory.

At the present time not enough has been written about this reformulation of the learned helplessness theory to allow clear discussion of the issues. However, Klein et al. (1976) and Weiner (1977) have turned an important spotlight on the role of attributional processes in depression. Moreover, as Rehm (1977) has pointed out, the role of self-evaluative processes has played an important part in many theories of pathological behaviour, including depression. What is required is more systematic research in this area.

In addition to attribution and evaluations, those beliefs and attitudes which predispose the individual to the activation of the negative cognitive triad are essential features of Beck's model of depression. Moreover, they may offer some insight as to why certain individuals tend to make negative self attributions and exaggerate the degree of loss in the environment. It is these considerations which are taken up in the following sections.

XI. TYPES OF MALADAPTIVE BELIEFS IN DEPRESSION

Beck (1974, 1976) has argued that precipitating factors in depression need not always be discrete events. They can be insidious stresses which persist over a period of time, such as the gradual withdrawal of affection by a significant other. Moreover the individual may be continually dissatisfied with his or her performance as a wife, parent, income producer, student, etc. In brief, the individual sees high discrepancies between his aspirations and goals and his actual performance.

Beck argues that this discrepancy may be due to the individual striving for unrealistic goals or aspirations, with unrealistic expectations.

Brown et al. (1977) have offered some interesting insights into the vulnerability of self-esteem following life events, e.g. loss. However there is little theory that attempts to relate how beliefs and attitudes about achievements and goals can also lead to a vulnerable and negative view of the self, the world and the future and to depression.

The next few sections attempt to throw some light on this area and perhaps add some marginal refinements to the Beckian and cognitive approach to depression.

The area of psychology that offers a useful starting point for such thinking, is achievement theory. Unfortunately this area has become highly specialised and plagued by a number of internal problems, especially concerning the constructs used and the measurement of those constructs (Weiner, 1972, Birney et al., 1969, Weinstein, 1969, McReynolds and Guevara, 1967). Nevertheless as Weinstein (op. cit.) points out, achievement theory as developed from Murray (1938) Atkinson (1964) and McClelland (1951) has inspired a lot of research and has generated a number of important insights. Indeed it is these insights which may offer some useful ideas for understanding why some individuals may become vulnerable to depression at a cognitive level.

XII. ACHIEVEMENT THEORY AND DEPRESSION

As mentioned, achievement theory has developed from the work of Murray (1938), Atkinson (1964), McClelland (1951) and recently by Weiner (1972). Its main investigatory tool has been the Thematic Apperception Test which is believed to produce an index of the individual's motivational concerns. Weiner (1972) offers a clear summary of the principles and parameters of the theory:

". . . The theory specifies that achievement related behaviour is a resultant of a conflict between a hope of success (approach motivation) and a fear of failure (avoidance motivation). The approach and avoidance tendencies, in turn, are a function of achievement-related needs (need for achievement and anxiety about failure), the expectancy of success and failure, and the incentive value of achievement tasks is determined by the probability of success." (P. 263)

As Weiner (1972) has pointed out, more concern should be given to the 'perceived' probability of success. This involves judgements and attributions about stable and unstable conditions. Weiner (1972) has offered a reformulation of achievement theory in terms of attribution theory. Though this is important it will not concern us here. However, the point that attribution and achievement motivation are locked together in an interactive way can be seen by research that has examined these issues. To quote from Weiner's (1972) excellent book again, the products of some of this research can be summarised as follows:

"Individuals differing in level of achievement needs differ in their dispositions to allocate causation to these (task difficulty, luck, effort, ability) factors. Persons high in achievement needs ascribe success to high ability and high effort, and ascribe failure to a lack of effort. Persons low in achievement needs, on the other hand, relatively attribute success to external factors and failure to a lack of ability. The causal dispositions, in turn, mediate between achievement tasks and the final achievement-related responses of approach behaviour, intensity of performance, persistence of action and risk-preference." (P. 417)

In general the low need achiever is considered to have a stronger motivation to

avoid the aversive consequences of failure than to approach the positive consequences of success, i.e. is considered to have high fear of failure.

If the prior analysis is correct (Beck, 1967, 1976, Klein et al., 1976, Weiner, 1977, 1978), then the above quotation might be taken to argue that the depression-prone individual who attributes failure to himself may be a fear of failure motivated person. Although the author suspects this as a possibility in some cases, the issue is more complex than this. Some research has shown that the fear of failure motivated individual can adopt either a low probability of failure task (ensuring success) or a high probability of failure task (blaming task difficulty for failure) (Atkinson, 1964, Peck and Whitlow, 1975, Weinstein, 1969). However, thi issue is not yet settled (Weiner, 1972). More important is the possibility that the strength of the fear of failure motive is determined by the perception of attached reinforcements contingent on the success/failure situation. In other words, some individuals may overtly appear as high need achievers, but are attempting achievements for secondary gains or reinforcements (e.g. love, respect, prestige, etc.).

The possibility of such a distinction between differently motivated achievers was noted by McClelland et al. (1953). They argued that a high need achiever could be distinguished from a high value achiever. The high need achiever was considered to be an individual who lived up to internalised standards. He or she prefers moderate risks and strives for achievement in fantasy and actuality. Moreover, such individuals are relatively independent of social or authoritarian influences. In contrast to these individuals are value achievers who overtly avow to high standards which are unrelated to fantasy or actual performance and are significantly influenced by authority and social influence such as social approval. McClelland et al. (op. cit.) speculated that value achievers developed in response

to:

"Authoritarian pressure from their parents to be ambitious and the resultant motive which has originated in external sources shows itself as a fear of being unsuccessful." (P. 419)

This last quotation raises some interesting questions since it would appear that the value achiever is similar if not identical to the fear of failure person. Unfortunately the theory of achievement motivation has not taken this issue very far. Weiner (1972) has very little to say about value achievement. However, logically it would seem that the high value achiever would share many common characteristics with the fear of failure motivated individual. One important difference that might exist between them concerns aspiration level. The value achiever is likely to have overt high aspirations, attempting to gain social approval, etc. while the same does not necessarily apply to the fear of failure motivated person (Weiner, 1972, Birney et al., 1969).

XIII. VALUE ACHIEVEMENT AND DEPRESSION

In 1954, Cohen et al. reformulated a theory of manic-depressive illness within the framework of McClelland et al. and De Charms et al. (1955) achievement theory. Critical to Cohen's et al. (op. cit.) reformulation was the distinction between value achievers and need achievers. Special emphasis was placed on hypothesised similarities between the value achiever's and the manic-depressive's orientation to success. Cohen et al. (1954) speculated that the premanic-depressive child was used by the parents (especially the mother) as the method of obtaining social approval and advancement. It was through the achievements and behaviour of the child that the family would be socially judged by others and would socially prosper.

As a result, in adulthood the premanic-depressive individual in response to parental pressure had internalised achievement strivings developed as a means of obtaining social approval. Thus achievement was pursued for secondary gains (social approval, respect, etc.) rather than the intrinsic rewards associated with success, characteristic of the need achiever.

Experimental evidence for this formulation has been presented by a number of studies, the most important being Cohen et al. (1954), Becker (1960), Becker et al. (1963), Becker and Nichols (1964). These studies, using remitted manic-depressives did present evidence that manic-depressives were high in value achievement. Becker (1960) compared twenty-four male remitted manic-depressives with thirty non-psychiatric controls and found that manic depressives performed like high value achievers, where achievement and conforming behaviour were related to the aim of gaining the approval of others rather than satisfying internalised standards. Interestingly, on need achievement scores the manic depressives were not significantly different from the controls.

Unfortunately an attempt to replicate these findings with female remitted manic depressives failed (Becker and Altrocchi, 1968). Becker (1974) offers possible explanations for this failure to replicate earlier results.

With very few exceptions (Becker, 1974) this line of research has not been further developed. One particularly promising area is the relationship between fear of failure and value achievement, for there is good reason to propose that in many respects the value achiever and the fear of failure motivated individual share many common characteristics. First, as McClelland et al. (1953) pointed out, the value achiever is "fearful of being unsuccessful". This fear is related to a belief that failure not only results in the loss of hoped for rewards but also

results in a loss of social approval and other secondary reinforcers. It is precisely because achievement is pursued for these secondary gains that failure has such serious implications. Because of past reinforcement contingencies by the peers, the self estimate is probably rooted in an ability to maintain social approval, love and respect. In this sense the fear of failure motivated individual and the value achiever seem identical (Birney et al., 1969).

XIV. FEAR OF FAILURE AND THE SELF ESTIMATE

The interrelationship between self estimate and achievement involves a host of factors, but for some individuals maintaining a steady self estimate seems rooted in externally controlled factors such as significant others. Holt (1969) who has spent much time observing children in the classroom offers this interesting insight which bears centrally in this issue. He points out:

> "Note the danger of using a child's concept of himself to get him to do good work. We say, 'You are the kind of sensible, smart, good, etc. etc. boy or girl who can easily do this problem, if you try'. But if the work fails, so does the concept. If he can't do the problem, no matter how hard he tries, then, clearly, he is not sensible, smart, or good.
>
> If children worry so much about failure, might it not be because they rate success too highly and depend on it too much?" (P. 55)

Gibby and Gibby (1967) have found that induced failure in children resulted in a lowering of the self estimate and a belief that significant others (parents and teachers) would think less highly of them as a result of failing.

These findings in conjunction with Holt's (op. cit.) observation, draws attention to the potentially damaging situation in which significant peers attach

the child's own view of his worth to achievement strivings. In other words, the determinants of self estimate are related to how the parents and significant others manipulate the child's own concept of himself. Thus the parents may lead the child to believe that it is what others think of him or her that really determines social worth. Alternatively, through the manipulation of reinforcement contingencies, the child may come to believe that it is only through success that a self-estimate allied with secondary reinforcers, e.g. love and respect, can be maintained.

The structure of reinforcements that accompany such manipulations can only be speculated on. If parents withold love or attention when the child is judged to fail at some important social or academic task, then it seems probable that subsequent social, economic and/or academic striving may be primarily aimed at maintaining self-esteem and ensuring a continual flow of secondary reinforcers (e.g. love and respect).

Teevan and McGee (1965, reported in Birney et al., 1969) attempted to investigate the reinforcement patterns of mothers whose children had been identified as having a high fear of failure. These mothers apparently expected earlier independent behaviour from their children, and punished failure whilst being neutral to success (comparisons were made with the reinforcement patterns of mothers whose children were low in fear of failure).

The problems that might be consequent on such reinforcement manipulations are pointed out by Birney et al. (1969):

"The problem with instructional techniques that use punishment as a motive source is that the individual's chief concern is with the avoidance of the

punishment and only secondarily with the escape route that the punisher might have in mind. If there are a number of options available, the best-educated guess we have is that he will choose the most dependable alternative, the one that has worked best for him in the past." (P. 12)

As Birney et al. (1969) point out, it is very difficult to predict exactly what strategy for the avoidance of punishment will be adopted. However, one strategy for avoiding the aversive consequences of failure (e.g. reduction of love and respect by peers) may, in the predepressive child, be self devaluing and punitive behaviour. The aim of such behaviour is to reinstate those reinforcers (e.g. love and sympathy) that the child believes will be withheld if or when he fails.

Forrest and Hokanson (1975) have examined the common observation that depressive conditions have a significant self punitive quality. They hypothesised that:

". . . one component of depression may be related to the instrumental value that depressive, self-demeaning displays have in controlling aversiveness and threat from others." (P. 347)

In other words, self-punitive behaviour may be an adopted strategy for controlling aggressive or punishing behaviour from others. These authors predicted that depressives would adopt higher rates of self-punishing behaviour when attacked in interpersonal situations, than normal controls. They also argued that these self-punishing behaviours should manifest a faster reduction in autonomic arousal in depressed as compared to non depressed controls.

The results from an experimental situation in which self-punitive behaviour was positively reinforced, confirmed both predictions.

The view that organisms will expose themselves to negative stimuli if these negative stimuli have been paired with the avoidance of more aversive situations is well documented in animal literature (Sandler, 1964, Mackintosh, 1975). Moreover, as Forrester and Hokanson (1975) point out:

"The depressed group's pattern of responding suggests a learning history in which the reinforced mode to aggression was either friendly or self-punitive." (P. 355)

These results have an important bearing on the possible role of fear of failure in depression. If the pre-depressive child is fearful of failure because the consequences are often a reduction in social approval, love and respect from significant others, then the self-devaluing behaviour might be a useful strategy to salvage some of these reinforcers (love, sympathy, support, attention, etc.). In other words the child may learn that expressing severe disappointment and self blame for failure may reduce the probability of the parents responding aggressively to the child's failure, and hopefully produce instead sympathy and support.

Unfortunately this strategy may produce an unstable self estimate. On the one hand the individual may strive to achieve various goals, primarily for secondary gains (social approval etc.). Due to parental manipulation of reinforcement contingencies, the child may come to believe that his self-esteem is very much dependent on what other people think of him and being a success in the world. This outcome would seem to generate rules in the individual which Beck (1976) believes are important pre-disposing factors in depression. Moreover, failure to reach these internalised goals produces the appraisal that the individual will no longer be loved or respected with the associated appraisal of loss. In order to maintain some of these interpersonal reinforcements, the individual may adopt a strategy of self-punitive behaviour that is aimed at reducing hostile interpersonal interactions producir

supportive behaviour from others.

This type of analysis has implications for Coyne's (1976) formulation of depression, although Coyne does not, as has been discussed here, refer back to possible early learning experiences. Essentially, Coyne (op. cit.) argues that persistent demands for support from the depressed individuals soon become aversive to others so that they reduce rather than increase the supportive behaviour towards the depressed individual. This produces more distress in the depressed individual and more demands which have the unfortunate effect of reducing supportive behaviour from others still further.

Lewinsohn (1974, 1975), like Coyne, stresses the importance of socially reinforced behaviour although he underlined different factors. However, an emphasis in social skills is central to both theories, and, as Blaney (1977) points out, they use similar research material to support their views. Ferster (1973, 1974) also stresses the behaviour of the depressed individual and calls attention to the amount of passive avoidance of the depressed person.

It is too large a task to unite all the different approaches within one framework, but there does seem to be a coming together of the different approaches, both in depression and general psychology (Mahoney, 1976). Moreover, an investigation of early learning experiencesin the depression-prone individual may shed interesting light on a number of factors and show that the behavioural and cognitive theories of depression may happily be married in a useful and more comprehensive framework.

XV. STRATEGIES AND EVIDENCE OF STRATEGIES

In this last section, the relationship between fear of failure, the strategies

adopted for coping with failure and depression will be examined. In discussing the fear of failure issue in children, Holt (1969) points out that the strategies adopted by many children

". . . have been consistently self-centred, self-protective, aimed above else at avoiding trouble, embarrassment, punishment, disapproval, or loss of status. This is particularly true of the ones who have had a rough time in school. When they get a problem, I can read their thoughts on their faces. I can almost hear them 'Am I going to get this right? Probably not; what'll happen to me when I get it wrong? Will the teacher get mad? Will the older kids laugh at me? Will they keep me back this year? Why am I so dumb? and so on." (P. 58-59)

A strong fear of failure probably generates considerable internal turmoil, when confronted with the possibility of failing. Undoubtedly such turmoil produces internal speech of the form Holt seems to indicate. Although this cannot be taken further here, it would seem to be relevant to the self-monitoring (Rehm, 1977) and inner speech directed theories and therapies of psychopathology (Miechenbaum, 1977, Mahoney, 1974).

An important implication of fear of failure is the attention allocated to the potential failure situation. As Birney et al. (1969) point out:

"Whether the fear is a relatively stable personality trait or one that is created by the conditions of the achievement situation, it is a reaction to cues that signal future failure. Since the failure has not yet occurred, the individual has the opportunity to engage in avoidance behaviour. As the fear increases in magnitude, we would expect a greater tendency to behave in a defensive manner." (P. 209)

It was argued that one possible defensive behaviour might be to devalue self. Thus even before a failure event has occurred the depressive-prone individual may selectively attend to possible failure cues and self-devalue if these cues are perceived as constituting a significant threat to achievement. The depression-prone individual may thus be caught in the crossfire of two related motivations. A high value achievement may motivate the individual to aspire to high goals. However, a high fear of failure may sensitise the individual to becoming aware early in the achievement task of possible failure cues, so that even in his achievement striving the individual may adopt certain defensive behaviours which may include self-devaluation. Unlike the fear of failure individual who may not have a high value achievement motive, the depression-prone individual cannot back off from attempting the task. To do so would be to endanger social approval and the maintenance of love and respect, etc. Thus the individual may pursue high aspirations while attending to the potentially negative cues within the achievement situation. Each time such negative cues are encountered the individual attempts to defend against social disapproval, hostility and loss of love by devaluing himself.

The outcome would be an individual who appears never satisfied with his achievements since he is sensitised to possible failure cues and adopts a defensive strategy as a result. As Beck (1967, 1974b, 1976) points out, such a situation is common in depression. Moreover because the achievement situation is so linked with obtaining secondary reinforcers (love and respect, etc.) failure may produce a significant expectation of loss (Beck, 1974b, 1976).

If self devaluation is a defensive strategy adopted in the pre-depressive child, then such a strategy may become aversive in adulthood, resulting in a

loss of support. Such a state of affairs would lead to further perceptions of failure and loss, with more self-devaluation and further loss of support.

There is some evidence to suggest that depressives are more sensitive to aversive events and even when doing well at tasks underestimate the amount of positive reinforcement they have obtained. Lewinsohn et al. (1973) has shown that depressed students are more sensitive to negative events (mild electric shocks) than non-depressed students ('sensitivity' was measured by galvanic skin response (G.S.R.) to the shock). Wener and Rehm (1975) demonstrated that depressed students underestimated the number of times they made a correct response on a supposed intelligence task. These authors argued that depressed students selectively distorted their recall. Equally possible is the interpretation that they were more attentive to the failure cues (failure was signalled by a light). As these authors pointed out, an underestimate of the correct responses implies an overestimate of incorrect responses. However, this study did not directly measure recall for negative (failure) events. Nelson and Craighead (1977) in a modified design of Weiner and Rehm's (op. cit.) experiment, attempted to examine whether depressed compared to non-depressed students would overestimate the degree of (negative) failure events. The results of this study did not produce straightforward results. At a high rate of positive reinforcement (70% positive, 30% negative), depressed students did significantly underestimate their performance. In the high failure condition (70% negative versus 30% positive reinforcement), however, the non-depressed students underestimated the number of incorrect responses whereas the depressed students were more accurate in their estimates.

If the comparison is made simply between the two groups then the depressed students did significantly underestimate their success and overestimate the

failures, as compared to non-depressed students. When the relation between these estimates are made with actual performance, the results are more difficult to interpret. However, since both experiments (Weiner and Rehm, 1976 and Nelson and Craighead, 1977) presented the task as skilled rather than luck determined tasks, the different expectancies that each group might have brought to the experimental situation should be considered. For example, Loeb et al. (1971) found that although depressives as compared to non-depressives had similar levels of aspiration, depressives expected to perform less well and rated their actual performance less favourably and Miller and Seligman (1973) found that depressed students expected to do poorly in skilled tasks. Thus receiving 70% success may not have been expected by the depressed group and their evaluation of their success was below actual performance because of the influence of this negative expectancy. For normal non depressed students, 70% success was more in line with their expectations so they estimated correctly. In the high failure situation (70% failure), however, the actual performance was more in line with the depressed students' expectations and thus they estimated actual performance accurately. For the non-depressed group, 70% failure may not have been congruent with their expectations and thus they underestimated the amount of actual failures.

Since individuals attend to information which confirms rather than disconfirms their expectancies (Neisser, 1967, 1976), distortions in the allocation of attention might arise accordingly. Thus selective attention may become distorted towards negative events though either a high fear of failure and/or a low expectation of success. It is likely, however, that there is a complex and synergistic interaction between these two phenomena which subsequent research may disentangle.

Moreover, it should be pointed out that low expectancies of success is often a common characteristic found in individuals who are high in fear of failure (Birney et al., 1969).

In conclusion to this section, it can be speculated that fear of failure is a reflection of certain irrational beliefs. Such beliefs may exist as "I will not be loved if I fail", i.e. there is the belief that certain reinforcers will be withheld or lost if the individual fails in certain tasks. It is these beliefs that produce a high fear of failure, and in addition an oversensitivity to negative cues.

XVI. CONCLUSION

This chapter began with an examination of Beck's cognitive theory of depression. Attention was drawn to the assumptions and general premises on which this approach rests. In addition the basic axioms of this approach were discussed and the relationship between the various aspects of the theory such as irrational rules and attitudes, the negative cognitive triad and cognitive distortions, which produce the lowering of mood and other symptoms of depression were outlined. It was suggested that Beck's theory identifies three main cognitive events of therapeutic concern: (1) event evaluation and appraisal, (2) attributions, (3) irrational rules and attitudes.

Within the earlier framework (chapter 3) of a coping behaviour - neurochemical change approach, it was suggested that evaluation of the aversive properties and consequences of an event, and the appraisal of available coping options to reduce these consequences to a large degree determined the subsequent response set (i.e. physiological, emotional and behavioural) to that event. In this chapter it was pointed out that such appraisals in turn depended upon factors such as the attributions

the individual made about the event and the interpretation of the event in terms of its personally relevant characteristics. This interpretation was governed to some extent by the rules and attitudes of the individual.

In the latter part of the chapter it was argued that these rules and attitudes, together with the tendency to make particular internal attributions, may reflect conflicting motivations. That is, an individual may be motivated to achieve success for secondary gains (e.g. love and respect, social approval, etc.) yet at the same time be highly motivated to avoid failure, indeed this high fear of failure occurring because of the individual's belief that secondary reinforcers will be withdrawn if he or she fails at whatever task is at hand. Moreover, because of such beliefs, the individual may appraise his environment as having significant loss properties when he perceives himself as not achieving certain standards.

This form of analysis was considered useful and was discussed in terms of offering possible insights into aetiological questions. Moreover this approach poses some interesting questions regarding the attitudes of depressed patients to various situations, and perceptual and attentional sensitivity to positive and negative events. In addition, if the analysis followed here has any validity, it should be possible to argue that high fear of failure subjects are more vulnerable to affective disturbances than low fear of failure subjects. The two experiments reported in the next chapters investigate some of these factors and attempt to throw some light on the important cognitive components of mood disorders.

CHAPTER FIVE

EXPERIMENT ONE: AIMS, DESIGN AND METHOD

The review of the literature in the previous two chapters has shown that research in depression has tended to concentrate on biological changes on the one hand, and psychological changes on the other. It was argued that some of the biological changes believed to occur in depression may, in some instances, be explained by psychological factors, namely the determinants and acquisition of coping behaviour. Coping behaviour was analysed in terms of primary and secondary appraisal, which in turn was considered to be partially dependent on attitudes, rules and schemas, and other internal cognitive factors.

I. AIMS

The aims of this study are therefore to investigate some of the internal cognitive factors which might be operative in depressive illness. Of such cognitive factors, two aspects were selected for particular attention:

A. perceptual and attentional factors, and

B. attitudinal factors.

The perceptual and attentional aspects considered to be most relevant were perceptual sensitivity and attention to positive and negative events. The attitudinal and cognitive parameters which appeared most relevant from previous studies and the analysis put forward in the literature review were, attitudes to perceived control over reinforcers, to failure in test situations, general anxiety and negative thought content. This study was intended to examine the above parameters in depressed

and recovered depressed patients as compared to control groups.

The questions to be answered can be summarised thus:

1) in what way do depressed patients differ from control groups in the perception and attention to positive and negative events?

2) in what way do depressed patients differ from control groups in their attitudes on the various parameters mentioned above?

3) how specific to depression are the cognitive and attitudinal distortions which have been implicated by previous research?

4) (a) in what way do depressed patients change in their attention and attitudes after recovery?

(b) how similar to the normal control group do recovered patients become?

II. DESIGN

A combination of a follow-up and cross-sectional design was used. The follow-up part of the design involved testing depressed patients when ill and after recovery. The cross-sectional part of the design involved comparing the results from testing the depressed patients when ill and on recovery with two control groups who were a group of normal non psychiatric subjects and a group of psychiatric out-patients suffering from general anxiety.

It was hoped to obtain twenty subjects in each of the four groups.

Comparing the depressed group with the two control groups would answer questions one, two and three outlined above. Comparing the depressed group when ill and recovered and comparing the recovered depressed group with the two control groups would answer questions 4(a) and 4(b) outlined above.

General hypotheses:

(1) Depressed patients will have a negative bias in their perception and attention,

and their attitudes (as measured) will be more maladaptive than the other two control groups.

(2) Recovered depressed subjects will retain some degree of negative bias in their perception, attention and attitudes which will differentiate them from normal controls.

III. METHOD

A. SELECTION OF PATIENTS

(1) Depressed group (Dep.)

The criterion for the selection of depressed patients in this study was decided by a group meeting of the MRC Brain Metabolism Unit's clinical staff. It was decided that any patient diagnosed as suffering from a primary depressive disorder would be suitable for participation in this study. Consequently both unipolar and bipolar depressed in-patients would be included, since the aim of the study was to examine certain cognitive parameters of depressed mood, without regard to diagnostic sub-types. Four patients seen in this group were considered to be suffering from a bipolar depressive illness. Special care was taken to exclude those patients who showed evidence of:

(a) Schizophrenia

(b) Organic brain damage

(c) Alcohol abuse

Since the author does not have a clinical qualification, patients were selected and allocated by the senior members of the clinical staff, all of whom had many years of clinical and research experience in the affective disorders. These

included Dr. I. Blackburn (principal clinical psychologist), Dr. J. Loudon (senior registrar), Dr. G.W. Ashcroft (consultant psychiatrist and Director of the MRC Brain Metabolism Unit), Dr. D. Eccleston (consultant psychiatrist) and Dr. I. Glen (consultant psychiatrist). Each patient was in the care of one or more of the above clinical members of staff at the time of testing. A total of twenty depressed in-patients participated in this group.

(2) Recovered depressed group (R. Dep.)

This group consisted of the same patients who had been tested when depressed, after recovery. Since clinical diagnosis was the selection criterion for the depressed group, it was considered necessary that the same criterion (i.e. clinical diagnosis) should also be applied to the selection of the recovered depressed patients. Thus a patient participated as a subject in this group if, in the opinion of the senior clinical member of staff in charge of the patient's treatment, he or she had completely recovered from his or her depressive illness. Some patients were seen as soon as possible after discharge, though the majority were seen just before (one to two days) their discharge. Of the twenty patients seen when depressed, only fifteen were seen on recovery. Of the five that were not seen again, two could not be re-tested because of domestic factors (they immediately left the area on discharge), and three had not recovered at the time of closure of the project, one being considered still ill (though less severely) over one year after the original testing.

(3) Anxious control group (Anxious)

As for the selection of depressed and recovered depressed patients, anxious

patients were allocated to this group on the advice and diagnosis of one or more of the MRC senior clinical members of staff. In addition, four patients from the Professorial Unit diagnosed as clinically anxious by the professorial team were included in this group. All the patients were diagnosed as suffering from generalised, free-floating anxiety, requiring clinical intervention. Some of these patients had phobic symptoms in addition to generalised anxiety. They had all either just started treatment or were about to commence treatment for anxiety neurosis, with tranquillising drugs and/or relaxation therapy. All subjects were considered highly anxious at the time of testing. Seventeen suitable patients were obtained for this group.

It was considered that this control group would offer a stringent test of the specificity of certain of the cognitive and attentional factors examined in depression.

(4) Normal control group (Normal)

This group consisted of normal subjects with no past psychiatric history. It consisted mostly of hospital and university personnel (e.g. nurses, MRC technicians, secretaries, occupational therapists, cleaners, pharmacology students, etc.). Any person who had any knowledge of the study was excluded. All subjects, except one, who had been approached by the author to take part in this study, gave their consent. Twenty subjects took part in this group.

(5) Age

The mean scores and group statistics of the four groups on the differences in age are given in tables 5.1 - 5.4. Because part of the study used a follow-up

design (depressed group and recovered depressed group), the data have been analysed separately for the depressed versus anxious versus normal group, and recovered depressed versus anxious versus normal group. The analysis of the difference in age scores between the groups uses the one way analysis of variance followed by the t test. This was the method used for the analysis of differences between the groups on the parameters measured in this experiment and is explained in more detail in the analysis of data section.

The mean scores and group statistics for the depressed versus anxious versus normal group data are shown in table 5.1.

Table 5.1 Age scores

Groups	(n)	Range	Mean	S.D.	
Dep.	(20)	18-65	45.6	14.08	$F = 4.664$
Anxious	(17)	22-57	33.7	10.43	$p < 0.05$
Normal	(20)	23-58	38.9	10.41	

Table 5.2 shows the results of the t-test applied to the individual bi-group comparisons.

Table 5.2 Differences in age

Comparisons	diff. between Means	SE diff.	t	p	df
Dep. v Normal	6.7	3.7	1.79	NS	38
Dep. v Anxious	11.8	3.91	3.03	0.005	35
Normal v Anxious	5.13	3.91	1.3	NS	35

(Two tailed test)

The mean scores and group statistics for the recovered depressed versus anxious versus normal groups are shown in table 5.3.

Table 5.3 Age scores

Group	(n)	Range	Mean	S.D.	
R.Dep.	(15)	19-65	46.4	13.7	F = 4.876
Anxious	(17)	22-57	33.7	10.43	$p < 0.05$
Normal	(20)	23-58	38.9	10.41	df = 49

Table 5.4 shows the results of the t-test applied to the individual bi-group comparisons.

Table 5.4 Differences in age

Comparisons	diff. between Means	SE diff.	t	p	df
R.Dep. v Normal	7.9	3.62	2.18	0.05	33
R.Dep. v Anxious	12.7	3.78	3.36	0.01	30

(two tailed test)

The results in tables 5.1 - 5.4 show that the depressed patients were not significantly different in age distribution from the normals but were significantly older than the anxious subjects. The fifteen recovered patients were found to be significantly older than the anxious patients ($p < .01$) and significantly older than the normal controls ($p < 0.05$).

(6) Sex

The number of male and female subjects in each group are shown in table 5.5.

Table 5.5 Sex differences

Groups	Male	Female
Dep.	12	8
R. Dep.	10	5
Anxious	10	7
Normal	9	11

$\chi^2 = 1.86$ p = NS df = 3

There were no significant differences found between the pattern of distribution of males and females in each group.

B. MEASURES

The perceptual and attentional parameters were measured in two ways, (1) by an analysis of electrodermal activity monitored through an experimental session of problem solving and (2) by measuring perceptual sensitivity thresholds to lights that had been previously paired with positive and negative events.

(1) Perceptual and attentional measures

(i) Psychophysiological

During a problem solving task in which electrodermal activity was continuously monitored, subjects were presented with ten solvable and ten unsolvable questions. Twenty seconds was allowed for the solution of the problem. A correct answer was immediately followed by the illumination of a light, four feet in front of the subject, which was situated slightly to the subject's left. This light remained on for five seconds. A failure to solve a question resulted in the illumination of a light, in the same horizontal plane as the left light but slightly to the subject's right, this

light also remained on for five seconds. This procedure provided nine elements of information which were considered relevant:

(a) Electrodermal activity was analysed in the five seconds immediately before the illumination of the left (indicating success) light. This unit of data from now on will be referred to as "pre-left activity", i.e. PREL.

(b) Electrodermal activity was analysed in the five seconds immediately before the illumination of the right (indicating failure) light. This unit of data from now will be referred to as "pre-right activity", i.e. PRER.

(c) Since both measures may be considered to offer some index of anticipatory responding to either the left light or right light presentation, it is possible to obtain a measure of bias of such responding by subtracting PRER from PREL, i.e. PREL - PRER. A positive figure would thus indicate a positive bias and a negative figure a negative bias.

(d) Electrodermal activity was analysed during the five seconds the left was actually on, following the successful completion of a question. This unit of data from now on will be referred to as "during left light activity", i.e. DUL.

(e) Electrodermal activity was analysed during the five seconds the right light was actually on, following the failure to solve a question. This unit of data from now on will be referred to as "during right light activity", i.e. DUR.

(f) Since both measures may be considered to offer some index of

responding to signalled success and failure events, it is possible to obtain a measure of bias in this responding by subtracting DUR from DUL, i.e. DUL - DUR. Again a positive figure would indicate positive bias and a negative figure a negative bias.

(g) Adding the two five-second epochs of each left light presentation (PREL + DUL) provides a combined activity measure to the left light over the measured epochs. This unit of data from now on is referred to as total measured activity to left light, i.e. TL.

(h) Similarly, adding the two five-second epochs of each right light presentation (PRER + DUR) provided a combined activity measure to the right light over the measured epochs. This unit of data from now on is referred to as total measured activity to the right light, i.e. TR.

(i) As before, both measures can be subtracted (TL - TR) to give a measure of bias for the combined left light and right light electrodermal activity data.

(ii) Perceptual sensitivity thresholds

In the subsequent part of the experiment, the perceptual and attentional sensitivity thresholds were measured to the left light (which had signalled success) and the right light (which had signalled failure). The subject was requested to observe the lights in front of him and to make a response when he could see that either the left light or the right light had come on. The luminosity of the lights was varied from below the subject's visual threshold to above it and the subject made his response as soon as he/she was aware

that either light was on. This procedure provided three elements of information:

(a) A perceptual sensitivity threshold for the detection of the left light (which had previously been paired with, and had earlier acted as the signal for success). This unit of data will be referred to as SENL.

(b) A perceptual sensitivity threshold for the detection of the right light (which had previously been paired with, and had earlier acted as the signal for failure). This unit of data will be referred to as SENR.

(c) A measure of bias between these two sensitivity thresholds was obtained by subtracting the sensitivity threshold of the left light from the sensitivity threshold of the right light. This subtraction meant that a positive score could be taken to indicate that the sensitivity threshold to the left light was lower than the sensitivity to the right light, i.e. there was a lower perceptual threshold for a stimulus that had been paired with (positive) success events than with (negative) failure events. A negative score would mean the converse. This unit of data will be referred to as SEN. diff.

(2) Measures of attitudes and other cognitive factors

(i) Perceived locus of control

The attitude towards perceived control over reinforcers was measured by the internal external scale devised by Rotter (1966). This self-report scale consists of twenty-nine items, with each item having two sub-statements. The subject is asked to circle the letter next to the sub-statement that he believes to be most true. For example:

6. (a) Without the right luck one cannot become an effective leader.

(b) Capable people who fail to become leaders have not taken advantage of their opportunities.

If the subject circles the sub-statement indicating that he believes certain external factors are necessary for advancement (in this case sub-statement (a)), he receives a score of one. Circling sub-statement (b) would receive no score, thus the higher the score, the higher is an individual's externality score, and the greater his belief in a lack of control.

The overall reliability of this scale has been found to vary between 0.49 and 0.85 (Peck and Whitlow, 1975). Rotter (1975) has estimated that this scale has been used in over 600 published studies and that there is still a great deal of interest in it. However there is some debate about what this scale actually measures. As pointed out earlier, Weiner (1972) has argued that it confounds the dimensions of causality. Lefcourt (1976) has argued that perceived internal control is positively related with access to opportunity. Those who are able, through position, to have access to valued outcomes are more likely to hold internal expectancies, as compared to minority racial and poorer groups who are more likely to have external expectancies. In addition, factor analytic studies tend to show that the I-E scale measures more than one control factor. For example Gurin et al. (1969) found four factors, control ideology (which is the general control held by most people in society), personal control (which is the control one personally has), systems modifiability (which is the extent to which society's problems can be overcome) and race ideology (which contains items that refer to race). Although other factor analytic studies have not found the same factors, there is some

agreement that the I-E scale is generally concerned with the two factors of personal control and system's modifiability.

In spite of these possible problems of interpretation, various studies have used this scale on psychiatric populations. Harrow and Ferrante (1969) examined the relationship between locus of control and various psychiatric disorders. Manics were found to be the most internal and schizophrenics the most external. On recovery manic patients became more external and the schizophrenics remained relatively external. The largest change in scores wa obtained by the depressives who moved from an external position when ill to a more internal position when recovered. The I-E scale has also been found to relate to depth of psychopathology. Shybut (1968) and Palmer (1971) found that psychiatric patients in general were more external than normal controls. He also found a relationship between a scale that measured competence, derived from education, marital status and occupational level, and the I-E scale. In general the more competent the patient, the more internal he tended to be, (Palmer, op. cit.).

Two other studies have found the opposite relationship between psychopathology and locus of control. Goss and Morosko (1970) found that alcoholics scored in a more internal direction than did the normative sample reported by Rotter (1966). However, within this group externality correlated with anxiety, depression and other psychopathological characteristics (as assessed by the M.M.P.I.). Berzins and Ross (1973) found a similar relationship in a drug addicted group of hospitalised patients. These authors attempted to explain their results in terms of an effort of self

control by these patients via drug use over their anxieties, depression, internal conflicts and so forth.

In addition to these clinical studies, research on depressed students has also found significant positive correlations between depressed mood and the I-E scale (Abramowitz, 1969, Warehime and Woodson, 1971, Calhoun et al., 1974, Prociuk et al., 1976). Calhoun et al. (op. cit.) found that women, in contrast to men, showed an unreliable relationship between depressed mood and I-E but showed a positive correlation between accepting personal responsibility for depression and depressed mood. Procuik et al. (op. cit.) also found a positive relationship between I-E and depressed mood in students. In addition they found a positive correlation between hopelessness (as measured on the Beck's et al. (1974) hopelessness scale) and I-E.

Thus, in general, although most studies have found a significant correlation between depression and externality, this relationship seems to hold for other negative affects. Lefcourt (1976) presented a summary of nineteen correlational studies in which thirteen found a weak but significantly positive correlation between various measures of anxiety and externality. In fact, four studies conducted by Ray and Kutahn (1968) found correlations of between 0.13 and 0.36 with test anxiety, though only three of the correlations reached significance at the 0.05 level.

For reasons discussed in chapter 4, pp. 114-115 , the distinction between the external versus internal predictions of Seligman's and Beck's theories of depression cannot be measured by this scale when the subjects

are ill. However, given Beck's arguments concerning the question of predisposition to depression and Harrow and Ferrante's (1969) finding of the change in depressives' I-E scores on recovery, one factor that requires examination is how a locus of control measure differentiates recovered depressed and normal controls.

(ii) Attitudes to test and assessment situations and fear of failure

Attitudes to test and assessment situations were measured with the Test Anxiety Scale (T.A.S.) (Sarason, 1972). The T.A.S. has a true/false format and was developed from the earlier Test Anxiety Questionnaire (T.A.Q.) (Mandler and Sarason, 1952). The T.A.Q. was found to have a test-retest reliability of 0.91 (Mandler and Cowan, 1958). The shorter version of the T.A.S. is taken directly from the T.A.Q. (Sarason, 1958). The scale used here is the longer version (37 items) of the T.A.S. (Sarason, 1972). Examples of the items that appear on this scale are:

17. I seem to defeat myself while working on important tests — True/False

31. I don't enjoy eating before an important test — True/False

This scale was primarily designed to assess test and assessment anxiety in academic situations. For this study this orientation was found to be too specific on certain questions. As a result some of the questions were slightly modified. This was carried out by removing some of the emphasis on narrow academic situations. For example question 21 was rewritten from:

21. I would rather write a paper than take an examination for my grade in a course True/False

to

I would rather show my abilities in other ways than by taking written tests True/False

The original and the slightly adapted versions of this scale are presented in the appendix. It was considered that although the phrasing of some of the questions needed to be changed, this did not detract from the overall aims and validity of the scale.

Sarason (1960, 1972) has pointed out that although the concepts of test anxiety and general anxiety overlap, it is not sufficient to make the two concepts synonymous. A correlation of 0.53 was reported between the Taylor Manifest Anxiety Scale and the early test anxiety measure, the T.A.Q., by Raphelson (1953).

As to what the test anxiety measures, there seems to be general agreement that it is the tendency to emit negative interfering responses or cognitions under assessment conditions. Mandler and Sarason (1952) argued that high test anxious individuals have a tendency to respond to test and assessment situations with feelings of hopelessness and inadequacy together with anticipations of loss of status and consequent punishment. A similar view is expressed by Liebert and Morris (1967) and Sarason (1972). Others have expressed a more cognitive view of test anxiety, arguing that high test anxiety produces cognitions such as "I'm stupid","maybe I won't pass", etc. (Doctor and Altman, 1968, Phares, 1968). This view seems in line

with Meichenbaum's (1977) internal dialogue view of test anxiety. It has also been suggested (Wine, 1971) that highly test anxious individuals direct their attention to external interfering characteristics rather than the task at hand.

All these interpretations have significant implications for a cognitive theory of depression since if Beck is correct in his contention that depressives tend to self devalue and are more negative in their allocation of attention, then they should have a high test anxiety score. Moreover, because test anxiety has been interpreted in a way which indicates that the subject is more concerned with the negative consequences of failing than the positive outcome of success, it has been linked with the fear of failure concept. In fact the T.A.S. and T.A.Q. have been the measures most used to determine the strength of fear of failure (Weiner, 1972, Weinstein, 1969, Birney et al., 1969). The results of this work, primarily using the T.A.Q. in achievement motivation studies, have been neatly presented by Weiner (1972, p. 203) and are given in table 5.6.

Table 5.6

Resultant achievement motivation classification as a function of hope of success (generally assessed with T.A.T.) and strength of the fear of failure (generally assessed with T.A.Q.)		
Hope of success (Need for achievement)	Fear of failure (Anxiety)	Resultant achievement Motivation
High	Low	High
High	High	Intermediate
Low	Low	Intermediate
Low	High	Low

Although the T.A.Q. has been used extensively as a measure of fear of failure some important criticisms have been put forward against it (Birney et al., 1969, Weiner, 1972). Moreover, Weinstein (1969) found low correlations between different projective and questionnaire measures of need for achievement and fear of failure, and concluded that many of the measures did not assess the same thing. Furthermore, the use of the T.A.S. in this study as a measure of fear of failure is totally dependent on its high correlation with the T.A.Q.

Nevertheless, in spite of these problems, test anxiety has been the most consistently used measure of fear of failure. Moreover, from the approach that has been developed here, the concept of test anxiety (with the hypothesis put forward by many workers that high test anxiety is the proneness to emit interfering negative responses and self statements) stands by itself as being an important concept to examine within Beck's theory of depression.

iii. Negative thought content

The parameter of negative thinking was measured by administering the Beck Story Completion Test (B.S.C.T.). This questionnaire was obtained by writing directly to its author. Weintraub et al. (1974) argued that the story completion test was

"derived as a projective index of the presence of depressive cognitive content" (P. 911)

These authors hypothesised that the depressive affective state would be closely related to a dimension they labelled as cognitive style. This dimension consisted of four sub-sections, (1) expectation of discomfort, (2) expectation of failure, (3) negative perception of interpersonal relationships, and (4) negative perception of the self. In this study, however, it was considered more appropriate to analyse the combined scores generated by these sub-sections as a total negative thought content score, rather than by independent analysis. This procedure was considered valid in view of Beck's theory of depression and because Weintraub et al. (1974) found significant correlations between all the sub-sections and depression. Their work was, however, conducted on a normal population. An investigation of the relationship between the sub-sections of this scale in the groups taking part in the study was considered outside the main aims of the study.

Beck (1976) provided the author with two alternative story completion tests, both consisting of five stories. In this study the two alternative tests were combined, producing a total of ten stories. On this test the subject is required to read each story and circle the number in each sub-section

(A-D) that best describes how he imagines the situation. An example of the stories used, with the response alternatives, in each sub-section, are given below.

Paragraph: In our school athletics team, a big event was always the fencing matches. Once I was in the team in the championships. This was to be my first time fencing in a team championship. We practised especially hard before the event because we really wanted to win.

Group A

1. I was confident that I'd perform well.
2. It would have surprised me if we hadn't won, we were all too good
3. I knew beforehand that our team was relatively weak.
4. I had a sinking feeling that I would fail the team.

Group B

1. We were decisively beaten.
2. The team won the championships.
3. The team lost to another school.
4. For the third year in succession, we won.

Group C

1. The other team-members went out of their way to give me encouragement and practice before my first big match.
2. Some of my team-mates were hesitant to help me practise for the event.
3. The other members wished me good luck before the match.

4. I didn't get along with the other members in the team and practice was a pretty lonely process.

Group D

1. I was not one of the top members of our team.
2. My poor performance convinced me to give up fencing.
3. I was an outstanding fencer.
4. I was proud to be a member of the winning team.

Each story generated four scores between 0 and 3. The higher the score the more negative the thought content. The maximum negative thinking score from any one story was 12, making a maximum score over the ten stories of 120.

(iv) Performance evaluations

In addition to these various self-report measures designed to examine specific attitudes, measures of performance evaluation and appraisal on a problem solving task were also obtained. These measures arose from questions asked of a subject after a test session which included a problem solving task. The questions concerned the evaluation of three performance scores, (1) the evaluation of how the subject had actually performed, (2) the evaluation of the subject's ability to improve his score if he were to try the task again, and (3) the evaluation of his aspiration level, i.e. what sort of score he would be pleased with.

In addition to measures of perceptual, attentional and attitudinal parameters, general measures concerned with the depth of depression and the

degree of anxiety were also measured. Moreover general background electrodermal activity was also analysed during the experimental session. These are described in the next section.

(3) General and Background measures

(i) Beck Depression Inventory

Depth of depression was measured by the Beck Depression Inventory (Beck et al., 1961). This is a self-rating scale which consists of a series of statements grouped in twenty-one sections from A to U. Each section consists of between four and six items in a graded form and the subject is asked to circle the number next to the item that most applies to him or her. For example,

A. 0. I do not feel sad

1. I feel blue or sad

2a. I am blue or sad all the time and I can't snap out of it.

2b. I am so sad or unhappy that it is very painful.

3. I am so sad or unhappy that I can't stand it.

Depth of depression is measured as a cumulative score of the items circled. The number next to the item gives the score to be counted. Normative data for this scale have been provided by Beck et al. (1961) and Metcalfe and Goldman (1965). See table 5.7.

Table 5.7

Norms for the Beck Depression Inventory

Depth of Depression	American Study		British Study	
	Mean	S.D.	Mean	S.D.
None	10.9	8.1	5.4	5.8
Mild	18.7	10.2	14.3	8.3
Moderate	25.4	9.6	24.2	10.8
Severe	30.0	10.4	29.5	6.5

The level of scoring is lower in the British study than the American, more marked in the none to mild categories than in the moderate to severe. Metcalfe and Goldman (1965) believe that this may be due to cultural differences.

The Beck Depression Inventory (B.D.I.) has been used in numerous studies on depression, often as the major independent measure of depression Its validity, as assessed by the degree of concordance with doctors' ratings, has been found to be between 0.65 and 0.67 in the American study (Beck et al., op cit.) and 0.61 in the British study (Metcalfe and Goldman, op. cit.) In addition correlations between an observer rating scale (the Hamilton Rating Scale of Depression) and the B.D.I. have been reported as 0.75 (Schwab et al., 1967) and 0.82 (Williams et al., 1972).

Although the B.D.I. is considered a very useful and reliable scale, some have argued that the stage of illness may affect the concordance between the B.D.I. and other observer rating scales such as the Hamilton. It is argued that the acute phase of a depressive illness may distort self

ratings of depression and lower the correlation between the B.D.I. and observer rating scales. As the illness and the distorting effects of the illness recede, the concordance between the different types of rating may increase (Carroll et al., 1973, Paykel et al., 1973, Prusoff, et al., 1972, Donovan and O'Leary, 1976).

(ii) General Anxiety Scale

The level of General Anxiety was measured with the General Anxiety Scale (Sarason, 1972). This is a seventeen item scale with a true/false format. Examples of items on this scale are

3) I worry about my social adjustment more than most people do True/False

13) I am inclined to take things hard True/False

Sarason (1958) presented a slightly longer version of this scale as being primarily derived from items on the Taylor Manifest Anxiety Scale (Taylor, 1953) and items on the M.M.P.I. Thus this scale is intimately related to well established anxiety measures. The original General Anxiety Scale (Sarason, 1958) was designed to assess:

". . . the extent to which the individual experiences anxiety in a wide range of situations other than testing." (P. 340)

It is arguable that a more comprehensive anxiety scale may have been desirable. However, the version of the General Anxiety Scale used in this study was presented as an appropriate anxiety scale in studies also examining Test Anxiety (Sarason, 1972). Moreover, Sarason (1958) has used the

General Anxiety Scale with a psychiatric population and found a correlation of 0.46 between the original true/false format of the T.A.S. (Sarason, 1958) and the General Anxiety Scale. The test-retest reliability in this study of the General Anxiety Scale (G.A.S.) was found to be above 0.78.

Two additional considerations influenced the decision to use this particular scale. Firstly this study was primarily concerned with attentional, perceptual and attitudinal parameters in depression and an anxiety scale was only required to give a general background measure. Secondly, and more importantly, subjects were required to spend between $1^1/_2$ and 2 hours completing all parts of the experiment including the completion of the questionnaires, thus the brevity of the G.A.S. was an asset in this case, albeit at the sacrifice of comprehensiveness. Even with this short scale, some depressed patients took $2^1/_2$ to 3 hours completing all parts of the experiment.

All scales and measures used in this study appear in full in the appendix

(iii) Background electrodermal activity

Although not part of the main aims of this study, the general background electrodermal activity of each subject was analysed. For reasons explaine in the 'analysis of data' section, the measure of background electrodermal activity was taken as the mean number of spontaneous fluctuations per minute occurring in three experimental periods. This provides the following three measures:

(a) The mean number of spontaneous fluctuations per minute occurring during the completion of the T.A.S. This unit will be referred to as

SFTAS.

(b) The mean number of spontaneous fluctuations per minute occurring during the problem solving task. This unit of data will be referred to as SFPS.

(c) The mean number of spontaneous fluctuations per minute occurring during the light detection (vigilance) part of the experiment. This unit of data will be referred to as vigilance electrodermal activity and will be written as SFVI.

C. TESTING PROCEDURE

(1) When a subject was considered suitable for testing he/she was brought to the psychophysiological laboratory in the MRC Metabolic Ward. The subject was seated just outside the experimental cubicle in which the testing was to take place. The procedure of the experiment was briefly described to the subject during which time either the author or the psychophysiological technician attached skin resistance recording electrodes. When this was completed, the subject was taken into the experimental cubicle and was seated in a comfortable chair facing two lights, one to the subject's left, the other to the subject's right. Once the subject had settled in the chair, the polygraph trace was started. The experimenter then left the cubicle for approximately five minutes and re-entered the cubicle again when the trace of the electrodermal activity appeared reasonably stable. The subject was able to remain in contact with the experimenter at all times via the use of a room to room intercommunication system.

At the end of a five minute rest period, the experimenter re-entered the cubicle and asked the subject to complete the T.A.S. A board was placed across the arms of the chair to provide the subject with a writing surface. The experimenter then left the cubicle whilst the subject completed the T.A.S. The beginning and completion of the T.A.S. was marked on the polygraph trace.

(2) When the T.A.S. was completed the experimenter re-entered the experimental cubicle, removed the T.A.S. and provided the subject with a blank answer sheet. The experimenter then seated himself on the subject's left and notified him that the experiment would now begin. He then read to the subject the following instructions:

"This test consists of two parts. In the first part of the test I am going to ask you to try to solve twenty questions, and you should mark your answers down on the blank answer sheet you have been provided with. I will explain the second part of the experiment when you have finished answering the questions.

These questions will be presented on cards. On each card there is a series of letters. These letters follow some definite rule. The idea is for you to examine each series of letters carefully to discover the rule and then write down what you think the next letter in the series should be. Here are some examples to give you an idea of what you have to do."

The experimenter then presented five simple examples and asked the

subject to give the answer to each. When the experimenter was satisfied that the subject understood the task he proceeded with the instructions:

"There are twenty cards in all and you can spend twenty seconds with each card. If you do not know the answer after this time, I will pass on to the next card.

If you obtain the correct answer you will see the left light come on for five seconds. This light means you have got the correct answer and counts as a success. If you run out of time or if your answer is incorrect, you will see the right light come on for five seconds. This light means your answer is wrong and counts as a failure.

Do you understand the instructions?"

When the experimenter was satisfied that the subject understood the instructions he notified the psychophysiological technician that the experiment was about to begin. The first card was then presented.

During the course of the session, when the subject wrote down a correct answer he was immediately presented with the left light. At the end of the twenty seconds or if the subject got the answer wrong, he/she was immediately presented with the right light.

When all the twenty cards had been presented in this manner, the experimenter removed the subject's answer sheet and read the second set of instructions.

"We are now ready to begin the second part of the experiment. In this part I am going to place a board across the arms of your chair (this was then done). As you can see there are two buttons mounted on the right hand side

of the board which you can easily operate with your right hand.

This part of the test is concerned with how sensitive you are to low intensity lights. I would like you to watch these two lights in front of you (the same lights that came on when you either succeeded or failed at the previous questions), closely. The luminosity of these lights will be varied. You will not be able to see the light when it is first on because it will not be bright enough, but the brightness will slowly increase, and we would like you to press a button as soon as the light just becomes visible to you. When the left light just becomes visible to you, press the left button, when the right light just becomes visible, press the right button. As soon as you have made a response, the next trial will begin. It will always be either the left light or the right light that will come on. In other words, these lights will always come on separately, never together. I will present you with a practice light first to make sure you have the idea. O.K. ?"

The experimenter then left the cubicle and commenced the last part of the experiment. Each subject received ten left lights to detect and ten right lights. These detection trials were randomly allocated at the beginning of the project and each subject saw the same random allocation; while the detection task was going on, electrodermal activity was continuously recorded.

(3) At the end of the twenty light detections, the experimenter re-entered the cubicle and informed the subject that the experiment was now at an end. Before

leaving the cubicle, however, the experimenter asked the subject three questions:

"I would like you to think over the time when you were answering the questions on the cards, the cards with the letters on them. On that task, how many of the twenty questions do you think you actually got correct?" Secondly, "if you were to do the questions again now, how many questions do you think you would get correct this time?" And lastly, "on a test like that, how many correct answers would you be pleased and satisfied with?"

At the end of this test session the psychophysiological electrodes were removed and the subject was taken to a different room and presented with the BSCT, BDI, IE and GAS questionnaires. Occasionally subjects were not able to spend more time at the hospital due to domestic or employment commitments. In these cases the subjects were given stamped addressed envelopes and requested to return the completed questionnaires as soon as possible. Although this was an undesirable procedure, it was unavoidable in some cases. However, the majority of subjects completed their questionnaires immediately after testing.

D. CONDITIONS OF TESTING

Patients allocated to the two ill groups (depressed and anxious) and the recovered depressed group were tested as soon as possible following notification of the suitability of a patient. Since one of the measures used was electrodermal activity, all patients were required to be drug free for at least seventy-two hours before being tested. Advice on the amount of time each patient needed to abstain

from medication for valid psychophysiological measures to be taken was given by Dr. G. Ashcroft, the Unit Director. Seventy-two hours was considered to be the minimum acceptable period.

One unfortunate but unavoidable consequence of this requirement was that it acted as a limiting factor in the selection and availability of patients. In depressed patients with a long psychiatric history who were receiving chemotherapy, it was sometimes considered unethical and potentially dangerous to curtail medication suddenly as this could lead to unpleasant side effects such as nausea and dizziness. In such cases, patients are normally weaned off their drugs slowly. Thus if the withdrawal of medication for a few days was not considered to be in the best interests of the patient, the patient was considered unsuitable for inclusion in this study. The same problem arose in the selection of patients for the anxious group where drugs had been an important method of treatment. Although all the depressed patients who were off drugs or agreed to come off drugs gave their consent to take part in this study, three potential anxious group subjects refused to curtail their medication, and two believed they were too anxious to take part.

In the depressed group, patients who were about to start a course of electric convulsive therapy (E.C.T.) had sometimes had their medication witheld beforehand. Some of these patients were considered suitable for this study. However, one such patient was excluded because she was considered to be too deluded and retarded to take part. Thus the demand characteristics of the experiment tended to exclude psychotically or extremely retarded depressed subjects. Nevertheless the majority of the depressed subjects tested were considered to be moderately to

severely depressed at the time of testing.

E. APPARATUS

The experiment was conducted in the MRC psychophysiological laboratory. Within the laboratory is housed a sound-proof testing cubicle (internal measurements 8 ft by 5 ft). During the period of time the experiment was in progress the subject was seated in a comfortable chair at one end of the cubicle. The temperature was kept constant at 68°F for all subjects tested, controlled by an Atronicair thermostat heating unit, model TA 4464. The overall illumination of the room also remained constant using a Weston model V, universal light exposure meter. The psychophysiological recording equipment and other necessary control apparatus was housed outside the sound-proof cubicle.

(1) Psychophysiological apparatus

The measurement of the electrodermal activity was carried out in accordance with the method of measurement laid down by Lader and Wing (1966). This method involved passing a small constant current (14 $\mu a/Cm^2$) through the skin and recording its resistance on a Grass polygraph drive amplifier with pen trace recorders. The model used in this experiment was the Grass polygraph model 7. This method provided a continuous ink pen trace recording of electrodermal activity throughout the course of the experimental procedure.

This method of recording involves the use of two, double element, electrodes. One of these electrodes is placed on an inactive electrodermal site, the other on an active site. Here, as described by Lader and Wing (1966), the inactive site was the abraded surface of the lateral aspect of the left arm, just above the

elbow. The active site was the distal segment of the left thumb, upon which an annular ~~from~~ corn plaster (i.d. 9.5 mm) was placed to act as a masking device (Lader and Wing, op. cit.). The electrode jelly which acted as the contact medium between the surface of the skin and the electrode was K-Y jelly, the recommended contact medium for this system of electrodermal recording (Murray, 1976).

The elements of the electrodes were made of lead which avoids the problems of polarisation (Lader and Wing, op. cit.).

(2) Additional apparatus

In addition to recording electrodermal activity the grass polygraph had the facility of being able to mark and differentiate, on the pen trace, two stimulus events. This facility was used in both parts of the experiment. Two domestic push button controls were mounted on a board two inches apart and connected to the stimulus marker system of the polygraph. During the problem solving part of the experiment, these buttons were operated by the experimenter to indicate and mark, on the pen trace, the successes and failures of the subject (one button made a mark just above the time constant output recording, the other made a mark just below it). By observing the trace, the psychophysiological technician was able to discern whether the subject had achieved a correct answer or an incorrect answer according to the mark made on the trace by the experimenter using the button system. According to which mark appeared on the trace, the psychophysiological technician switched on either the left light for five seconds or the right light for five seconds.

During the light detection part of the experiment, the board on which the

stimulus markers were mounted was placed across the arms of the subject's chair. These buttons were then operated by the subject according to whether he or she saw a left or right light come on. By observing the pen trace, it was possible to tell which light had been detected and at what moment.

The control panel of the lights themselves allowed for each light to be turned on and off independently. During the problem-solving part of the experiment, the psychophysiological technician operated these lights according to the appropriate mark that appeared on the pen trace.

The illuminosity of the lights could also be independently varied by the use of two variable transformers which were wired in series between the power source and the on/off control panel of the lights. These two transformers allowed for very small changes to be made in the luminosity of the lights. The voltage of the circuit at any particular time was measured by an avometer connected in the circuit. Thus a reading of the number of volts required to generate a correct detection was the measure used for the subject's sensitivity thresholds to the lights. The avometer supplied the sensitivity level for the last (light detection) part of the experiment.

(3) Problem solving material

The test material used during the problem solving part of the experiment was designed by the author. It consisted of twenty cards. On each card a series of five letters had been printed. Ten of these cards had series which followed some simple rule, e.g.

Card no. 7) T. U. V. W. X.

17) R. S. R. S. R.

The other ten cards had series which did not follow any particular rule, they were thus unsolvable, e.g.

Card no. 3) F. G. L. N. S.

20) D. E. B. A. F.

The subject was led to believe that all questions were solvable, but the test was designed so that everyone achieved ten and only ten correct answers.

The complete sequence of cards used in this experiment is presented in the appendix.

The time allowed for the solution of each card (twenty seconds) was measured by the experimenter with a stop-watch.

F. ANALYSIS OF THE DATA

(1) Psychophysiological data

Electrodermal activity is the activity determined by the electrical properties of the skin. These properties appear to change according to the psychological status of the person. The actual workings of this system are quite complex and still not fully understood. However, it is known that the electrical activity of the skin on the volar surfaces depends on eccrine sweat gland activity, and it is only from these surfaces (the palms of the hand and soles of the feet) that electrodermal activity can be measured for any psychological relevance. Both the amount and the chemical composition of the sweat produced by these glands is important.

The myoepithelial lining (the muscle tissue surrounding the lumen of the sweat gland) is rhythmically activated by as yet some unknown mechanism in a manner which narrows the lumen of the lower

portion of the sweat gland so that the level of sweat in the tuble periodically rises and falls. Thus sweat is produced on the surface of the skin which then acts as a conductance to an electrical current (Lykken, 1968). This rhythmic activity of the myoepithelial lining appears to depend on some index of arousal, (Lykken, op. cit.). In any event the consequent changes in the electrical properties of the volar surfaces are generally regarded as being an index of arousal (Edelberg, 1972, Venables and Martin, 1967, Lykken, 1968, Lange, 1971).

Because of the great complexity in such measures (which cannot be further discussed here), psychophysiological measures, especially electrodermal activity require many arbitrary decisions in their analysis (Lader, 1975, 1977, Lykken, 1968). The first question that arises is how should the data be analysed. There are many alternatives as outlined by Edelberg (1972). For this experiment, the method chosen was the number of spontaneous fluctuations that occurred over a specified epoch. Although this is not a sophisticated analysis, it does have certain advantages and has been used in other studies (Silverman et al., 1959 , McDonald et al., 1964). It was considered that for this study a more sophisticated analysis of electrodermal activity would not generate more meaningful results on the parameters under investigation (Arbuthnott, 1977). In addition, studies of this kind should be cautious in their use of sophisticated analysis since many events are occurring to the subject (e.g. having the experimenter present problems, lights coming on and off, plus, occasionally, talking) thus, unlike habituation type experiments (Lader and Wing, 1966), it is not always possible to relate particular responses to particular events.

The criterion for counting spontaneous fluctuations was taken as a 1% change in background responding as suggested by Edelberg (1972) and Lader (1975). Lader (1977) pointed out that responses which immediately followed each other should only be counted as separate if there is a visible inflexion in the trace recording.

Thus the data analysed consisted of the number of spontaneous fluctuations over the specified epochs, that is the number of spontaneous fluctuations occurring in the PREL epoch, the PRER epoch, the DUL epoch, the DUR epoch and so on, for all the epochs measured, as previously outlined. The scores derived from this analysis constituted the total spontaneous fluctuation score for each specified epoch. Thus a score of PREL = 8, means that over the ten occasions on which the subject was presented with a (success) left light; the total number of spontaneous fluctuations (in the five seconds immediately before the light was presented) over the ten trials equals 8.

In addition, the mean number of spontaneous fluctuations per minute was taken as the background level of electrodermal activity during the T.A.S. completion, problem solving, and light detection part of the experiment. To avoid the problem of double counting, all the spontaneous fluctuations accounted for by the other specified epochs were excluded from the background score. The time of these epochs was also subtracted from the time taken up by the two experimental sessions, problem solving and light detection. In the light detection part of the experiment the two seconds before and the three seconds after a detection were also excluded from the background analysis (Arbuthnott, 1977).

(2) Perceptual sensitivity thresholds

The perceptual sensitivity threshold to the left light (for each subject) was taken as the mean number of volts (over the ten detections required), necessary to generate a correct detection.

The perceptual sensitivity threshold score to the right light was taken as the mean number of volts (over the ten detections required), necessary to generate a correct detection.

The difference of the means between these two thresholds was taken as the measure of bias in the detection of lights that had previously been paired with success and failure.

(3) Performance evaluations

The question concerning the subject's evaluations of his performance, ability to improve, and aspirations on the problem solving task, generated three units of data, called Judgement Discrepancy (J.D.), Expectance Discrepancy (E.D.) and Aspiration Discrepancy (A.D.). These scores were obtained in accordance with a slightly modified method of Inglis's (1960) scoring system. The system used here was: (- indicates a subtraction)

J.D. = The number of questions the subject thought he got correct - The number of correct answers he actually obtained

E.D. = The number of questions the subject thought he would get correct if he were to do the task again - The number the subject thought he had got correct the first time.

A.D. = The number of correct answers the subject would be pleased and satisfied with − The number of correct answers he actually obtained

(4) Statistical analysis

Two types of statistical analyses were conducted on the data generated by this experiment. Pearson product moment correlations were calculated between all variables measured. In addition, a one way analysis of variance (Guilford, 1956) was used to examine differences between groups. A significant F ratio was then followed by a student's 't' test which provided the intergroup variable significance. The 't' test, following the one way analysis of variance, used the total population or error variance of the groups compared. The formula for this statistical procedure is

$$t = \frac{\mu_1 - \mu_2}{\sqrt{\frac{\sigma^2}{n_1} + \frac{\sigma^2}{n_2}}}$$

μ_1 and μ_2 are the respective means of the two groups being compared, σ^2 is the total population (or error) variance and n_1 and n_2 the respective numbers in each group. The denominator of this equation is $\sqrt{\frac{\sigma^2}{n_1} + \frac{\sigma^2}{n_2}}$, known as the standard error of the difference between the means (Kerlinger, 1973, p. 206).

Because this study used a design which involved a follow-up and cross-sectional approach, there was a problem of interdependence between the depressed and recovered depressed groups. Consequently, to overcome this

problem, the one way analysis of variance was conducted in three blocks. The first block consisted of comparisons between the depressed, anxious and normal groups. The second block consisted of comparisons between the recovered depressed, anxious and normals groups. The third block consisted of paired comparisons between the recovered depressed and the depressed group (only the fifteen subjects who were seen again on recovery were used in the depressed group for this comparison). This analysis was conducted by calculating the change scores (Dep. - R.Dep.) and testing the hypothesis that the change scores were not significantly different from zero (i.e. no change).

This method of analysis uses the following formula to calculate 't':

$$t = \frac{\text{mean difference between the means}}{\text{standard error of the mean}}$$

$$\text{S.E.} = \frac{\text{S.D.}}{\sqrt{N}} \qquad (N = 15)$$

G. SPECIFIC HYPOTHESES

(1) Background and General

(i) It was predicted that the depressed group would have the significantly highest B.D.I. scores of all the groups.

(ii) It was predicted that the anxious group would have the significantly highest G.A. scores of all the groups.

(iii) No prediction was made concerning the background level of spontaneous fluctuations since variability is dependent on a number of factors such as retardation (Lader, 1975) and depressed patients have been found to have both higher and lower than normal spontaneous fluctuation activity (Lader, (op. cit.).

(2) Attentional and Perceptual

(i) It was hypothesised that on the measures used to examine the parameters of attentional and perceptual sensitivity to positive and negative events, the depressed group would show the greatest negative bias (see P. 145-148).

(ii) On recovery from depression it was hypothesised that the recovered depressives would lose some of the negative bias as might be shown on the above parameters, but would continue to have sufficient negative bias to distinguish them from normal controls (see P. 145-148).

(3) Attitudinal and Cognitive

(i) It was hypothesised that both the anxious and depressed groups would be significantly more external than the normal controls.

(ib) On recovery it was hypothesised that the depressed group would become significantly more internal than the normal controls.

(ii) It was hypothesised that both the anxious and depressed groups would have significantly higher test anxiety than normals. However, it was predicted that the depressed group would have the highest level of test anxiety.

(iib) On recovery from depression it was hypothesised that the recovered depressed group would have a lower test anxiety score than when ill, but would still be significantly higher than normal controls (see P. 152-155).

(iii) It was hypothesised that the depressed group should have the highest degree of negative thinking as measured by the B.S.C.T. (a scale specially designed for measuring negative depressive thought content).

(iiib) On recovery from depression it was hypothesised that the negative thinking scores would be decreased, although recovered depressives would still think more negatively than normal controls.

(iv) It was hypothesised that the depressed group, on the problem solving task, would (a) underestimate their performance (thus having the highest negative Judgement Discrepancy Score), (b) be the least likely to believe they could improve their scores (thus having the lowest positive Expectancy Discrepancy Scores) and (c) would have a higher level of aspiration (thus having the highest positive Aspiration score).

(ivb) It was hypothesised that the recovered depressed group would retain some tendency to underestimate their performance and ability to improve, and would continue to set higher standards of excellence for themselves than the two control groups. Moreover it was hypothesised that these tendencies would be strong enough to differentiate significantly the recovered depressed groups from the normal control group.

CHAPTER 6

RESULTS

The following results consist of two parts: (1) within group analyses and (2) between group analyses.

WITHIN GROUP ANALYSES

This section reports: firstly, the intercorrelations between all the variables within groups independently, i.e. the depressed group when ill and after recovery, the anxious group and the normal group; secondly, change scores were calculated (Dep.-R.Dep.) for the patients who were seen twice (before and after recovery from a depressive illness) and correlations between the change scores derived.

BETWEEN GROUP ANALYSES

A. Comparison of Depressed v Anxious v Normal groups.

A one-way analysis of variance followed by t tests where the F test was significant was applied to all variables for comparing differences between the Depressed, Anxious and Normal groups.

B. Comparison of Recovered Depressed v Anxious v Normal groups.

Similarly, a one-way analysis of variance followed by t tests where indicated was applied to all variables for comparing the differences between the depressed patients seen again on recovery, the Anxious and Normal groups.

C. Comparison of Depressed patients when ill and after their recovery.

't' tests were applied to the calculated change scores (Dep.-R.Dep.); the change score was tested against zero, i.e. no change.

All results are presented under three sub-headings: (1) General and background, (2) Attentional and perceptual, (3) Attitudinal and cognitive.

I. WITHIN GROUP ANALYSIS

Pearson's product-moment correlation coefficients were calculated to investigate the inter-relationship between variables.

Each table shows only the significant correlations (with level of significance) for the variables under each sub-heading, e.g. (1) General and background, etc. The groups are examined in the order of (A) Depressed group intercorrelations, (B) Recovered Depressed group intercorrelations, (C) Anxious group intercorrelations (D) Normal group intercorrelations.

A. Depressed group intercorrelations

(1) General and background

Table 6.1 shows the significant correlations for the general and background variables in the depressed group.

Table 6.1 General and background significant correlations

Age	BSCT -0.52 *	PREL -0.50 *	SFAS -0.46 *	TL -0.45 *
BDI	None			
GA	TAS 0.57 **			
SFTAS	AD 0.46 *			
SFPS	AD 0.47 *	Age -0.46 *		
SFVI	None			

* $p < 0.05$ ** $p < 0.01$ $n = 20$ $df = 19$

Table 6.1 shows that age was negatively correlated with negative thinking (BSCT), the anticipatory electrodermal activity to the left (success) light (PREL), the background electrodermal activity during the problem solving task (SFPS), and the total measured electrodermal activity to the left (success) light (TL). All these correlations were at the 5% level.

Self rated depression as measured by the Beck Depression Inventory (BDI) did not correlate with any other variable. General anxiety (GA), however, correlated with test anxiety (TAS) at the 1% level.

The background electrodermal measures showed that both the mean number of spontaneous fluctuations during the completion of the test anxiety scale (SFTAS) and during the problem solving task (SFPS) correlated with aspiration discrepancy (AD). In addition, as mentioned earlier, SFPS correlated with age. All these background electrodermal correlations were significant at the 5% level.

(2) Attentional and perceptual intercorrelations

Table 6.2 shows the significant correlations for the attentional and perceptual parameters in the depressed group.

From table 6.2 it can be seen that the anticipatory electrodermal activity to the left (success) light (PREL) correlated with aspiration discrepancy (AD) at the 0.1% level and negative thinking (BSCT) at the 5% level. Age was negatively correlated with this variable. No significant correlation was found for 'anticipatory' electrodermal activity to the right (failure) light (PRER), however the difference between these two measures

Table 6.2 Attentional and perceptual significant correlations

PREL	AD 0.66 ***	BSCT 0.50 *	Age -0.50 *
PRER	None		
PREL-PRER	SEN.diff. 0.64 **	SENR 0.50 *	
DUL	AD 0.62 **		
DUR	None		
DUL-DUR	None		
TL	AD 0.68 ***	BSCT 0.47 *	Age -0.45 *
TR	None		
TL-TR	SEN.diff. 0.56 **	SENR 0.48 *	
SENL	None		
SENR	SEN.diff. 0.54 *	PREL-PRER 0.50 *	TL-TR 0.48 *
SENR-SENL (SEN. diff.)	PREL-PRER 0.64 **	TL-TR 0.56 *	SENR 0.54 *

*** $p < 0.001$ ** $p < 0.01$ * $p < 0.05$ $n = 20$ $df = 19$

(PREL-PRER), indicating differential anticipatory electrodermal activity to success and failure, was found to have a high correlation (at the 1% level) with the difference in perceptual sensitivity scores (SEN diff.). In addition, PREL-PRER correlated with the perceptual sensitivity to the right light (SENR) at the 5% level.

The only measure of electrodermal activity during the actual presentation of the left and right lights in the problem solving task that revealed a significant correlation was the measure of electrodermal activity

during the presentation of the left (success) light (DUL). This correlated with aspiration discrepancy at the 1% level. No significant correlation was found for the measure of electrodermal activity during the presentation of the right light (DUR).

The analysis of the total measured electrodermal activity (the five seconds immediately before and the five seconds during) of the presentation of the left (TL) and right (TR) lights showed that TL correlated with aspiration discrepancy (AD) at the 0.1% level and was positively correlated with negative thinking (BSCT) and negatively correlated with age, both at the 5% level. In addition, the difference between these two measures (TL-TR) correlated with the difference in perceptual sensitivity to the left and right lights (SEN diff.). TL-TR also correlated with the perceptual sensitivity to the right light (SENR).

The perceptual sensitivity measures to the left and right lights revealed that there was no significant correlations for the sensitivity measure to the left light (SENL) but the sensitivity measure to the right light (SENR) correlated with the difference in the perceptual sensitivities (SEN diff.), the difference in the anticipatory electrodermal responding to the left and right light (PREL-PRER) and the difference in the total electrodermal activity to the left and right light (TL-TR). All these correlations were significant at the 5% level. The difference between the perceptual sensitivity scores to the left and right lights (SEN diff.) showed similar correlations; at the 1% level with PREL-PRER, and at the 5% level with TL-TR. In addition, as just mentioned, SENR and SEN diff. were also

correlated at the 5% level.

(3) Attitudinal and cognitive correlations

Table 6.3 shows the significant correlations for the attitudinal and cognitive variables in the depressed group.

Table 6.3 Attitudinal and cognitive significant correlations

I-E	BSCT 0.71 ***			
TAS	GA 0.57 **			
BSCT	I-E 0.71 ***	Age -0.52 *	PREL 0.49 *	TL 0.47 *
JD	None			
ED	None			
AD	TL 0.68 ***	PREL 0.66 ***	SFPS 0.47 *	SFTAS 0.46 *

*** $p < 0.001$ ** $p < 0.01$ * p 0.05 $n = 20$ $df = 19$

Table 6.3 shows that negative thinking (BSCT) and externality (I-E) were highly correlated, at the 0.1% level. Moreover, negative thinking was negatively correlated, at the 5% level of significance, with age and positively correlated with anticipatory electrodermal activity to the left light (PREL) and with total measured electrodermal activity to the left light (TL).

As previously mentioned, following table 6.1, general anxiety (GA) and test anxiety (TAS) scores correlated at the 1% level.

The analysis of the performance estimate scores revealed no significant correlations for either judgement discrepancy (JD) or expectancy discrepancy (ED). However, aspiration discrepancy (AD) was found to

be significantly correlated, at the 0.1% level, with total measured electrodermal activity and anticipatory electrodermal activity to the left light (TL and PREL). Aspiration discrepancy (AD) also correlated with background electrodermal activity during both the problem solving task (SFPS) and during the completion of the test anxiety scale (SFTAS) at the 5% level of significance.

B. Recovered Depressed

(1) General and background intercorrelations

Table 6.4 shows the significant correlations for the general and background variables for the depressed patients seen after recovery.

Table 6.4 General and background significant correlations

Age	PREL 0.58 *	TL-TR 0.55 *	AD 0.53 *
BDI	GA 0.68 **	AD 0.61 **	
GA	BDI 0.68 **	TAS 0.60 *	AD 0.57 *
SFTAS	None		
SFPS	SENL 0.70 **	SENR 0.64 *	JD 0.54 *
SFV	JD 0.7 **		

** $p < 0.01$ * $p < 0.05$ $n = 15$ $d.f. = 14$

From table 6.4 it can be seen that in the patients recovered from depression age was positively correlated with anticipatory responding to the left light (PREL and the difference between the total measured activity to the left light (TL)

and right light (TR) (that is TL-TR). Age also correlated with aspiration discrepancy (AD). All these correlations were at the 5% level.

The level of self rated depression as measured by the Beck Depression Inventory (BDI) had high correlations (1% level) with both general anxiety (GA) and aspiration discrepancy (AD). In addition, general anxiety was correlated with aspiration discrepancy (AD) and test anxiety (TAS) at the 5% level of significance.

The background measure of electrodermal activity during the completion of the test anxiety scale (SFTAS) revealed no significant correlation, whereas the background measure of electrodermal activity during the problem solving task (SFPS) correlated at the 1% level with the perceptual sensitivity score to the left light (SENL) and to the right light (SENR) at the 5% level. SFPS also correlated with judgement discrepancy (JD) at the 5% level. In addition the background measures of electrodermal activity during the light detection (vigilance (SFVI) part of the experiment also correlated with judgement discrepancy at the 1% level.

(2) Attentional and perceptual intercorrelations

Table 6.5 shows the significant correlations for the attentional and perceptual variables in the recovered depressives.

Table 6.5 Attentional and perceptual significant correlations

PREL	Age 0.58 *		SEN.diff. 0.56 *	
PRER	SENR 0.66 *		SENL 0.54 *	
PREL-PRER	SEN diff. -0.87 ***			
DUL	None			
DUR	ED 0.54 *			
DUL-DUR	None			
TL	None			
TR	SENR 0.56 *	SENL 0.56 *	TA -0.52 *	
TL-TR	SEN.diff. -0.60 *			
SENL	SFPS 0.7 **	PRER 0.54 *		
SENR	PRER 0.66 *	SFPS 0.64 *	TR 0.56 *	SEN.diff. 0.53 *
SENL-SENR (SEN.diff.)	PREL-PRER -0.87 ***	TL-TR -0.60 *	SENR 0.53 *	

*** $p < 0.001$ ** $p < 0.01$ * $p < 0.05$ n = 15 d.f. = 14

From table 6.5 it can be seen that anticipatory electrodermal activity to the left light (PREL) correlated with age and with the difference between the perceptual sensitivity scores (SEN.diff.). On the other hand, the anticipatory electrodermal activity to the right light (PRER) correlated with the perceptual sensitivity to the right light (SENR) at the 1% level and the left light (SENL) at the 5% level. The difference between these two measures (PREL-PRER) was very highly negatively correlated at the 0.1% level, with the difference in perceptual sensitivity scores. (SEN diff.)

The electrodermal activity during the presentation of the left light and right light only revealed one significant correlation, at the 5% level, between the electrodermal activity during the presentation of the right light and expectancy discrepancy (ED).

The total measured electrodermal response to the left and right lights showed that there was no significant correlation for the left light (TL), but response to the right light (TR) was significantly correlated with the perceptual sensitivity scores to both the left light (SENL) and the right light (SENR). TR correlated negatively with test anxiety (TAS), all these correlations being at the 5% level of significance. It was also found that the difference between the total measured electrodermal activity to the left light (TL) and the right light (TR), that is TL-TR, correlated at the 5% level of significance with the difference in perceptual sensitivity scores (SENR-SENL), i.e. SEN.diff. This was an inverse correlation.

Perceptual sensitivity to the left light was found to be significantly correlated with background electrodermal activity during the problem solving test (SFPS) at the 1% level, and with the anticipatory electrodermal activity to the right light (PRER) at the 5% level. Perceptual sensitivity to the right light was also found to be correlated with PRER and SFPS and with the total measured electrodermal activity to the right light.

The difference between the perceptual sensitivity scores (SENR-SENL i.e. SEN.diff.) correlated negatively with PREL-PRER at the 0.1% level and negatively with TL-TR at the 5% level, both of which were mentioned earlier. SEN diff. also correlated with SENR at the 5% level.

(3) Attitudinal and cognitive

Table 6.6 shows the correlations for the attitudinal and cognitive variables for the recovered depressed subjects.

Table 6.6 Attitudinal and cognitive significant correlations

I-E	None		
TAS	GA 0.6 *	BSCT 0.56 *	TR -0.52 *
BSCT	TA 0.56 *		
JD	SFVI 0.7 **	SFPS 0.54 *	
ED	DUR 0.54 *		
AD	BDI 0.61 *	GA 0.57 *	Age 0.53 *

** $p < 0.01$ * $p < 0.05$ $n = 15$ d.f. = 14

From table 6.6 it can be seen that externality did not correlate with any other variable while test anxiety (TAS) correlated with general anxiety (GA), negative thinking (BSCT) and was negatively correlated with the total measured electrodermal activity to the right light (TR), all correlations occurring at the 5% level of significance.

The performance estimate scores for these recovered subjects revealed that judgement discrepancy was correlated at the 1% level with the background level of electrodermal activity during the light detection (vigilance) (SFVI) part of the experiment and at the 5% level with the background level of electrodermal activity during the problem solving task (SFPS). Expectancy discrepancy correlated with electrodermal activity during the presentation of the right light (DUR) at the 5% level. Aspiration discrepancy (AD) correlated with the level of self reported depression

(BDI), general anxiety (GA) and age, at the 5% level.

C. Anxious control group

(1) General and background

Table 6.7 shows the general and background correlations in the anxious control group.

Table 6.7 General and background significant correlations

Age	None
BDI	GA 0.67** BSCT 0.58*
GA	BDI 0.67** TA 0.66** BSCT 0.60* DUL -0.52*
SFTAS	None
SFPS	None
SFVI	I-E 0.50*

** $p < 0.01$ * $p < 0.05$ n = 17 df = 16

From table 6.7 it can be seen that age did not correlate significantly with any variable.

The level of self rated depression (BDI) correlated with general anxiety at the 1% level, and negative thinking (BSCT) at the 5% level. In addition, general anxiety was also correlated with test anxiety at the 1% level and with externality (I-E) and electrodermal activity during the presentation of the left light (DUL) at the 5% level. Background electrodermal activity during the light detection task (SFVI) correlated with externality (I-E) at the 5% level.

(2) Attentional and perceptual

Table 6.7 shows the significant correlations for the attentional and perceptual variables in the anxious control group.

Table 6.8 Attentional and perceptual significant correlations

PREL	None		
PRER	None		
PREL-PRER	None		
DUL	GA -0.52 *	SENR -0.52 *	TAS -0.50 *
DUR	TAS -0.57 *		
DUL-DUR	SENR -0.52 *		
TL	None		
TR	None		
TL-TR	SENR -0.58 *		
SENL	SEN diff. -0.51 *		
SENR	TL-TR -0.5 *	DUL -0.52 *	DUL-DUR -0.52 *
SENR-SENL (SEN diff.)	SENL -0.51 *		

* $p < 0.05$ $n = 17$ $df = 16$

From table 6.8 it can be seen that anticipatory electrodermal activity to the left and right lights (PREL and PRER) did not yield any significant correlations. On the other hand electrodermal activity measured during the actual presentation of the left light (DUL, signalling a success) revealed a negative correlation with general anxiety, perceptual

sensitivity to the right light (SENR) and test anxiety (TAS). Electrodermal activity measured during the actual presentation of the right light (DUR, signalling a failure) also correlated negatively with test anxiety (TAS). The difference between these two measures of electrodermal activity (DUL-DUR) correlated negatively with the perceptual sensitivity to the right light (SENR).

With regard to total measured electrodermal activity to signalled success (left light) and failure (right light) events, a negative correlation was found between the difference of total measured electrodermal activity to left and right lights (TL-TR) and the perceptual sensitivity to the right light (SENR). This indicates that the higher the degree of electrodermal activity of the left light compared to the right light, the lower the perceptual sensitivity to the right light (SENR).

Apart from the above mentioned correlations between SENR and other variables, the perceptual sensitivity to the left (success) light (SENL) was found to correlate negatively with the difference between the perceptual sensitivity measures (SENR-SENL , i.e. SEN.diff.), showing that the lower the sensitivity to the left light, the greater the difference between these perceptual measures (i.e. SENR-SENL).

All the correlations in table 6.8 occurred at the 5% level of significance.

(3) Attitudinal and cognitive

Table 6.9 shows the attitudinal and cognitive correlations for the anxious control group.

Table 6.9 Attitudinal and cognitive significant correlations

I-E	BSCT 0.68 **	SFVI 0.50 *	
TAS	GA 0.66 *	DUR -0.57 *	DUL -0.50 *
BSCT	I-E 0.68 **	GA 0.60 *	BDI 0.57 *
JD	None		
ED	None		
AD	None		

** $p < 0.01$ * $p < 0.05$ $n = 17$ $df = 16$

Table 6.9 shows that externality (I-E) was highly correlated with negative thinking (BSCT) at the 1% level of significance. In addition, externality correlated with the background level of electrodermal activity during the light detection (vigilance) part of the experiment.

Test anxiety (TAS) correlated positively with general anxiety (GA) and negatively with electrodermal activity during the presentation of both the left light (DUL) and the right light (DUR), all at the 5% level of significance.

Negative thinking (BSCT) in addition to being correlated with externality, correlated with general anxiety (GA) and level of depression (BDI); all correlations being at the 5% level.

The performance estimate scores revealed no significant correlations in this group.

C. Normal control group

(1) General and background

Table 6.10 shows the significant correlations for the general and

background variables in the normal group.

Table 6.10 General and background significant correlations

Age	I-E -0.46 *			
BDI	SFPS 0.65 **	PRER 0.64 **	TR 0.58 **	SFTAS 0.58 **
				PREL 0.47 *
GA	BSCT 0.45 *			
SFTAS	BDI 0.58 **	TAS 0.47 *		
SFPS	BDI 0.65 **			
SFVI	AD 0.46 *			

** $p < 0.01$ * $p < 0.05$ $n = 20$ $df = 19$

From table 6.10 it can be seen that age was negatively correlated with externality (I-E) at the 5% level.

In the normal group the level of self reported depression (BDI) had a positive correlation on a number of the psychophysiological measures including, at the 1% level of significance, the background electrodermal measures during the completion of the test anxiety scale (SFTAS) and during the problem solving (SFPS). BDI also correlated with anticipatory electrodermal activity to the right light (PRER) and over the total measured epochs of electrodermal activity to the right light (TR). BDI correlated with anticipatory electrodermal activity to the left light (PREL) at the 5% level.

General anxiety correlated with negative thinking and, in addition to the correlations mentioned between BDI and SFTAS and SFPS, SFTAS was negatively correlated at the 5% level with test anxiety (TAS) and

electrodermal activity during the light detection (vigilance) part of the experiment was correlated with aspiration discrepancy.

(2) Attentional and perceptual

Table 6.11 shows the significant correlations for the attentional and perceptual variables in the normal control group.

Table 6.11 Attentional and perceptual significant correlations

PREL	BDI 0.47 *
PRER	BDI 0.64 **
PREL-PRER	None
DUL	None
DUR	None
DUL-DUR	JD -0.5 *
TL	None
TR	None
TL-TR	JD -0.45 *
SENL	SEN diff. -0.47 *
SENR	None
SENR-SENL (SEN diff.)	SENL -0.47 *

** $p < 0.01$ * $p < 0.05$ n = 20 df = 19

From table 6.11 it can be seen that anticipatory electrodermal activity to the left light (PREL) correlated with level of depression (BDI) at the 5%

level and anticipatory electrodermal activity to the right light (PRER) correlated with level of depression (BDI) at the 1% level. It was also found that the two psychophysiological difference measures DUL-DUR (the difference between electrodermal activity to the left and right lights during their presentation) and TL-TR (the difference between the total measured epochs over the left and right lights) correlated with judgement discrepancy (JD).

The only correlation found for the perceptual parameters was a negative correlation between the perceptual sensitivity to the left light with the difference between perceptual sensitivity to the left and right lights (SEN diff.) at the 5% level.

(3) Attitudinal and cognitive intercorrelations

Table 6.12 shows the significant correlations for the attitudinal and cognitive intercorrelations in the normal group.

Table 6.12 Attitudinal and cognitive significant correlations

I-E	age -0.46 *
TAS	SFTAS -0.47 *
BSCT	GA 0.45 *
JD	DUL-DUR -0.5 * TL-TR -0.45 *
ED	None
AD	SFVI 0.46 *

* $p < 0.05$ $n = 20$ $df = 19$

From table 6.12 it can be seen that externality was negatively correlated with age at the 5% level. Test anxiety (TAS) was also negatively correlated with the background electrodermal activity during the completion of the test anxiety scale (SFTAS) and negative thinking (BSCT) was positively correlated with general anxiety (GA) at the 5% level. The performance estimate scores revealed that judgement discrepancy (JD) was negatively correlated with both the difference in electrodermal activity during the presentation of the left and right lights (DUL-DUR) and the difference between the total measured electrodermal activity to the left and right lights (TL-TR) at the 5% level of significance. Expectancy discrepancy (ED) did not correlate with any other variable and aspiration discrepancy (AD) was positively correlated with background electrodermal activity during the light detection (vigilance) part of the experiment.

BRIEF DISCUSSION OF INTRA-GROUP CORRELATIONS

With the large number of correlations found in this study, it is important to point out that at the 5% level of significance, one in twenty of these correlations may reach significance by chance. Moreover, with such a large number of correlations, although all significant correlations have been reported, it is not possible to discuss the implications of all of them in detail. Consequently this short discussion will confine itself to the results which have the most bearing on the theoretical perspective advanced earlier.

One of the most striking observations is the absence of a correlation between the Beck depression inventory and any other variable in the depressed group. Moreover negative thinking (BSCT) and self rated depression were far from being significantly correlated in this group. This finding may be explained in two ways: firstly, as Paykel et al. (1973) and others have pointed out, a depressive illness may distort self rated depression as measured by the BDI. However, if the illness did have a distorting effect on this measure, there is no reason to believe that the other self-report scales are not equally vulnerable. This does raise the serious problem of the value of self-report scales with moderately to severely ill patients in this type of research; secondly, and related to the first explanation, it might be argued that although negative thinking, fear of failure and other cognitive factors may be important aetiologically, as the illness worsens, other factors become important in determining the course of the illness. In terms of the coping model of depression put forward in chapter three, it may be argued that negative thinking, in addition to exaggerating perceived stress, in some individuals can also act as an internal constraint on adaptive coping behaviour which in turn may predispose the individual

to a biological vulnerability. For other individuals coping alternatives are available that limit the mood disturbance by reducing biological vulnerability. The idea that certain 'anti-depressive' behaviours are used by individuals when experiencing low mood has been suggested by other authors (e.g. Rippere, 1976).

It is interesting to note that the level of depression and negative thinking were correlated in the anxious group, where presumably the level of depression was lower. In the recovered depressed group, however, the level of depression was correlated with general anxiety and with aspiration discrepancy, but again not with negative thinking. Interestingly, in the recovered depressed group aspiration discrepancy was also correlated with general anxiety and the recovered depressives were the only subjects who showed these correlations. Thus, as implied by Beck, the perceived discrepancy between estimated actual performance and level of aspiration may be an important problem in the depression-prone individual.

The attentional and perceptual measures used to indicate bias towards positive and negative events produced some intriguing results. However, it is interesting to note that in the depressed group, the measure of bias in anticipatory responding to signalled success and failure events (that is PREL-PRER) had a high correlation with the measure of perceptual sensitivity bias to lights that had previously signalled those events (that is SENR-SENL, SEN diff.). The measure of bias of total electrodermal activity (TL-TR) correlated with the difference between the perceptual sensitivity to the right and left lights (SEN diff.) for this group. This may indicate tha a negative bias found in one modality of responding is not necessarily specific only to that modality. In other words, if depressed patients are indeed more sensitive to negative events compared to positive events, this phenomenon may be measurable

in more than one system. The positive correlation between the difference measure of anticipatory electrodermal activity to the left and right lights (PREL-PRER) and the difference in perceptual sensitivity thresholds (SENR-SENL, i.e. SEN.diff.) in the depressed subjects became a high negative correlation when these subjects were recovered from their depression. This may indicate that on recovery from depression the changes in the different modalities have moved in opposite directions! In addition, it also appears that in very general terms, the psychophysiological correlations found for the depressed group and recovered depressed group occurred mainly in the anticipatory epochs, that is electrodermal activity in the five seconds before a light signalling success or failure was presented (i.e. PREL and PRER). On the other hand, in the anxious control group there was a tendency for the psychophysiological correlations to occur in the five second epochs during the actual presentation of the lights signalling success and failure (i.e. DUL and DUR). It is difficult to interpret this finding except to speculate that, in the depressed group, as far as success and failure events are concerned, the anticipation of these events may be more important than their occurrence, whereas in the anxious group it is the response to the actual stimuli signalling success and failure that is important. It is, of course, a very tenuous suggestion at this stage but may warrant further investigation.

The measures of attitudinal and cognitive factors revealed some interesting relationships. It was found that in the depressed group and the anxious group, externality (I-E) had a high correlation with negative thinking. The tendency for psychiatric patients in general (with the possible exception of drug addicts and alcoholics e.g. Lefcourt, 1976) to have higher externality scores than normal

controls may reflect higher negative thinking. Moreover, it was argued (Weintraub et al., 1974) that negative thinking as measured by the BSCT was primarily centred on depressive thought content. However, in both the anxious group and the normal group negative thinking (BSCT) correlated with general anxiety. Further, in the recovered depressed group negative thinking (BSCT) correlated with test anxiety. In fact, in this group (R.Dep.) this was the only correlation found for negative thinking. If these correlations are found to be consistent using other anxiety scales, then some doubt must be thrown either on the specificity of the BSCT for measuring depressive thought content, or on the specificity of negative thinking as measured by the BSCT to depression. The correlations with BSCT in the recovered depressed group as compared to the anxious control group may, however, indicate that the sources of 'negative thinking' are different in the two groups. In contrast to the recovered depressed group's single correlation of negative thinking with test anxiety, the anxious group's negative thinking (BSCT scores) correlated with general anxiety, externality and level of depression. This may imply that in the depression-prone individual test anxiety, which has been used as a measure of fear of failure, may be a salient factor that might predispose the individual to think negatively. In the anxious group, however, test anxiety did not correlate with negative thinking (BSCT).

The correlations between the various performance estimates revealed that for the anxious group, judgement discrepancy, expectancy discrepancy and aspiration discrepancy did not correlate with any other variable. In the normal group, judgemen discrepancy correlated negatively with two of the electrodermal measures of attentiona bias and aspiration discrepancy correlated with background electrodermal activity

during the light detection (vigilance) part of the experiment (SFVI). In the depressed group, however, aspiration discrepancy was correlated with anticipatory electrodermal activity and total electrodermal activity to the left light. It thus appears that the higher the aspiration discrepancy in depression, the greater the electrodermal activity to success events. This may reflect a mismatch between expected and actual performance reflected in high electrodermal activity to an unexpected positive event. More interesting perhaps is the finding that aspiration discrepancy in the recovered depressives correlated with level of depression, general anxiety and age. Thus, in the depression-prone individual aspiration discrepancy may be an important cognitive factor related to both the level of depression and the level of anxiety. Moreover it was only in this particular group of subjects that these correlations were found.

There is, of course, a limit to the more detailed and theoretical discussion that can be conducted on these correlations until the analysis of variance results have been examined. Nevertheless, four general points seem to have emerged: (1) the level of self-reported depression in depressed patients does not bear out the relationship with other variables that might have been expected from the literature, (2) negative thinking (BSCT), as measured by a scale specifically designed to measure depressive thought content, has been found to correlate with both the level of self-reported depression and anxiety in an anxious group but with neither in a clinically depressed group, and (3) psychophysiological parameters of attention tend to reveal that it is anticipatory responding to positive and negative events that shows the most salient correlations in depressed subjects, but it is psychophysiological responding to the actual signalled success and failure events

that show the most salient correlations in clinically anxious subjects and (4) in depression-prone subjects (R.Dep.) the discrepancy between estimated (actual) performance and the aspirated to level of performance appears significantly related to both the level of self reported depression and anxiety. Moreover, it is only in the depression prone group that this relationship appears to exist.

The correlations between the change scores (Dep. - R.Dep.) of the fifteen patients seen when depressed and after recovery are reported in the next three sub-sections.

1. General and background

Table 6.13 shows the significant correlations for changes in general and background variables.

Table 6.13 General and background significant correlations

BDI	TL-TR -0.55 *	I-E 0.54 *	BSCT 0.52 *
GA	TAS 0.64 **	BSCT 0.54 *	DUL-DUR -0.53 *
SFTAS	None		
SFPS	None		
SFVI	None		

** $p < 0.01$ * $p < 0.05$ n = 15 df = 14

From table 6.13 it can be seen that the changes in self rated depression on recovery (BDI) correlated negatively with the changes in the total electrodermal activity to the left and right lights (TL-TR). In addition, the change in self reported depression (BDI) correlated with the change in externality (I-E) and negative thinking (BSCT), all these correlations occurring at the 5%

level of significance.

The change in general anxiety correlated with the change in test anxiety (TAS) at the 1% level, general anxiety also correlated with the change in negative thinking and with the change in electrodermal activity to the left and right lights during the presentation (DUL-DUR) at the 5% level.

The changes in background measures of electrodermal activity during the three phases of the experiment showed no significant correlations.

(2) Attentional and perceptual

Table 6.14 shows the significant correlations with changes in the attentional and perceptual variables.

Table 6.14 Attentional and perceptual significant correlations

PREL	SENL 0.57 *			
PRER	SENL 0.54 *			
PREL-PRER	None			
DUL	TAS -0.54 *			
DUR	BSCT 0.53 *			
DUL-DUR	SEN diff. 0.67 **			
TL	TAS-0.55 *			
TR	AD 0.54 *			
TL-TR	TAS-0.70 **	BDI -0.55 *		
SENL	PREL 0.57 *	PRER 0.54 *		
SENR	DUL-DUR 0.54 *			
SENR-SENL (SEN.diff.)	DUL-DUR 0.67 **	AD -0.59 *	I-E 0.52 *	DUR -0.52 *
	SENR 0.50 *			

** $p < 0.01$ * $p < 0.05$ n = 15 d.f. = 14

Table 6.14 shows that the change in anticipatory electrodermal activity to the left and right light (PREL and PRER) correlated at the 5% level with the change in the perceptual sensitivity measure to the left light (SENL). On the other hand the change in electrodermal activity during the presentation of the left light correlated negatively with the change in test anxiety (TAS), while the change in electrodermal activity during the presentation of the right light (TL) correlated positively with negative thinking (BSCT). Also at the 5% level the change in the difference or bias of these measures (i.e. DUL-DUR) correlated with the change in the difference or bias in the perceptual sensitivity measures to the left and right lights (i.e. SEN.diff.).

The change in total measured electrodermal activity to the left light (TL) correlated negatively with the change in test anxiety (TAS), while the change in total measured electrodermal activity to the right light (TR) correlated with the change in aspiration discrepancy, both correlations occurring at the 5% level. The change in TL-TR correlated negatively, at the 1% level of significance, with the change in test anxiety and at the 5% level and with the change in self-reported depression (BDI).

The perceptual sensitivity measures revealed that, as mentioned, the change in SENL correlated with the change in PREL and PRER and also, as mentioned, the change in SENR correlated with the change in DUL-DUR. The change in the difference (or bias) in perceptual sensitivity to the left light and right light (SEN.diff.) correlated with the change in the difference in electrodermal responding during the presentation of the left and right lights (DUL-DUR) at the 1% level and correlated negatively at the 5% level with the change in aspiration

discrepancy and positively with change in electrodermal activity during the presentation of the right light (SENR) and with the change in externality (I-E), all at the 5% level of significance.

3. Attitudinal and cognitive

Table 6.15 shows the significant correlations for the changes in the attitudinal and cognitive variables.

Table 6.15 Attitudinal and cognitive significant correlations

I-E	BDI 0.54 *	SEN. diff. 0.52 *		
TAS	TL-TR -0.70 **	GA 0.64 **	DUL-DUR -0.62 *	TL -0.55 *
	DUL -0.54 *	BSCT 0.52 *		
BSCT	GA 0.54 *	DUR 0.53 *	TAS 0.52 *	BDI 0.52 *
JD	ED -0.52 *			
ED	JD -0.52 *			
AD	SEN diff. -0.6 *	TR 0.54 *		

** $p < 0.01$ * $p < 0.05$ $n = 15$ $df = 14$

Table 6.15 shows that the change in externality (I-E) correlated with the change in self reported depression (BDI) and the change in the difference in the perceptual sensitivity measure to the left and right light (SEN diff.), both at the 5% level.

As mentioned following table 6.14, the change in test anxiety (TAS) correlated with a number of the psychophysiological variables including TL-TR at the 1% level and DUL-DUR, TL and DUL at the 5% level. In addition, the change in

test anxiety (TAS) correlated at the 1% level with the change in general anxiety (GA) and with the change in negative thinking (BSCT) at the 5% level. The change in negative thinking (BSCT), in addition to correlating with the change in test anxiety, correlated with the change in general anxiety (GA), the change in the level of self-reported depression (BDI) and the change in the electrodermal activity during the presentation of the right light (DUR).

The change in judgement discrepancy (JD) and expectancy discrepancy (ED) was found to be inversely correlated at the 5% level and the change in aspiration discrepancy was found to be negatively correlated at the 5% level with the change in differential sensitivity (SEN diff.) and the change in total measured activity to the right light (TR).

Bearing in mind the qualifications about multiple correlations as discussed on p. 199 some of the correlations listed above are worth discussing.

The change in self reported depression (BDI) was found to correlate with the change in negative thinking (BSCT). This is an interesting finding since it will be recalled that BDI and BSCT did not correlate in either the depressed or recovered depressed group. In addition, the change in externality (I-E) was found to correlate with the change on the BDI. In view of the high externality (I-E), negative thinking (BSCT) correlation found for the depressed group, it might be the case that this correlation between change in I-E and change in BDI reflects the possibility that I-E may to some extent measure negative thinking. If such is the case, then the fact that the change in both externality (I-E) and negative thinking (BSCT)

correlated with the change in BDI may indicate that negative thinking is causally related to depression, although it does not determine the absolute level. Again this provides some support for the multi-component approach to depression.

Interestingly the change in TL-TR, a measure of negative (psychophysiological) bias, correlated with the change in self-rated depression (BDI). However, like I-E and BSCT, TL-TR did not correlate with depth of depression in the depressed and recovered depressed. It would seem that like I-E and BSCT, some negative bias may be causally related to depression but not in absolute terms.

As in the previous correlations, the attentional and perceptual parameters produced results which are significant but which are difficult to interpret. The change in psychophysiological bias scores DUL-DUR was found to have a high correlation with the change in perceptual sensitivity bias (SEN diff.). This again may indicate that biases and changes in one system occur in other systems, as mentioned in the previous brief discussion. The change in the perceptual bias score, SEN diff. also correlated with the change in externality (I-E) and aspiration discrepancy (AD). This is an interesting finding since it will be recalled that in the depressed group aspiration discrepancy correlated with self-rated depression and anxiety. It may be that changes in aspiration discrepancy are reflected in changes in the allocation of attention, which in turn affects the change in negative thinking and through this the change in depression. These links are admittedly tenuous, but the correlations both here and as discussed earlier, do suggest this possibility.

The change in test anxiety did not correlate with the change in BDI but did

correlate with the change in negative thinking (BSCT). In addition, there were very high correlations between the changes in test anxiety and the change in the psychophysiological bias scores DUL-DUR and TL-TR. This may indicate that like aspiration discrepancy, test anxiety exerts an indirect effect on depression by influencing the orientation of the individual's response pattern. In other words, it might be hypothesised that test anxiety tends to make the individual more attentive to negative events and cues which in turn triggers negative thinking, which in turn influences mood.

These are the most salient findings that require discussion at this stage. A further more integrated discussion will re-examine some of these issues in the light of subsequent results.

II. BETWEEN GROUPS ANALYSIS

The differences between the groups were investigated using one way analysis of variance and student's t tests. The results of these statistical operations are presented in three sections:

A. Comparison of Depressed group (Dep.) v Anxious group v Normal group

B. Comparison of Recovered Depressed group (R. Dep.) v Anxious group v Normal group

C. Comparison of Depressed group (Dep.) v Recovered Depressed group (R. Dep.)

A. DEPRESSED v ANXIOUS v NORMAL CONTROLS

(1) General and background

(i) Beck Depression Inventory (B.D.I.)

The mean scores of the three groups on the B.D.I. are shown in table 6.16.

Table 6.16 B.D.I. scores

Group	Range	Mean	S.D.	
Dep.	10-48	24.75	9.91	$F = 4.52$
Anxious	0-32	17.06	9.58	$p < 0.001$
Normal	0-6	1.3	1.87	(d.f. = 54)

Table 6.17 shows the results of the t tests applied to the individual group comparisons.

Table 6.17 Differences between mean B.D.I. scores

Comparisons	diff. between Means	t	p	(df)
Dep. v Normal	23.45	9.34	0.001	38
Dep. v Anxious	7.7	3.09	0.005	35
Normal v Anxious	15.75	6.33	0.001	35

As expected (hypothesis 1.i), the depressed group had the significantly highest level of self reported depression. Moreover, the mean (24.75) provides some evidence that as a group these patients were moderately to severely depressed. The anxious group also showed a moderate level of depression, though significantly less so than the depressed group.

(ii) General Anxiety Scale (G.A.S.)

The mean scores on the G.A.S. are shown in table 6.18.

Table 6.18 G.A.S. scores

Groups	Range	Mean	S.D.	
Dep.	4-15	11.0	3.32	F = 55.23
Anxious	8-17	12.35	2.83	p< 0.001
Normal	0-8	3.50	2.16	(d.f. 54)

Table 6.19 shows the results of the t tests applied to the individual group comparisons.

Table 6.19 Differences between mean G.A.S. scores

Comparisons	diff. between means	t	p	df
Dep. v Normal	7.5	8.43	0.001	38
Dep. v Anxious	1.35	1.45	NS	35
Normal v Anxious	8.85	9.52	0.001	35

The results using the General Anxiety Scale show that both anxious and depressed groups differed significantly from normals but did not differ between themselves, thus disconfirming hypothesis 1.ii. This may be attributable to the scale used, or it may reflect the fact that depressives also have high anxiety. This has been found many times and some (e.g. Wolpe, 1971) have suggested treating depression by treating the anxiety component.

Tables 6.20 - 6.25 deal with the mean number of spontaneous fluctuations per minute in each of the three separate stages of the experiment: (1) during T.A.S. completion, (2) during the problem solving task, (3) during the signal (light) detection task.

(iii) Mean number of spontaneous fluctuations per minute during T.A.S. completion (SFTAS)

The mean scores for this psychophysiological variable are shown in table 6.20.

Table 6.20 SFTAS scores

Groups	Range	Mean n	S.D.	
Dep.	0.5-5.2	2.8	1.3	F = 10.85
Anxious	1.7-12.2	6.4	3.1	p< 0.001
Normal	0.6-9.5	4.9	2.5	df = 52

Table 6.21 shows the results of the t tests applied to the individual group comparisons.

Table 6.21 Differences between SFTAS scores

Comparison	diff. between means	t	p	df
Dep. v Normal	2.13	2.80	0.01	38
Dep. v Anxious	3.64	4.61	0.001	35
Normal v Anxious	1.51	1.91	NS	35

(iv) Mean number of spontaneous fluctuations per minute during problem solving task (SFPS)

The mean scores on this psychophysiological variable are shown in table 6.22.

Table 6.22 SFPS scores

Groups	Range	Mean	S.D.	
Dep.	0.1-5.3	2.8	1.5	F = 5.34
Anxious	1.1-11.42	5.0	2.5	p < 0.01
Normals	1.2-9.6	4.8	2.6	(df = 54)

Table 6.23 shows the results of the t tests applied to the individual group comparisons.

Table 6.23 Difference between SFPS scores

Comparisons	diff. between means	t	p	df
Dep. v Normal	1.94	2.73	0.01	38
Dep. v Anxious	2.15	2.9	0.01	35
Normal v Anxious	0.21	0.28	NS	35

(v) Mean number of spontaneous fluctuations per minute during the signal detection (vigilance) task (SFVi)

The mean scores for this psychophysiological variable are shown in table 6.24.

Table 6.24 SFVi scores

Groups	Range	Mean	SD	
Dep.	0.1-5.3	1.05	0.42	F = 7.21
Anxious	1.1-11.42	3.25	2.73	$p < 0.005$
Normal	1.2-9.6	2.0	1.30	(df = 54)

Table 6.25 shows the results of the t tests applied to the individual group comparisons.

Table 6.25 Difference between SFVi scores

Comparisons	diff. between means	t	p	df
Dep. v Normal	0.95	1.69	NS	38
Dep. v Anxious	2.20	3.8	0.001	35
Normal v Anxious	1.26	2.17	0.05	35

The data show that during the completion of the T.A.S. the depressed group had the least amount of automic activity as measured by the mean number of electrodermal spontaneous fluctuations per minute. They were significantly lower than both the normal and anxious groups. Interestingly the normal group and anxious group just failed to be significantly different from each other, although the anxious group had the highest level of spontaneous fluctuations.

During the problem solving session the depressed group again showed th lowest amount of electordermal activity, compared to the normal and the anxious groups. During this task the normal and anxious groups were not significantly different.

During the vigilance task the mean spontaneous fluctuations of the normal group dropped to the level of the depressed group and both these groups were significantly lower than the anxious control group.

(2) Attention and perceptual

(i) Psychophysiology

The means and standard deviations over the nine measured epochs analysed during the problem solving task are shown in table 6.26. The t statistic and level of significance are given below where relevant.

The nine elements of psychophysiological data used as a measure of attention to positive and negative events show that the depressed group were significantly different in their responding to the left light signalling success. This difference appeared to the number of 'anticipatory'

Table 6.26 Psychophysiological attentional parameters

Variables		SECTION I			SECTION 2			SECTION 3		
		PREL	PRER	PREL-PRER	DUL	DUR	DUL-DUR	TL	TR	TL-TR
Groups										
Dep.	Mean	5.15	4.35	0.80	2.70	4.1	-1.4	7.8	8.5	-0.70
	SD	(2.8)	(2.85)	(2.21)	(2.49)	(3.19)	(2.62)	(4.95)	(5.28)	(4.01)
Anxious	Mean	7.41	6.76	0.65	4.94	5.24	-0.29	12.35	12.00	0.35
	SD	(3.45)	(3.35)	(3.44)	(2.66)	(2.39)	(2.42)	(4.77)	(5.06)	(4.46)
Normal	Mean	7.65	6.05	1.6	4.20	4.35	-0.15	12.05	10.4	1.65
	SD	(3.56)	(3.36)	(3.08)	(3.19)	(2.37)	(2.96)	(5.43)	(4.39)	(4.09)
COMPARISONS										
Dep. v Normal	t	2.42						2.65		
	p	(0.05)	NS	NS	NS	NS	NS	(0.05)	NS	NS
Dep. v Anxious	t	2.08			2.42			2.72		
	p	(0.05)	NS	NS	(0.05)	NS	NS	(0.01)	NS	NS
Normal v Anxious	t									
	p	NS	NS	NS	NS	NS	NS	NS	NS	NS

responses (PREL) and the total number of responses (TL) over the measured epochs. These differences may be explained in terms of the significantly lower background level of spontaneous electrodermal activity during the problem solving task. However, if this is the case, then it might also be expected that such a difference would also be evident to the right light activity, but this was not found. This tends to indicate th that some negative bias (either lower responding to the left (success) or higher responding to the right (failure) did occur. Moreover, inspection of the mean bias scores DUL-DUR and TL-TR shows that the depressed group had the highest negative bias although this was not significant. Unfortunately then, these data make it difficult to confirm or reject hypothesis 2.i, although the results tend towards accepting it. Nevertheless, this finding does seem worthy of further investigation, perhaps using more sophisticated data analysis.

Tables 6.27 - 6.31 are concerned with the perceptual sensitivity thresholds to the left and right lights, derived from the light detection (vigilance) part of the experiment. These results will be discussed together following tables 6.27 - 6.31.

(ii) Perceptual sensitivity to the left light (positive) (SENL)

The mean scores are shown in table 6.27.

Table 6.27 SENL scores

Groups	Range	Mean	SD	
Dep.	18.33-25.25	20.64	1.1	$F = 4.54$
Anxious	16.7-24.1	19.85	1.2	$p < 0.05$
Normal	17.05-20.65	19.13	0.9	(df = 54)

Table 6.28 shows the results of the t tests applied to the individual group comparisons.

Table 6.28 Differences in sensitivity threshold to the left light

Comparison	diff. between means	t	p	df
Dep. v Normal	1.51	3.02	0.005	38
Dep. v Anxious	0.79	1.52	NS	35
Normal v Anxious	0.72	1.38	NS	35

(vii) Perceptual sensitivity for the right light (negative) (SENR)

The mean scores for SENR are shown in table 6.29.

Table 6.29 SENR scores

Groups	Range	Mean	SD	
Dep.	17.1-22.75	20.62	1.09	F = 1.01
Anxious	16.9-23.8	20.11	1.76	NS
Normal	17.5-22.25	19.91	1.92	

(viii) Difference between the left and right perceptual sensitivity thresholds

This is calculated by subtracting the mean sensitivity threshold to the left light from the mean sensitivity threshold to the right light for each individual. Thus a positive difference reflects a lower threshold to the left compared to right light detections, i.e. a greater sensitivity (lower threshold for detection) to the left light previously paired with success.

Table 6.30 shows the mean scores for SEN diff.

Table 6.30 SEN diff. scores

Groups	Range	Mean	SD	
Dep.	(-1.4) - (+1.4)	-0.03	0.67	
Anxious	(-0.4) - (+1.15)	0.15	0.45	$F = 8.440$ $p < 0.001$
Normal	(-0.65) - (+2.25)	0.78	0.76	(df = 54)

Table 6.31 shows the results of the t tests applied to the individual group comparisons.

Table 6.31 Difference between sensitivity thresholds (SEN diff.)

Comparisons	diff. between means	t	p
Dep. v Normal	0.8	3.8	0.001
Dep. v Anxious	0.18	0.8	NS
Normal v Anxious	0.63	3.0	0.005

The tables 6.27 - 6.31 show that as in the case of the psychophysiological parameters, it is only the sensitivity to the left light that shows any significant difference between the groups.

Tables 6.30 and 6.31 show the difference and bias in each group's left/right sensitivity thresholds. These tables show that in both the anxious and the normal group there was a small positive bias. That is the left light (previously paired with success) had a lower detection threshold than the right light (previously paired with failure) for these two groups. This bias was small, (0.15 of a volt for the anxious group an 0.78 of a volt for the normal group). In the depressed group, on the other hand, this bias was in the opposite direction, though again small

(0.03 of a volt). Nevertheless this comparison of the difference in sensitivity bias was found to be significant between the depressed group and the normal group.

It would appear from this data that the depressed subjects tended to have either a raised detection threshold to the left (previously paired with success) light, or a lowered detection threshold to the right (previously paired with failure) light compared to the normal subjects. However, since the depressed subjects had the highest threshold to both the left and the right light, the exact nature of the bias is difficult to determine. Nevertheless some negative bias does seem to be operative in the depressed group as compared to the normal control group. The anxious group also had a tendency to be 'less positive' than the normals, although this did not reach significance.

The finding that the normal group had a lower threshold for the detection of the left light is in line with other work on positive biases in cognitive processing (e.g. Lloyd and Lishman, 1975). Moreover this finding of a positive bias for the normal subjects helps to explain some of the negative correlations found with SEN diff. and other variables It now appears that high TA and BSCT for example, may exert an influence by reducing the degree of positive bias on certain psychological processes. This point will be taken up later. For the moment, though, it can be seen that these data offer some confirmatory evidence for hypothesis 2, i. Although the extent to which negative (or perhaps more accurately a less positive bias) is specific to depression may be

questionable, in view of the relationship of the anxious group's SEN diff. scores to the depressed and normal group's SEN diff. scores.

(3) Attitudinal and cognitive

(i) Internal External Scale

The mean scores on the I-E scale are shown in table 6.32.

Table 6.32 I-E scores

Group	Range	Mean	SD	
Dep.	7-23	12.45	4.25	F = 7.17
Anxious	8-19	12.41	2.9	p< 0.005
Normal	1-16	8.6	3.56	(df = 54)

Table 6.33 shows the results of the t tests applied to the individual group comparisons.

Table 6.33 Differences between mean I-E scores

Comparisons	diff. between Means	t	p	df
Dep. v Normal	3.85	3.35	0.005	38
Dep. v Anxious	0.04	0.03	NS	35
Normal v Anxious	3.81	3.12	0.005	35

As predicted (hypothesis 3.i) the anxious group and the depressed group both had significantly higher external scores than the normal group However, there was no significant difference between the anxious and the depressed group. This finding does cast some doubt on Seligman's

(1975) view of the relationship between anxiety and depression with regard to the perception of controllability, though, as mentioned earlier, the I-E scale does confound the stable/unstable and internal/external dimensions.

(ii) Test Anxiety Scale (TAS)

The mean scores on the TAS are shown in table 6.34.

Table 6.34 TAS scores

Groups	Range	Mean	S.D.	
Dep.	9-36	25.6	8.23	$F = 14.4$
Anxious	10-35	25.71	8.18	$p < 0.001$
Normal	6-28	14.4	6.18	($df = 54$)

Table 6.35 shows the results of the t tests applied to the individual group comparisons.

Table 6.35 Differences between mean TAS scores

Comparisons	diff. between means	t	p	df
Dep. v Normal	11.2	4.67	0.001	38
Dep. v Anxious	0.11	0.04	NS	35
Normal v Anxious	11.3	4.52	0.001	35

As predicted (hypothesis 3.ii), the results show that both the anxious and the depressed group had significantly higher TAS scores than the normal group but did not differ between themselves. This is an interesting finding since if TAS is some measure of fear of failure, then

this construct is useful, although its specificity to depression is questionable. Moreover, if the fear of failure construct can be related to certain rules, i.e. "In order to be happy, I have to be successful in whatever I undertake", then again the specificity of these rules to depression may be questionable. However, such a view depends on a number of tentative links between the TAS and the 'rules' as outlined by Beck. Nevertheless, it would seem a profitable area for future research.

(iii) Beck Story Completion Test (BSCT)

The mean scores of the three groups on negative thinking (BSCT) are shown in table 6.36.

Table 6.36 BSCT scores

Groups	Range	Mean	SD	
Dep.	27-79	50.15	15.24	$F = 10.72$
Anxious	29-92	56.35	18.72	$p < 0.001$
Normal	11-52	35.05	8.6	(df = 54)

Table 6.37 shows the results of the t test applied to the individual group comparisons.

Table 6.37 Differences between mean BSCT scores

Comparisons	diff. between mean	t	p	df
Dep. v Normal	15.05	3.7	0.001	38
Dep. v Anxious	6.2	1.29	NS	35
Normal v Anxious	21.30	4.45	0.001	35

Both the anxiety and depressed groups were found to have significantly higher negative thinking scores than the normal group. What was unexpected was that the anxious group had a slightly higher mean and range on this scale than the depressed group. Moreover it will be recalled that it was only in the anxious group that BDI and BSCT correlated.

This finding does not support the prediction (hypothesis 3.iii) that the depressed group would have the highest level of negative thinking. Since this scale was purposely designed for depression, it may indicate that negative thinking as measured by the BSCT is not specific to depression in any narrow sense.

Tables 6.38 - 6.41 deal with the discrepancy scores derived from the subjects' estimation of their performance, their ability to improve their score and their aspiration on the problem solving task. The results will be given together and briefly discussed together.

(iv) Judgement discrepancy (JD)

The mean judgement discrepancy scores are shown in table 6.38.

Table 6.38 JD scores

Groups	Range	Mean	SD	
Dep.	(-5) - (+4)	-0.95	2.35	F = 0.197
Anxious	(-5) - (+4)	-0.64	2.52	NS
Normal	(-5) - (+5)	-0.5	2.06	(df = 54)

(v) Expectancy discrepancy (E.D.)

The mean judgement discrepancy scores and group statistics are shown in table 6.39.

Table 6.39 E.D. scores

Group	Range	Mean	SD	
Dep.	(0) - (+4)	0.55	1.1	F = 1.12
Anxious	(0) - (+2)	0.18	0.53	NS
Normal	(0) - (+3)	0.60	0.99	(df = 54)

(vi) Aspiration discrepancy (A.D.)

The mean scores and statistics on aspiration discrepancy are shown in table 6.40.

Table 6.40 A.D. scores

Groups	Range	Mean	SD	
Dep.	(0) - (+12)	5.45	2.99	F = 3.98
Anxious	(0) - (+13)	6.47	3.99	$p < 0.05$
Normal	(0) - (+10)	3.45	3.05	(df = 54)

Table 6.41 shows the results of the t tests applied to the individual group comparisons.

Table 6.41 Difference between mean aspiration discrepancy scores

Comparisons	diff. between means	t	p	df
Dep. v Normal	2.0	1.89	NS	38
Dep. v Anxious	1.02	0.93	NS	35
Normal v Anxious	3.02	2.75	0.01	35

These results reveal some surprises. First, although the depressed subjects did tend to undervalue their performance (judgement discrepancy) compared to normals and anxious subjects, this tendency did not reach significance. Second, on the expectancy discrepancy score, the depressed were more like the normals than the anxious group were like the normals. Since this parameter is a measure of the subject's perceived ability to improve his score, it was expected that the depressed subjects would show the lowest perceived ability to improve (hypothesis 3.iv). The non significance of these results is unexpected in view of the literature discussed earlier.

The aspiration level also revealed that it was the anxious group that significantly differed from the normal group and not the depressed group, although the depressed subjects did not significantly differ from the anxious, and the difference between the normals and depressed only just failed to reach significance at the 0.05 level. Thus, in general, hypothesis 3.iv was not confirmed although the results were in the expected direction.

B. RECOVERED DEPRESSED v ANXIOUS v NORMAL CONTROLS

(1) General and background

(i) Beck Depression Inventory (BDI)

The mean scores for the BDI are shown in table 6.42.

Table 6.42 BDI scores

Groups	Range	Mean	SD	
R.Dep.	0-35	11.53	11.88	F = 16.583
Anxious	0-33	17.06	9.58	$p < 0.001$
Normal	0-6	1.3	1.89	(df = 49)

Table 6.43 shows the results of the t test applied to the individual group comparisons.

Table 6.43 Differences between mean BDI scores

Comparisons	diff. between means	t	p	df
R.Dep. v Normal	10.23	3.65	0.001	33
R.Dep. v Anxious	5.53	1.84	NS	30
Anxious v Normal	15.75	5.43	0.001	35

The high depressed score of the recovered depressed with one subject scoring over thirty is problematic. However, subjects could not be excluded from this group because of a high Beck score since it had been a senior clinical staff member's diagnosis that had constituted the selection criteria. When a high Beck score was obtained (five subjects scored over fifteen), the experimenter notified the clinical

member of staff concerned. In all cases the clinician was sure that the patient had recovered from the depressive illness and could be seen as a recovered depressed subject. As mentioned earlier, three of the patients seen as depressed did not satisfy the clinical staff's criteria for recovery and therefore could not be seen again.

(ii) General Anxiety (GAS)

The mean scores on the GAS are shown in table 6.44.

Table 6.44 GAS scores

Groups	Range	Mean	SD	
R.Dep.	1-13	7.00	4.12	$F = 38.98$
Anxious	8-17	12.35	2.83	$p < 0.001$
Normal	0-8	3.50	2.16	(df = 49)

Table 6.45 shows the results of the t tests applied to the individual group comparisons.

Table 6.45 Differences between mean GAS scores

Comparisons	diff. between means	t	p	df
R.Dep. v Normal	3.5	3.37	0.01	33
R.Dep. v Anxious	5.35	5.0	0.001	30
Anxious v Normal	8.86	8.8	0.001	35

The analysis of general anxiety scores shows that the recovered depressed group was significantly more anxious than the normal group but significantly less anxious than the anxious group. Thus both anxiety

and depression were reduced on recovery from a depressive illness, though both were higher than for the normal control group.

Tables 6.46 - 6.49 are concerned with the mean number of spontaneous fluctuations per minute, in each of the three separate stages of the experiment (1) during TAS completion, (2) during the problem solving task and (3) during the signal (light) detection.

(iii) Mean number of spontaneous fluctuations per minute during TAS completion (SFTAS)

The mean score for this psychophysiological variable are given in table 6.46.

Table 6.46 SFTAS scores

Groups	Range	Mean	SD	
R.Dep.	0.1-9.0	4.95	2.5	F = 1.54
Anxious	1.7-12.2	6.4	3.1	NS
Normal	0.6-9.5	4.9	2.5	

(iv) Mean number of spontaneous fluctuations per minute during problem solving task (SFPS)

The mean score for the psychophysiological variable are given in table 6.47.

Table 6.47 SFPS scores

Groups	Range	Mean	SD	
R.Dep.	0.4-7.7	3.63	2.12	F = 1.41
Anxious	1.1-11.42	5.0	2.5	NS
Normal	1.2-9.6	4.8	2.6	

(v) Mean number of spontaneous fluctuations during signal detection (vigilance) task (SFVI)

The mean scores for this psychophysiological variable are shown in table 6.48.

Table 6.48 SFVI scores

Groups	Range	Mean	SD	
R.Dep.	0.4-7.7	1.33	1.56	F = 4.095
Anxious	1.1-11.41	3.25	2.73	$p < 0.05$
Normal	1.2-9.6	1.99	1.30	(df = 49)

Table 6.49 shows the results of t tests applied to the individual group comparisons.

Table 6.49 Differences between SFVI scores

Comparisons	diff. between means	t	p	df
R.Dep. v Normal	0.67	1.01	NS	33
R.Dep. v Anxious	1.92	2.82	0.01	30
Normal v Anxious	1.26	1.97	NS	35

Tables 6.46 - 6.49 show that during the completion of the TAS and the problem solving task there was no significant difference between the compared groups in electrodermal spontaneous activity. The significantly lower level of electrodermal activity of the depressed group compared with the anxious and normal group has disappeared on recovery from depression. During the (light detection) vigilance task, the recovered depressed group had a lower level of electrodermal activity than the anxious group.

(2) Attentional and perceptual

(i) Psychophysiology

The means and standard deviations over the nine electrodermal epochs analysed during the problem solving task are shown in table 6.50. However, no significant difference between any of these variables were found following the analysis of variance.

There was no significant difference found on any of the nine elements of psychophysiological data. The significantly lower number of spontaneous fluctuations found for the depressed group to the left light (PREL and TL) compared to the two control groups discussed earlier, has disappeared on the depressed group's recovery. Thus the prediction of hypothesis 2.ib was not confirmed. Moreover, the difference between the anxious and depressed group on the measured DUL had also disappeared on recovery.

Table 6.50 Psychophysiological attentional parameters

Variables		SECTION 1			SECTION 2			SECTION 3		
		PREL	PRER	PREL-PRER	DUL	DUR	DUL-DUR	TL	TR	TL-TR
Groups										
R. Dep.	Mean	5.67	4.93	0.73	3.07	4.47	-1.4	8.80	9.47	-0.67
	SD	(2.85)	(2.52)	(3.26)	(2.31)	(2.45)	(2.56)	(4.44)	(3.93)	(4.03)
Anxious	Mean	7.41	6.76	0.65	4.94	5.24	-0.29	12.35	12.00	0.35
	SD	(3.45)	(3.5)	(3.44)	(2.66)	(2.39)	(2.42)	(4.77)	(5.06)	(4.46)
Normal	Mean	7.65	6.05	1.60	4.20	4.35	-0.15	12.05	10.40	1.65
	SD	(3.56)	(3.36)	(3.08)	(3.19)	(2.37)	(2.96)	(5.43)	(4.39)	(4.09)

(ii) Perceptual sensitivity threshold to the left (positive) light (SENL)

The mean scores for SENL are shown in table 6.51.

Table 6.51 SENL scores

Groups	Range	Mean	SD	
R.Dep.	17.3-23.45	20.00	1.84	F = 1.492
Anxious	16.7-24.1	19.85	1.83	NS
Normal	17.05-20.65	19.13	1.21	

(iii) Perceptual sensitivity threshold to the right (negative) light (SENR)

The mean scores for SENR are shown in table 6.52.

Table 6.52 SENR scores

Groups	Range	Mean	SD	
R.Dep.	17.3-25.0	20.31	2.15	F = 0.244
Anxious	16.8-23.8	20.11	1.76	NS
Normal	17.5-22.25	19.91	1.08	

(iv) Difference between the left and right sensitivity thresholds (SEN diff.)

This is the mean sensitivity score to the right light minus the mean sensitivity score to the left light for each individual. Thus a positive difference reflects a mean lower threshold to the left compared to a right light detection, i.e. a greater sensitivity (lower threshold for detection) for the left light previously paired with success.

Table 6.53 shows the mean (mean difference) scores for SEN diff.

Table 6.53 SEN diff. scores

Groups	Range	Mean	SD	
R.Dep.	(-1.4)-(+1.4)	0.28	0.84	F = 4.22
Anxious	(-0.4)-(+1.15)	0.15	0.45	$p < 0.05$
Normal	(-0.65)-(+2.25)	0.78	0.76	(df = 49)

Table 6.54 shows the results of the t test applied to the individual group comparisons.

Table 6.54 Difference between sensitivity thresholds

Comparisons	diff. between means	t	p	df
R.Dep. v Normal	0.5	2.17	0.05	33
R.Dep. v Anxious	0.14	0.56	NS	30
Normal v Anxious	0.63	2.74	0.01	35

Table 6.51 shows that in contrast to the significantly higher threshold to the left light found between the normal and depressed group, the recovered depressed group does not significantly differ on this parameter from the normal group. This finding of a change on the parameter examining psychological response to a stimulus which has been paired with success, i.e. a positive event, is consistent with the psychophysiological findings. In both cases significant differences found to the left light, between depressed and normal subjects, has disappeared on the depressives recovery.

As when depressed, table 6.52 shows that on the perceptual sensitivity to the right light there is no significant difference between the recovered

depressed group and the two control groups.

Tables 6.53 and 6.54 show that although to the left and right lights there may not be a significant threshold difference, the bias in perceptual sensitivity (that is SEN diff.) does differentiate between the normal control group and the recovered depressed subjects. Thus, although both groups show a small bias towards a lower detection of the left (positive) light, this bias is significantly greater in the normal group than in the recovered depressed group. It would seem that this provides some confirmation for hypothesis 2.ib.

(3) Attitudinal and cognitive

(i) Internal external scale

The mean scores on the I-E scale are shown in table 6.55.

Table 6.55 I-E scores

Groups	Range	Mean	SD	
R.Dep.	4-17	10.46	3.7	F = 5.754
Anxious	8-19	12.41	2.9	$p < 0.01$
Normal	1-16	8.6	3.56	(df = 49)

Table 6.56 shows the results of the t tests applied to the individual group comparisons.

Table 6.56 Differences between mean I-E scores

Comparisons	diff. between means	t	p	df
R.Dep. v Normal	1.87	1.62	NS	33
R.Dep. v Anxious	1.95	1.63	NS	30
Normal v Anxious	3.81	3.53	0.01	35

Hypothesis 3.ibwas not confirmed by this result. Analysis of externality scores revealed that the recovered depressed scores lay mid-way between the normal and the anxious groups' scores. There was no significant difference between the recovered depressed and the normal group, or the recovered depressed and the anxious group. The possibility that the recovered depressed externality score may change to a high internal score (reflecting a belief in self control and responsibility) was not supported in this study.

(ii) Test Anxiety Scale (TAS)

The mean scores on the TAS are shown in table 6.57.

Table 6.57 TAS scores

Groups	Range	Mean	SD	
R.Dep.	0-34	19.53	6.18	F = 7.57
Anxious	10-35	25.71	8.18	p 0.005
Normal	6-28	14.4	6.18	(df = 49)

Table 6.58 shows the results of the t tests applied to the individual group comparisons.

Table 6.58 Differences between TAS scores

Comparisons	diff. between means	t	p	df
R.Dep. v Normal	5.13	1.70	NS	33
R.Dep. v Anxious	6.17	1.98	NS	30
Normal v Anxious	11.3	4.14	0.001	35

The t statistic reveals that the significant difference between the groups resides in the normal-anxious comparison. The recovered depressed group were found to have a lower test anxiety score than when ill and were not significantly different from the normal group or the anxious group. As with the I-E scale, the recovered depressed group's scores on the TAS placed them mid-way between the anxious and the normal group. Thus, hypothesis 3, iib was not confirmed, although the results were in the predicted direction.

(iii) Beck Story Completion Test (BSCT)

The mean scores on negative thinking (BSCT) are shown in table 6.59.

Table 6.59 BSCT scores

Groups	Range	Mean	SD	
R.Dep.	22-67	40.2	8.6	$F = 11.33$
Anxious	29-92	56.35	18.72	$p < 0.001$
Normal	11-52	35.05	8.6	(df = 49)

Table 6.60 shows the results of the t tests applied to the individual group comparisons.

Table 6.60 Differences between mean BSCT scores

Comparisons	Diff. between means	t	p	df
R.Dep. v Normal	5.15	1.08	NS	33
R. Dep. v Anxious	16.15	3.27	0.01	30
Normal v Anxious	21.3	4.6	0.001	35

The analysis of negative thinking (BSCT) shows that unlike the depressed group, the recovered depressed group did not have significantly higher negative thinking scores than the normal controls. This may imply that when well the depression-prone individual does not think any more negatively than his normal counterpart. Thus, hypothesis 3iii b was not confirmed. Moreover, the recovered depressed subjects had significantly lower negative thinking scores than the anxious control subjects.

(iv) Judgement discrepancy (JD)

The mean scores on judgement discrepancy are given in table 6.61.

Table 6.61 JD scores

Groups	Range	Mean	SD	
R.Dep.	(-2)-(+3)	0.06	1.39	F = 0.525
Anxious	(-5)-(+4)	-0.64	2.52	NS
Normal	(-5)-(+5)	-0.50	2.00	

(v) Expectancy Discrepancy (ED)

The mean scores on expectancy discrepancy are shown in table 6.62.

Table 6.62 ED scores

Group	Range	Mean	SD	
R.Dep.	0-3	0.33	0.90	
Anxious	0-2	0.18	0.53	F = 1.2 NS
Normal	0-3	0.60	0.99	

(vi) Aspiration Discrepancy (AD)

The mean scores and group statistics on aspiration discrepancy are shown in table 6.63.

Table 6.63 AD scores

Groups	Range	Mean	SD	
R.Dep.	0-10	4.67	3.24	
Anxious	0-13	6.47	3.99	F = 3.56 $p < 0.05$
Normal	0-10	3.45	3.05	(df = 49)

Table 6.64 shows the results of the t test applied to the individual group comparisons.

Table 6.64 Differences between mean AD scores

Comparisons	diff. between mean	t	p	df
R.Dep. v Normal	1.22	1.04	NS	30
R.Dep. v Anxious	1.80	1.48	NS	33
Normal v Anxious	3.02	2.67	0.05	35

On all the scores of performance estimate, expectancy and aspiration (tables 6.61-6.64), there was no significant difference between the groups except for the previously mentioned difference between the normal and anxious group on aspiration discrepancy. It is, however, interesting to note that JD for the recovered depressed was positive though small, reflecting an overestimate of performance.

C. DEPRESSED v RECOVERED DEPRESSED

Change scores were calculated (Dep. - R.Dep.) for the depressed subjects when depressed and when recovered. This change score was then tested against zero to see if there had been any significant change. The procedure used the student's t test only.

(1) Difference in General and Background

The mean scores with the level of significance of the change in the general and background variables are given in table 6.65. As clearly expected, the subjects reported significantly less depression on recovery. Moreover their self reported level of anxiety was also lower. However there was no significant change found in any of the background electrodermal activity measures.

Table 6.65 Background and general scores

Dep.-R.Dep.	Mean	t	p	df
BDI	13.40	3.8	0.005	14
GA	3.46	3.87	0.005	14
SFTAS	-1.68	1.30	NS	14
SFPS	-0.62	1.20	NS	14
SFVI	-0.35	0.80	NS	14

(2) Attentional and perceptual differences

The means and level of significance on the attentional and perceptual sensitivity variables are given in table 6.66.

Table 6.66 Attentional and perceptual scores

Dep.-R.Dep.	Mean	t	p	df
PREL	-0.53	0.49	NS	14
PRER	-0.06	0.06	NS	14
PREL-PRER	-0.47	0.41	NS	14
DUL	-0.47	0.53	NS	14
DUR	-0.06	0.08	NS	14
DUL-DUR	-0.4	0.4	NS	14
TL	-1.33	0.74	NS	14
TR	-0.26	0.18	NS	14
TL-TR	-0.87	0.82	NS	14
SENL	0.63	1.13	NS	14
SENR	0.32	0.48	NS	14
SENR-SENL	0.30	1.12	NS	14

These results showed that there were no significant differences on any of the attentional and perceptual parameters. One interesting observation that does present itself is that the smallest change always occurred in the right (failure) modality and was reflected both in the psychophysiological and perceptual sensitivity data. It is difficult to say much about such a finding at this stage since all the results are not significant. However, an examination of the means suggests that as depression lessens, the individual becomes more attentive to positive cues and events rather than less attentive to negative cues and events.

(3) Attitudinal and cognitive changes

The means and level of significance for the measures on attitude changes and other cognitive changes are given in table 6.67.

Table 6.67 Attitudinal and cognitive scores

Dep.-R.Dep.	Mean	t	p	df
I-E	2.0	2.06	NS	14
TA	4.0	1.30	NS	14
BSCT	9.13	2.7	0.05	14
JD	0.33	0.86	NS	14
ED	0.33	0.28	NS	14
AD	-0.53	0.50	NS	14

The results show that the only significant change to occur on these variables was the degree of negative thinking, although I-E only just missed bein

significant at the 0.05 level. It will be noticed that test anxiety (TA) was far from being changed on recovery. It may be that while negative thinking acts as a mechanism that can induce depression, the proneness to think negatively, i.e. a vulnerability factor, may partially reside in high test anxiety and fear of failure

The lack of any significant differences on the performance estimate scores is interesting since they imply no change in any bias that might exist in the evaluation of performance ability to improve and level of aspiration. However, it is not possible to derive any particular conclusion from these performance estimate scores because of the general absence of significant results in this measure.

III. SUMMARY OF RESULTS AND SHORT DISCUSSION

Tables 6.68 and 6.69 provide a summary of results of the between group analyses.

Table 6.68 shows the results in three columns of the comparisons

Depressed (Dep.) versus Normal

Depressed (Dep.) versus Anxious

Anxious versus Normal

Table 6.69 shows the results in three columns of the comparisons

Recovered Depressed (R.Dep.) versus Normal

Recovered Depressed (R.Dep.) versus Anxious

Recovered Depressed (R.Dep.) versus Depressed (Dep.)

All the parameters measured are listed in column 1. The group depicted on the left in columns 2 - 4 is always the group with the highest score or value on that parameter and the > sign is used to show this.

The absence of the groups in a column, shown by —, implies no significant difference was found for this comparison.

Following each table is a written summary of the results.

Table 6.68 Summary of results: illness comparisons

1 Groups compared	2 Depressed vs Normal	3 Depressed vs Anxious	4 Anxious vs Normal
Variables			
General and Background			
BDI	Dep. > Normal	Dep. > Anxious	Anxious > Normal
GAS	Dep. > Normal	-	Anxious > Normal
SFTAS	Normal > Dep.	Anxious > Dep.	-
SFPS	Normal > Dep.	Anxious > Dep.	-
SFVI	-	Anxious > Dep.	Anxious > Normal
Attentional and perceptual			
PREL	Normal > Dep.	Anxious > Dep.	-
PRER	-	-	-
PREL-PRER	-	-	-

1	2	3	4
Groups compared	Depressed vs Normal	Depressed vs Anxious	Anxious vs Normal
Variables			
DUL	-	Anxious > Dep.	-
DUR	-	-	-
DUL-DUR	-	-	-
TL	Normal > Dep.	Anxious > Dep.	-
TR	-	-	-
TL-TR	-	-	-
SENL	Dep. > Normal	-	-
SENR	-	-	-
SENR-SENL (SEN. diff.)	Normal > Dep.	-	Normal > Anxious
Attitudinal and cognitive			
I-E	Dep. > Normal	-	Anxious > Normal
TAS	Dep. > Normal	-	Anxious > Normal
BSCT	Dep. > Normal	-	Anxious > Normal
JD	-	-	-
ED	-	-	-
AD	-		Anxious > Normal

Comparisons of the depressed subjects when ill with the two control groups show that the depressed group was significantly more depressed than the other two groups. In the measure of anxiety the depressed group were found to be no different from the anxious group and were significantly more anxious than the normal control

group.

The background measures of electrodermal activity showed that during both the completion of the test anxiety scale and during the problem solving task, the depressed group had a significantly lower level of spontaneous fluctuation activity per minute than the two control groups. These results are as expected, although anxious depressives are known to have higher spontaneous fluctuation activity than normals on an habituation test (Lader, 1975). During the light detection (or vigilance) part of the experiment the depressed group's spontaneous fluctuation activity did not differ from that of the normal group, although both the normal group and the depressed group had significantly lower electrodermal activity than the anxious control group over this period.

The attentional and perceptual parameters showed that electrodermal activity to a positive (success) event was the main measure that differentiated the groups. In what has been called the 'anticipatory epoch' (the five seconds before a signal indicating success or failure was presented) the depressed group were found to exhibit significantly less electrodermal fluctuations to 'anticipated' success, i.e. a positive event, than either the normal or anxious control groups. Moreover, during the actual five second presentation of the success signal (a left light), the depressed group showed significantly less electrodermal fluctuations than the anxious group. An examination of the two measured epochs combined, that is electrodermal fluctuations in the five seconds before a signal was presented and in the five seconds during the actual presentation of the signal, revelaed that again it was the electrodermal fluctuation to the (success) positive event, i.e. the left light, that differentiate the groups. The depressed group was found to have signifcantly less electrodermal

fluctuations over these combined measured epochs for success than either the normal group or the anxious group. It is important to point out that none of the measured epochs to the signalled failure event (the right light) revealed any significant difference between the groups. It seems that the depressed subjects are only distinguishable on their responses to a positive success event, but respond in a similar fashion to negative failure events as normal and anxious subjects. Although this seems a reasonable conclusion, it is possible that the differences in background responding during the problem-solving task may have affected this result. However, if this were the case, the background level of responding would be expected to produce significant differences for the failure event, i.e. the presentation of the right light as well as for the success event, i.e. the left light. Since a difference between the groups was found only in one direction (to the left light), it seems justified to argue that the depressed group does appear different in its electrodermal activity to the success event. However, the problem of different background electrodermal activity levels does unfortunately make this conclusion more tentative than if the background levels had shown no significant difference.

On all the attentional psychophysiological measures, the anxious group was found to respond in a similar (non-significant) manner to the normal control group.

The perceptual sensitivity measures showed that the depressed group had a significantly higher detection threshold for a light which had previously signalled a positive event compared to the normal controls. On the other hand an examination of the detection thresholds to a light which had previously acted as a signal for a negative event showed that there were no significant differences between the groups.

As in the case of the electrodermal measures, the perceptual sensitivity data tend to support the view that it is in the responding to positive events that depressives are distinguishable from normal control and anxious control subjects. The implications of these results will be further discussed in the final chapter. It should be pointed out, however, that on the two perceptual sensitivity measures, the depressed group did not significantly differ from the anxious controls.

An examination of the difference in perceptual detection thresholds, designed to find out whether the groups were different in their bias in perceptual sensitivities, revealed that the normal group tended to have a positive bias, that is they had a lower detection threshold for a signal that had been paired with a positive event than they did for a signal that had been paired with a negative event. The normal group were found to have a significantly higher positive bias than either the anxious group or the depressed group. However, this measure did not significantly discriminate between the anxious and depressed group. Again the implications of this finding will be discussed more fully in chapter nine.

The attitudinal and cognitive measures revealed that on the measure of externality both the depressed group and the anxious group were significantly more external than normal controls. On the measure of test anxiety, also used as a measure of fear of failure, the depressed group and the anxious group scored significantly higher than the normal controls. On the measure of negative thinking, again the depressed group and the anxious group were found to have significantly more negative thinking than the normal controls. However, on the measures of externality and also test anxiety, the anxious group and depressed group were not significantly different. On the Beck story completion test, a questionnaire designed to measure depressed thought content, the out-patient anxious

group scored marginally higher than the in-patient depressed group.

The performance estimate scores produced disappointing results. Although all the differences between the normal controls and the depressed group were in the expected direction, none reached significance. Although the majority of depressed patients did underestimate the number of questions they got correct during the problem solving task, two patients wildly over-estimated. The only significant result found on these measures was between the normal group and the anxious group on aspiration discrepancy.

Table 6.69 Summary of results: recovery comparisons

1	2	3	4
Groups compared	R.Dep. vs Normal	R.Dep. vs Anxious	Dep. vs R.Dep.
Variables			
General and background			
BDI	R.Dep. > Normal	-	Dep. > R.Dep.
GAS	R.Dep. > Normal	Anxious > R.Dep.	Dep. > R.Dep.
STFAS	-	-	-
SFPS	-	-	-
SFVI	-	Anxious > R.Dep.	-
Attentional and perceptual			
PREL	-	-	-
PRER	-	-	-
PREL-PRER	-	-	-

1 Groups compared	2 R.Dep. vs Normal	3 R.Dep. vs Anxious	4 Dep. vs R.Dep.
Variables			
DUL	-	-	-
DUR	-	-	-
DUL-DUR	-	-	-
TL	-	-	-
TR	-	-	-
TL-TR	-	-	-
SENL	-	-	-
SENR	-	-	-
SENR-SENL (SEN diff.)	Normal > R.Dep.	-	-
Attitudinal and cognitive			
I-E	-	-	-
TAS	-	-	-
BSCT	-	Anxious > R.Dep.	Dep. > R.Dep.
JD	-	-	-
BD	-	-	-
AD	-	-	-

The results of the comparisons between the depressed subjects after recovery with themselves when ill and with the normal and anxious control groups show that

while the level of self-reported depression was significantly lower on recovery, it was still higher than the normal controls. Moreover, although the level of depression was lower for the recovered depressives compared to the anxious group, this difference was not found to be statistically significant. The level of anxiety was also found to be significantly lower on recovery from depression than when depressed, and was significantly lower than for the anxious control group. However, the recovered depressives had a significantly higher level of anxiety than the normal control group.

The background level of electrodermal activity was not found to have significantly changed over recovery (i.e. comparing the depressives when ill and after recovery). On the other hand, the significantly lower background level of spontaneous fluctuations found in the depressed group compared to the normal and anxious control groups during the completion of the test anxiety scale and during the problem solving task, has disappeared on recovery. The anxious group, however, was found to have a significantly higher level of electrodermal spontaneous fluctuations during the light detection (vigilance) part of the experiment compared to the recovered depressive.

The electrodermal measures used as an attentional index to positive and negative events show that although there were no significant changes between the depressives when ill and after recovery, a sufficient change has occurred to remove the significant difference found between the depressives when ill and the two control groups on the anticipatory success (left light) activity and on the total measured electrodermal activity to the success (left) light. The measure of perceptual sensitivity threshold to the left light also shows that no significant change on recovery from depression had occurred, although sufficient change had taken place for the recovered depressed

group's left light sensitivity threshold to be no longer significantly different from the normal control group's. On the other hand, the measure of bias in perceptual sensitivity thresholds to a light that had signalled success and a light that had signalled failure, showed that not only had no significant change occurred on this variable on recovery from depression but also that the recovered depressives still had a significantly smaller positive bias than the normal control group. On all three measures of perceptual sensitivity, the recovered depressives were found to be not significantly different from the anxious control group.

The attitudinal and cognitive parameters showed that on the measure of externality (I-E scale) the recovered depressives had become more internal to the extent of removing the statistical difference found between them when depressed and the normal controls. However, this was not a sufficient change to produce a significant difference between the depressed subjects' scores when ill and on recovery. The test anxiety scale provided similar results, that is a sufficient reduction in the level of test anxiety had occurred on recovery to remove the statistical difference found between the depressives' scores when ill and the normal control scores but insufficient change had occurred to produce any significant difference between the level of test anxiety when depressed and on recovery. In fact, the only measure of the attitudinal and cognitive factors that significantly changed on recovery from depression was negative thinking. On this variable a significant change was found, which, in addition, was sufficient to remove the statistical difference found between the normal control group and the depressed group when ill, i.e. the recovered depressives were found to think no more negatively than the normal controls.

The performance estimate scores of judgement discrepancy, expectancy

discrepancy and aspiration discrepancy did not change on recovery from depression. In fact, all the changes on these three variables had 't' statistics below 0.9. Thus, these change scores did not even approach significance. However, since these measures did not differentiate between the depressed group when ill and the two control groups, it is difficult to interpret these results. Nevertheless, it can be seen that the aspiration discrepancy score remains higher in the recovered depressed than in the normals although not significantly so. Further discussion of these results are given in chapter nine.

CHAPTER SEVEN

EXPERIMENT 2

In chapter four it was pointed out that Beck (1976) has argued that certain attitudes and rules can predispose individuals to excessive sadness and depression. Some of these rules include attitudes such as:

1. In order to be happy I have to be successful in whatever I undertake

3. If I'm not on top, I'm a flop

5. If I make a mistake, it means I'm inept

(Beck, 1976, P. 255).

It was considered that one useful test of this aspect of Beck's theory of depression was to screen a normal population for such attitudes and to discover whether these individuals would be more prone to depression than individuals who did not have such attitudes. As a first step towards such a test of the theory, it was pointed out that the rules and attitudes which Beck argues are important predisposing factors to depression tend to centre on the common theme of the avoidance of failure. This avoidance may be derived from the belief that certain secondary reinforcers (e.g. love, respect) will be withdrawn on failure. The theory behind such reasoning was outlined in chapter four. The advantage of this approach is that, as a first attempt to test this part of the theory, it is possible to screen normal subjects for a high fear of failure, while it would be far more difficult to screen for specific rules and attitudes.

I. AIMS

The aims of this study were thus to identify high fear of failure subjects and low fear of failure subjects in a normal population and to examine whether the former group had a greater tendency to be more vulnerable to depression than the latter group, as measured on the parameters of experiment one.

The general questions to be answered can thus be summarised as follows:

1. Do high fear of failure subjects compared with high hope of success subjects differ on their ratings of depression and anxiety?

2. How do high fear of failure subjects differ from high hope of success subjects on the parameters of attention and perception to positive and negative events, on the various attitudinal and cognitive measures used in this study?

3. Are fear of failure subjects more vulnerable to depression than hope of success subjects?

II. DESIGN

The design involved distributing a specially devised inventory assessing the relative strength of approach and avoidance motivations to a large number of normals. From the completed inventories returned, the highest and the lowest responders were selected to take part in the same experiment as outlined in chapter five.

III. METHOD

A. SELECTION OF SUBJECTS

The subjects who took part in this experiment were chosen from a normal non psychiatric population. Participation in either a high fear of failure group or a high hope for success group was determined by the scores of the subjects on the Success-Failure Inventory (SFI) (McReynolds and Guevara, 1967).

In order to explain the selection of subjects in more detail, it is necessary to discuss the SFI.

A search of the literature in the area of fear of failure revealed that McReynolds and Guevara (op. cit.) had devised a scale, the SFI, which was designed to measure the relative strengths of fear-avoidant motivation (M_{fa}) and success-attainment motivation (M_s) in different individuals.

The inventory consists of twenty-two items with a true/false format. Eighteen of the items are keyed such that a true or false response indicates a failure-avoidant motivation (M_{fa}). The difference between the number of possible M_s and M_{fa} responses occurs because not all of the items are considered to be overlapping. Examples of this inventory and the scoring system are as follows:

1. I have a tendency to give up easily when I meet difficult problems true/false

If the individual gave a false response to this item he would receive a one point score for the success-attainment motivation (M_s). If the individual gave a true response to this item, he would receive a one point score for the failure-

avoidant motivation (M_{fa}).

20. I enjoy competitive sports true/false

In this case if the individual gave a false response to this item, he would receive a one point score for the failure avoidant motivation (M_{fa}). If the individual gave a true response to this item he would receive a one point score for the success-attainment motivation (M_s).

On some items, only one response scores a point. For example,

7. Success is too transient an experience for a person to sacrifice much to obtain it true/false

In this case a true response would not score any points, although a false response would score one point for the success-attainment motivation (M_s). The overall score of this scale is calculated by subtracting the number of points given to success-attainment motivation (M_s) from the number of points given to failure-avoidant motivation (M_{fa}), i.e. $M_s - M_{fa}$.

McReynolds and Guevara (1967) point out that this scale does not measure the absolute strengths of the motives to approach success-attainment or the motive to avoid failure, but measures the relative orientations. Thus a person with a high positive score in comparison with a person with a lower positive score could be said to be relatively less motivated to avoid failure than the latter. In other words, of the two individuals, the latter has a relatively higher failure avoidant motivation than the former.

It was considered that by distributing this inventory to a large number of normal individuals, it would be possible to discriminate those individuals who had a relatively high motivation to success-attainment from those

individuals who had a relatively high motivation to avoid failure.

One hundred and fifty inventories were distributed to normal subjects, of varying occupational and socio-economic status. Completed questionnaires were returned from hospital workers, factory (engineering) workers, post-office technicians, farm workers, shop workers, housewives, school teachers and other miscellaneous occupations.

Of the 150 questionnaires distributed 119 were returned. The means and group statistics of this population for this inventory are presented in table 7.1.

Table 7.1 SFI scores of whole population

Total normal group	Range	Mean	S.D.
N = 119	(-7) - (+18)	7.6	5.5

A covering letter was attached to each questionnaire which informed the individual that he or she may be required to take part in a short experimental session at the MRC Unit at the Thomas Clouston Clinic. Of the forty subjects asked to take part in this experiment on the basis of their scores on the SFI, 34 individuals agreed to act as subjects.

The cut-off scores which defined whether a subject would be tested as a high fear of failure or a high hope of success (i.e. high success-attainment motivation) subject was decided at the beginning of this project. Although it would have been desirable to have set the cut-off scores once all the completed inventories had been returned, this was not possible for two reasons. Firstly, not all the inventories could be distributed at the same time (in fact it took over three months to distribute the 150 questionnaires)

because the author wanted to get as heterogeneous a group as possible. Secondly it was considered desirable to test individuals as soon as possible after they had completed and returned their questionnaires to ensure co-operation and to save time.

As mentioned in the aims section, the idea was to test those individuals with a relatively high fear of failure compared to those with a relatively high hope of success, on the parameters used in the first experiment. In view of this general aim and the considerations mentioned above, the cut-off score for the two groups was arbitrarily determined. This decision was aided by McReynolds and Guevara's (1967) finding that normals tended to cluster at the positive top end of the scoring scale. In addition, the first twenty-five questionnaires returned acted as a general indication of the spread in scoring. This led to assymetrical cut-off scores.

(1) Fear of failure group (F.F.)

Subjects who scored 0 and below on the SFI were considered eligible to be placed in the high fear of failure (F.F.) group. Of the 119 questionnaires returned, 19 subjects scored 0 or below, i.e. indicating $M_s < M_{fa}$, as measured by this inventory. All nineteen individuals were approached and asked to take part in a short experiment, of which sixteen gave their consent.

(2) Hope of success group (H.S.)

Subjects who scored 12 or above on the SFI were considered eligible to be placed in the high hope of success (H.S.) group, indicating that $M_s > M_{fa}$ as measured by the SFI. Of the 119 questionnaires

returned, 26 subjects scored 12 or above, of these 18 agreed to participate in the experiment.

The mean and group statistics for the two groups selected to participate in the experiment, on the SFI, are given in table 7.2.

Table 7.2 SFI scores

Groups	(N)	Range	Mean	S.D.	
H.S.	18	12 - 18	14.27	1.99	F = 655.8
F.F.	16	-7 - 0	-3.56	2.06	$p < 0.001$
					df = 32

Thus, it can be seen that the F.F. group had a significantly higher motivation to avoid failure as measured by the SFI than the H.S. group, whicl for this study is the most important concern, rather than absolute cut-off scores. Moreover, as McReynolds and Guevara (1967) point out, the SFI is a relative measure and not an absolute measure.

(3) Age

The mean and group statistics for age are shown in table 7.3

Table 7.3 Age scores

Groups	(N)	Range	Mean	S.D.	
H.S.	(18)	19-47	27.67	7.46	N.S.
F.F.	(16)	20-48	30.5	8.28	

One-way analysis of variance revealed no significant difference between the ages of the two groups.

(4) Sex

The male/female distribution of the two groups is shown in table 7.4.

Table 7.4 Sex differences

	Male	Female
H.S.	9	9
F.F.	10	6

$\chi^2 = 1.08$, N.S.

χ^2 revealed no significant difference in the sex composition of the two groups.

B. MEASURES

The format and scoring system of the SFI was explained in the previous section. The reliability of this scale was found to be 0.82 (McReynolds and Guevara, 1967). These authors used this scale to measure attitudes to success and failure in a schizophrenic population compared to a normal control group and a mixed neurotic group. The 52 mixed neurotic consisted of 21 anxious patients, 21 depressed patients and 10 mixed neurotic reactions. The results of their comparisons are given in table 7.5.

Table 7.5 SFI scores (from McReynolds and Guevara, 1967)

Group	N	Mean	S.D.
Normals	103	10.67	4.28
Neurotics	52	4.50	6.56
Remitted schizophrenics	32	4.44	6.66
Active schizophrenics	104	1.19	5.93

Two results are of interest here. (1) The mixed neurotic group had a significantly higher motivation to avoid failure relative to the normal control group, and (2) did not significantly differ from remitted schizophrenics. Since the neurotic patients were a mixed group, it is difficult to draw any useful conclusions with regard to depression. However, it may be that a high fear of failure is a factor relevant to psychopathology in general rather than specifically to depression. This point will be discussed more fully later.

Once a subject had been selected for either group, all measures administered in the experimental session were the same as in Experiment One (see chapter five, pp.

C. PROCEDURE

When an individual agreed to take part in this experiment, an arrangement was made as to the most convenient time for the subject. This was usually early evening due to economic and domestic commitments. Once a subject arrived at the MRC Unit Metabolic Ward, the procedure was exactly the same as described in chapter five (pp. 163-166).

D. CONDITIONS OF TESTING

The conditions remained the same as described in chapter five (pp 167-169).

None of the subjects tested admitted to the use of any psychophysiologically disruptive drugs.

E. APPARATUS

The apparatus remained the same, as described in chapter five (pp. 169-172

F. ANALYSIS OF THE DATA

(1) Psychophysiological

The psychophysiological analysis of the data was the same as described in chapter five (pp. 172-176).

(2) Statistical analysis

The statistical analysis involved Pearson product moment correlations and one-way analysis of variance. This analysis is described in chapter five (pp. 176-177).

G. SPECIFIC HYPOTHESES

(1) Background and general

(i) It was predicted that the F. F. group would have significantly higher BDI scores than the H.S. group.

(ii) It was predicted that the F.F. group would have significantly higher general anxiety than the H.S. group.

(iii) No prediction was made concerning the background level of

spontaneous fluctuations.

(2) Attentional and perceptual

(i) It was hypothesised that on the measures used to examine the parameters of attentional and perceptual sensitivity to positive and negative events, the F. F. group would show the more negative bias.

(3) Attitudinal and cognitive

(i) It was hypothesised that the F. F. group would have the highest I-E score and would be significantly different from the H.S. group.

(ii) It was hypothesised that the F. F. group would have a significantly higher level of test anxiety compared to the H.S. group.

(iii) It was hypothesised that the F. F. group would have a significantly higher level of negative thinking as measured by the BSCT compared to the H.S. group.

(iv) It was hypothesised that the F. F. group on the problem solving task would (a) underestimate their performance, (b) be the less likely to expect to improve their performance, and (c) would have the higher aspiration score.

CHAPTER 8

RESULTS

As for chapter six, the results reported in this chapter are presented in two parts: (1) within group analyses and (2) between group analyses.

WITHIN GROUP ANALYSES

This section reports on the intercorrelations between all the variables in the fear of failure group (F.F.) and the hope of success group (H.S.) independently. In addition, since both groups of subjects are derived from a normal non-psychiatric population, the first set of correlations presented examines the intercorrelations of the combined groups.

BETWEEN GROUP ANALYSES

A one-way analysis of variance was applied to all variables for comparing differences between the fear of failure group versus the hope of success group (F.F. v H.S.).

All results are presented under the three sub-headings: (1) General and background, (2) Attentional and perceptual, (3) Attitudinal and cognitive.

I. WITHIN GROUP ANALYSES

Pearson's product-moment correlation coefficients were calculated to investigate the inter-relationship between variables.

Each table shows only the significant correlations (with level of significance) for

the variables under each sub-heading, e.g. (1) General and background, etc. The correlations are examined in the order of (A) Combined group intercorrelations (F.F. + H.S.), (B) Fear of failure group intercorrelations (F.F.), (C) Hope of success group, intercorrelates.

A. Total population intercorrelations

1. Background and general

Table 8.1 shows the significant correlations for the general and background variables.

Table 8.1 General and background significant correlations

Age	I-E -0.42 *, JD -0.38 *, DUR 0.35 *
BDI	GA 0.80 ***, BSCT 0.77 ***, TAS 0.61 ***, SFI -0.60 ***, I-E 0.49 ***
GA	BDI 0.80 ***, BSCT 0.71 ***, SFI -0.63 ***, TAS 0.60 ***, I-E 0.46 *, DUL -0.41 *, SEN.diff. -0.36 *
SFTAS	I-E -0.35 *
SFPS	None
SFVI	None

*** $p < 0.001$, ** $p < 0.01$ * $p < 0.05$ $n = 34$, d.f. = 33

From table 8.1 it can be seen that age was negatively correlated with externality (I-E) and judgement discrepancy (J.D.) but positively correlated with electrodermal activity during the presentation of the failure signal, the right light (DUR). These correlations were significant at the 5% level.

It can also be seen that level of depression (BDI) was highly correlated at the 0.1% level of significance with general anxiety (GA), with negative thinking (BSCT) and fear of failure as measured by both the TAS and SFI at the 0.1% level of significance and at the 1% level of significance with externality (I-E).

The pattern of correlations was found to be similar for general anxiety and level of depression since GA was also correlated at the 0.1% level with negative thinking (BSCT) and fear of failure as measured by the TAS and SFI. General Anxiety was correlated at the 1% level with externality (I-E) but correlated negatively with electrodermal activity during the presentation of the left (success) light (DUL) and the difference between the perceptual sensitivity measures to the left and right lights (SEN diff.).

The mean number of spontaneous fluctuations during the completion of the TAS (SFTAS) revealed a significant negative correlation with externality (I-E) at the 5% level. The other two background measures showed no significant correlations with any other variable.

2. Attentional and perceptual correlations

Table 8.2 shows the significant correlation for the attentional and perceptual parameters of the two groups F.F. and H.S. combined.

Table 8.2 Attentional and perceptual significant correlations

PREL	None
PRER	SENR -0.37 *
PREL-PRER	SENR -0.36 *
DUL	I-E -0.42 *, TAS -0.42 *, GA -0.41 *
DUR	I-E -0.40 *, age 0.35 *
DUL-DUR	None
TL	I-E -0.40 *
TR	None
TL-TR	SEN. diff. 0.36 *

SENL	SEN.diff. -0.42 *
SENR	PREL -0.37 *, PREL-PRER 0.36 *
SENR-SENL	SENL -0.42 *, GA -0.36 *, TL-TR 0.36 *

* $p < 0.05$ n = 34 d.f. = 33

All the correlations reported in table 8.2 occurred at the 5% level. It can be seen that 'anticipatory' electrodermal activity to the right (PRER) and the difference between anticipatory electrodermal activity to the left and right lights (PREL-PRER) were negatively correlated with the perceptual sensitivity to the right light (SENR).

The electrodermal activity during the left light presentation (DUL) correlated negatively with externality (I-E), test anxiety (TAS) and general anxiety (GA) and the electrodermal activity during the presentation of the right light (DUR) was negatively correlated with externality (I-E) and positively correlated with age. Moreover a negative correlation was found between the total measured electrodermal activity to the left light (TL) and externality (I-E). The difference between total measured electrodermal activity to the left and right lights (TL-TR) was found to be correlated with the difference in the perceptual sensitivity measures to the left and right light (SENR-SENL, i.e. SEN.diff.). As in the case of the depressed and recovered depressed group correlations (chapter 6, tables 6.2 and 6.5) this finding again tends to indicate that a bias in responding observed in one modality may be related to biases in other modalities.

The perceptual sensitivity measure to the left light was negatively correlated

with the difference in the perceptual sensitivity measures to the left and right lights (SEN.diff.), and the latter was found to be negatively correlated with general anxiety, indicating that general anxiety may have a tendency to reduce any positive perceptual sensitivity bias that appears to occur in a normal population (see chapter 6, tables 6.30 and 6.31).

3. Attitudinal and cognitive

Table 8.3 shows the correlations for the attitudinal and cognitive variables.

Table 8.3 Attitudinal and cognitive significant correlations

I-E	BSCT 0.53***, BDI 0.5**, SFI -0.48**, GA 0.46**
	age -0.42*, DUL -0.42*, DUR -0.40*, TL -0.40**
TAS	BDI 0.61***, GA 0.6***, BSCT 0.57***, SFI -0.56***
	DUL -0.42* I-E 0.35*
BSCT	BDI 0.77***, GA 0.71***, SFI -0.69***, TAS 0.57***, I-E 0.53*
SFI	BSCT -0.69***, GA -0.63***, BDI -0.60***, TAS -0.56***
	I-E -0.48**, ED 0.34*
JD	AD -0.63***, age -0.38*
ED	SFI 0.34*
AD	JD -0.63***

*** $p < 0.001$, ** $p < 0.01$ * $p < 0.05$ n = 34, d.f. = 33

From table 8.3 it can be seen that externality (I-E) correlated with negative thinking (BSCT) at 0.1% of significance, at the 1% level with levels of depression (BDI), fear of failure (SFI) and general anxiety (GA) and at the 5% level with test anxiety (TAS). Externality was found to be negatively correlat ed with age,

activity during the left (signalling success) light presentation (DUL) and during the right (signalling failure) light presentation (DUR), the mean number of spontaneous fluctuations during the completion of the test anxiety scale (SFTAS) and the total measured electrodermal activity to the left light (TL) at the 5% level and at the 1% level with fear of failure (SFI). It should be pointed out here that the lower the SFI score, the higher is the fear of failure, as explained in chapter seven.

Test anxiety (TAS) correlated at the 0.1% level of significance with level of depression (BDI), general anxiety (GA), negative thinking (BSCT) and negatively with fear of failure (SFI). (This last negative correlation between the two fear of failure measures is as expected because the two scales are measured from opposite directions as mentioned earlier.) Test anxiety also correlated negatively with electrodermal activity during the presentation of the left (signalling success) light (DUL) at the 5% level.

Apart from the high (0.1% level) negative correlations already mentioned between fear of failure (SFI) and negative thinking, general anxiety, level of depression, test anxiety and externality (the latter at the 1% level); fear of failure was positively correlated at the 5% level with expectancy discrepancy (ED) indicating that the lower the SFI score (i.e. the higher the fear of failure) the lower was the perceived ability to be able to improve in the problem solving task.

Negative thinking (BSCT) was found to be highly correlated (0.1% level) with the level of depression (BDI), general anxiety (GA), fear of failure (SFI), test anxiety (TAS) and externality (I-E).

Aspiration discrepancy (AD) and judgement discrepancy (JD) were found to be negatively correlated at the 0.1% level and in addition judgement discrepancy showed a small (5% level) negative correlation with age.

B. Fear of failure group

The next three sections examine the correlations within the fear of failure group only (N = 16).

1. General and background

Table 8.4 shows the significant correlations for the general and background variables, in the fear of failure group.

Table 8.4 General and background significant correlations

Age	AD 0.69 **, I-E -0.61 *, JD -0.52 *
BDI	GA 0.76 ***, BSCT 0.70 **, SFI -0.51 *, TA 0.50
GA	BDI 0.76 ***, BSCT 0.67 **
SFTAS	None
SFPS	SFI 0.56 *
SFVI	None

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$ n = 16 d.f. = 15

From table 8.4 it can be seen that age was correlated with aspiration discrepancy (AD) at the 1% level of significance and negatively correlated with externality (I-E) and judgement discrepancy (JD) at the 5% level.

Level of depression (BDI) was highly correlated with general anxiety (0.1%

level of significance), with negative thinking (BSCT) (1% level) and at the same level (5%) with both fear of failure (SFI) and test anxiety (TAS).

In addition to having a high correlation with the level of depression (BDI) general anxiety (GA) was also highly correlated with negative thinking (BSCT) (at the 1% level). The only background electrodermal activity measure that revealed a significant correlation was the mean number of spontaneous fluctuations during the problem solving task (SFPS) which was correlated with fear of failure (SFI) at the 5% level. This finding may indicate that a relatively high fear of failure tends to reduce automic responding under problem solving conditions.

2. Attentional and perceptual correlations

Table 8.5 shows the significant correlations for the attentional and perceptual variables in the fear of failure group.

Table 8.5 Attentional and perceptual significant correlations

PREL	None
PRER	None
PREL-PRER	ED 0.63 **
DUL	TAS -0.63 **, GA -0.61 *
DUR	TAS -0.68 **, SFI 0.56 *, I-E -0.50 *
DUL-DUR	None
TL	None
TR	None
TL-TR	None

SENL	None
SENR	None
SENR-SENL (SEN.diff.)	None

** $p < 0.01$ * $p < 0.05$ n = 16 d.f. = 15

It can be seen from table 8.5 that 'anticipatory' responding to both the left (success) light (PREL) and right (failure) light (PRER) did not correlate with any other variable. However, the difference between these two measures of responding PREL-PRER) correlated with expectancy discrepancy at the 1% level.

Both test anxiety (TAS) and general anxiety (GA) had a significant inverse relationship with electrodermal activity during the presentation of the left (success) light (DUL). Test anxiety, and externality (I-E) also had a negative correlation with electrodermal activity during the presentation of the right light (DUR). In addition the higher the fear of failure (the lower the SFI score) the lower was the degree of electrodermal activity during the presentation of the right (failure) light (significance level of 5%).

No significant correlation was found for the perceptual sensitivity measures.

2. Attitudinal and cognitive correlations

Table 8.6 shows the significant correlations for the attitudinal and cognitive variables in the fear of failure group.

Table 8.6 Attitudinal and cognitive significant correlations

I-E	SFI -0.57 *, BSCT 0.54 *, DUR -0.59 *
TAS	DUR -0.68 **, DUL -0.63 **, BDI 0.5 *
BSCT	BDI 0.70 **, GA 0.67 **, SFI -0.65 **, I-E 0.54 *

SFI	BSCT -0.65 **, I-E -0.57 *, SFPS 0.56 *, DUR 0.56 * BDI -0.51
JD	AD -0.78 ***, age -0.52 *
ED	PREL-PRER 0.63 **
AD	JD -0.78 ***, age 0.69 **

*** $p < 0.001$, ** $p < .01$ * $p < 0.05$ n = 16 d.f. = 15

From table 8.6 it can be seen that externality (I-E) correlated with fear of failure (SFI), negative thinking (BSCT) and negatively with electrodermal activity during the presentation of the right (signalling failure) light (DUR) at the 5% level of significance. As mentioned following table 8.5, test anxiety (TAS) had an inverse relationship with electrodermal activity during both the left and right presentation (DUL and DUR). In addition test anxiety correlated with level of depression (BDI) at the 5% level.

As mentioned earlier (negative thinking) BSCT was significantly correlated with level of depression (BDI), general anxiety (GA) and fear of failure (SFI) at the 1% level, and with externality at the 5% level.

Fear of failure (SFI), in addition to being correlated with negative thinking (BSCT) at the 1% level, was correlated at the 5% level with externality (I-E), background electrodermal activity during the problem solving task (SFPS), electrodermal activity during the presentation of the right light (DUR) and level of depression (BDI). The lower the SFI score the higher the (relative) fear of failure, thus a negative correlation implies a positive relationship between the degree of fear of failure and the correlated variable.

The three performance estimate scores showed that judgement discrepancy

(JD) was negatively correlated (at the 5% level) with age while aspiration discrepancy (AD) was positively correlated with age (at the 1% level). These two measures were also found to be negatively correlated with each other at the 0.1% level. Expectancy discrepancy (ED) was positively correlated at the 1% level, with bias in anticipatory electrodermal activity to the left (success) and right (failure) light (PREL-PRER).

C. Hope of success group

The next three sections show the significant correlations for the hope of success group.

1. General and background

Table 8.7 shows the significant correlations for the background and general variables in the hope of success group.

Table 8.7 Background and general significant correlations

Age	DUR 0.53 * I-E -0.51 *
BDI	SFPS 0.57 *, TAS 0.50 *
GA	None
SFTAS	I-E -0.62 **
SFPS	BDI 0.57 *
SFVI	SFI -0.48 *

** $p < 0.01$ * $p < 0.05$ $n = 18$ $d.f. = 17$

From table 8.7 it can be seen that age correlated with electrodermal activity during the presentation of the right light (DUR) and with externality (I-E) at the 5% level.

Level of depression (BDI) correlated with mean number of spontaneous fluctuations during the problem solving task (SFTAS) and test anxiety (TAS), both at the 5% level of significance. On the other hand, for this group general anxiety (GA) did not correlate with any other variable.

The background electrodermal activity measures showed that the mean number of spontaneous fluctuations per minute during the completion of the test anxiety scale (SFTAS) was negatively correlated with externality (at the 1% level) while the mean number of spontaneous fluctuations during the problem solving task (SFPS) was positively correlated, at the 5% level, with level of depression (BDI). In addition fear of failure was correlated with the mean number of spontaneous fluctuations during the light detection (vigilance) (SFVI) session, also at the 5% level.

2. Attentional and perceptual correlations

Table 8.8 shows the significant correlations for the attentional and perceptual variables in the hope of success group.

Table 8.8 Attentional and perceptual significant correlations

PREL	None
PRER	None
PREL-PRER	None
DUL	Age 0.53 *
DUR	None
DUL-DUR	None

TL	None
TR	None
TL-TR	None
SENL	None
SENR	None
SENR-SENL (SEN.diff.)	None

$^{*}p < 0.05$ n = 18 d.f. = 17

The only significant correlation found for these parameters was between age and electrodermal activity during the presentation of the right light (DUL) at the 5% level.

3. Attitudinal and cognitive

Table 8.9 shows the significant correaltions for the attitudinal and cognitive variables in the hope of success group.

Table 8.9 Attitudinal and cognitive significant correlations

I-E	SFTAS -0.62**, age -0.51*
TAS	BDI 0.51*
BSCT	SFI -0.52*, AD -0.52*
SFI	BSCT -0.52* SFVI -0.48*
JD	AD -0.50*
ED	None
AD	BSCT -0.52*, JD -0.50*

$^{**}p < 0.01$ $^{*}p < 0.05$ n = 18 d.f. = 17

This last table of correlations (table 8.9) shows that externality (I-E) was negatively correlated with both the mean number of spontaneous fluctuations during completion of the test anxiety scale (SFTAS) and age at the 5% level. Moreover as mentioned earlier test anxiety (TAS) and level of depression (BDI) were correlated at the 5% level.

Negative thinking (BSCT) correlated with fear of failure (SFI) and negatively with aspiration discrepancy (AD), both at the 5% level of significance.

Fear of failure (SFI), in addition to being correlated with negative thinking (BSCT) correlated negatively with the mean number of spontaneous fluctuations during the light detection (vigilance) part of the experiment (SFVI).

The performance estimate scores revealed that in addition to aspiration discrepancy (AD)being negatively correlated with negative thinking (BSCT), aspiration discrepancy (AD) and judgement discrepancy (JD) were also negatively correlated at the 5% level.

D. Short discussion

As mentioned in chapter six, at the 5% level of significance, one in twenty of the correlations reported here can occur by chance.

The results of the correlations show that for the combined groups and the fear of failure group, level of depression (BDI) was highly correlated with negative thinking (BSCT). This correlation, which was significant at the 0.1% level, confirms Weintraub's et al. (1974) finding of the relationship between mood and thinking. However, in this study as in their study, the subjects were from a non-psychiatric population. It will be remembered from the results in chapter six that this correlation does not appear to occur for clinically depressed subjects. Moreover, general anxiety in both the combined groups and the fear of failure group had a higher correlation with level of depression than did negative thinking. However, in both the combined groups and fear of failure group, general anxiety and negative thinking (BSCT) correlated highly with level of depression. In addition, both measures of fear of failure (SFI and TAS) were found to be correlated with the level of depression in the combined groups and fear of failure group. In fact, test anxiety was the only attitudinal variable that correlated with level of depression in the hope of success group.

Apart from correlating with the level of depression, general anxiety was found to correlate with a large number of variables in the combined groups. However, the separate group analyses revealed that in the fear of failure group, general anxiety correlated with level of depression and negative thinking, but did not correlate with any other variable in the hope of success group. It should be noted that no correlation was found

between test anxiety and general anxiety in either the fear of failure group or the hope of success group. Moreover, although in the combined group, general anxiety was found to be correlated with test anxiety, it was at a lower level than the correlation of general anxiety, with level of depression and negative thinking. This would seem to support Sarason's (1972) view that, although general anxiety and test anxiety do overlap, they nevertheless measure different things.

An examination of the correlations between the three variables, level of depression, general anxiety and negative thinking, reveal that all three are highly correlated with each other. Moreover it should be pointed out that the BSCT, a scale designed to measure depressive thought content, has again (see chapter six) been found to have a high correlation with general anxiety, which further raises the question of the specificity of depressive thought content. Either this scale is not good enough to measure specifically depressive thought content, or the idea that certain thoughts can be considered specific to depression is questionable.

The attentional and perceptual parameters did not yield many results that merit consideration here. However, as in the depressed subjects when ill (table 6.2) and on recovery (table 6.5), one of the measures used as an index of bias in electrodermal responding to success and failure was found to correlate with the measure of perceptual sensitivity bias. In this case the difference between total measured electrodermal activity to the left light (TL) and total measured electrodermal activity to the right light (TR), that is TL-TR, was found to be correlated

with the difference in perceptual sensitivity to lights that had signalled success (left light, SENL) and failure (right light, SENR), that is SENR-SENL (SEN.diff.). This finding appears to further confirm the idea, mentioned in chapter six, that a bias in the responding to one modality, or response system, may be observed in, and measurable in, other modalities, or response systems. Here it would seem that any increase or decrease in a positive or negative bias in electrodermal responding is paralleled by a change in the same direction as in the positive or negative bias in perceptual sensitivity to stimuli that had signalled positive (success) and negative (failure) events.

An examination of the attitudinal and cognitive correlations in the combined group's analysis shows that externality (I-E) was significantly correlated with negative thinking (BSCT), level of depression (BDI), fear of failure (SFI), general anxiety (GA) and test anxiety (TAS). In addition, externality correlated with a number of psychophysiological variables. However the individual group correlations revealed that in the hope of success group none of the above correlations with externality occurred except for a negative correlation with age and a positive correlation with background electrodermal activity during the completion of the TAS (SFTAS). In the fear of failure group, externality correlated with both fear of failure (SFI) and negative thinking (BSCT). This finding is of interest since it might be recalled (chapter six) that both the depressed group and the anxious group showed a correlation between externality and negative thinking, whereas neither the recovered depressed nor the normal controls did so. This may support the view that the more external an individual is the more negative his thinking is likely to be.

In all three analyses, test anxiety (TAS) correlated with level of depression. In both the hope of success group and the fear of failure group test anxiety correlated with BDI. This may suggest that test anxiety is a more general indicator of depression than, say, negative thinking, since in two groups chosen to differ on the level of fear of failure, test anxiety correlated with the level of depression (BDI) in both groups whereas other variables correlated with depression in one group or the other, but not in both.

It will be recalled (chapter 5, pp. 152-154) that the test anxiety scale has been used in a variety of studies as a measure of fear of failure. It is appropriate at this point, therefore, to consider the other measure of fear of failure that has been used in this study, that is the success/failure inventory (SFI). Although the combined group correlations showed SFI and TAS to be correlated, the individual group correlations revealed no significant correlation between these two measures. This absence of a correlation between two scales that are supposed to measure the same thing, in two groups, implies that the results using both scales should be treated cautiously. It may be, however, that the test anxiety scale is a specific (academic orientated) scale designed for students and not members of the general population, whereas the success/failure inventory is far more general and was designed for use both on a psychiatric population and the general population. In the combined group correlations the success/failure inventory was found to correlate with negative thinking (BSCT), general anxiety (GA), level of depression (BDI), test anxiety (TAS) and externality, all of which were found to correlate with test anxiety. The only additional correlation found using the SFI that did not occur with the TAS was with expectancy discrepancy. However, the individual

group correlations showed that whereas the test anxiety scale did not correlate with negative thinking (BSCT) in either the F. F. group or the H.S. group, the success/failure inventory did correlate with negative thinking (BSCT) in both these groups, and in the F. F. group also correlated with depth of depression (BDI).

Negative thinking (BSCT) has already been discussed in relation to other variables and therefore need not be discussed in detail again. However it should be noted that negative thinking was found to be inversely correlated with aspiration discrepancy in the hope of success group. It would seem that in a highly success motivated group the lower the aspiration discrepancy (reflecting a low aspiration level), the more negative is the thinking! Such a finding acts as a reminder that many different factors may contribute to negative thinking and to low mood and that the relationship between these factors can vary. For example, the idea that a high aspiration discrepancy can lead to negative thinking and depression may not always be true, indeed the reverse could be true depending on the success and failure orientations of the individual. It will be remembered that in the recovered depressives (i.e. the depression-prone subjects) aspiration discrepancy was found to be highly correlated with the level of depression (BDI) and also with general anxiety (GA).

The performance estimates of judgement discrepancy (JD), expectancy discrepancy (ED) and aspiration discrepancy (AD) revealed few significant correlations that merit discussion. However, judgement discrepancy (JD) was found to be inversely correlated with aspiration discrepancy (AD) in all three analyses, indicating that the smaller the difference between the number of answers the individual thought he answered correctly and the number he actually answered correctly on the problem solving task, the higher is the aspiration discrepancy.

A small negative correlation was also found between fear of failure (SFI) and expenctancy discrepancy indicating that the higher the fear of failure, the lower will be the perceived ability to be able to improve the score on the problem solving task.

In summary, four general points have emerged: (1) As found in the previous study (chapter six), the BSCT which was designed to measure depressive thought content had a high correlation with anxiety. However it would also appear that at mild levels of depression negative thinking does correlate with level of depression. (2) Unlike the other measures, the test anxiety scale was found to correlate with level of depression in both high fear of failure and high hope of success groups. (3) The attentional and perceptual parameters revealed (as found in chapter six) that a bias in one modality (TL-TR) correlated with a bias in a different modality (SEN.diff.). (4) A low aspiration discrepancy in the hope of success group was related to negative thinking.

II. BETWEEN GROUP ANALYSIS

The difference between the two groups was investigated by using a one-way analysis of variance. The results of this statistical operation are presented below. The high hope of success group is designated as H.S. and the high fear of failure group is designated F.F.

(1) General and background

(i) Beck depression inventory

The mean scores and F rates on the Beck depression inventory (BDI) are given in table 8.10.

Table 8.10 BDI scores

Group	Range	Mean	S.D.	
H.S.	0-3	0.7	1.16	$F = 13.56$
F.F.	0-19	6.0	5.9	$p < 0.001$
				$df = 32$

As predicted by hypothesis Ii, the F.F. group was significantly more depressed than the H.S. group. A mean of 6 is, of course, not indicative of depression. However five of the F.F. subjects scored over 10 on the BDI. It can be said therefore that, as a group, they were significantly different in their self reporting of mood.

(ii) General Anxiety

The mean scores and F ratio on the general anxiety scale (GAS) are shown in table 8.11.

Table 8.11 GAS scores

Group	Range	Mean	SD	
H.S.	0-5	2.83	1.72	F = 16.98
F.F.	2-15	7.25	4.17	$p < 0.001$
				df = 32

As predicted by hypothesis Iii (chapter 7), the F.F. group had a significantly higher level of general anxiety than the H.S. group. Again as a group the F.F. subjects rated themselves as being much more generally anxious than the H.S. group.

Tables 8.12, 8.13 and 8.14 show the means and F ratios for the background electrodermal activity measures.

(iii) Background electroderman activity

The mean number of spontaneous fluctuations per minute during completion of the test anxiety scale (TAS) and the F ratios (SFTAS) are shown in table 8.12.

Table 8.12 SFTAS scores

Group	Range	Mean	SD	
H.S.	1.3 - 9.5	4.88	2.19	F = 0.29
F.F.	0.8 - 9.9	4.39	3.09	N.S.

(iv) Background electrodermal activity

The mean number of spontaneous fluctuations per minute during completion of the problem solving task (SFPS) and the F ratios are shown

in table 8.13.

Table 8.13 SFPS scores

Group	Range	Mean	SD	
H.S.	1.0 - 9.5	3.56	2.22	F = 0.007
F.F.	0.8 - 8.5	3.51	2.27	N.S.

(v) Background electrodermal activity

The mean number of spontaneous fluctuations per minute during the light detection (vigilance) task (SFVI) and the F ratios are shown in table 8.14.

Table 8.14 SFVI scores

Group	Range	Mean	SD	
H.S.	0.1 - 4.7	1.63	1.17	F = 0.076
F.S.	0.01 - 3.8	1.52	1.12	N.S.

No hypothesis was formulated concerning differences in the background level of electrodermal activity over the three parts of the experiment for the two groups and the F ratios in the three tables above show that there were no differences between the groups on these measures, even though the groups were significantly different in their ratings of depression and anxiety.

(2) Attentional and perceptual

(i) Psychophysiological

The means and standard deviations over the nine measured epochs

Table 8.15 Psychophysiological attentional parameters

Variables		SECTION I			SECTION 2			SECTION 3		
		PREL	PRER	PREL-PRER	DUL	DUR	DUL-DUR	TL	TR	TL-T
Groups										
H.S.	Mean	6.4	4.5	1.94	3.78	3.94	-0.16	10.22	8.44	1.77
	SD	3.57	2.8	3.17	3.10	2.18	2.18	5.30	3.85	4.04
F.F.	Mean	6.69	5.13	1.57	2.5	4.0	-1.5	8.63	9.0	-0.37
	SD	3.65	4.49	3.56	1.9	2.66	1.55	4.86	6.37	3.16
Comparison										
H.S. v F.F.	F ratio						4.14			
	p	NS	NS	NS	NS	NS	0.05	NS	NS	NS

analysed during the problem solving task are shown in table 8.15. The F ratio and level of significance are given where relevant.

The nine elements of psychophysiological data used as an index of attention to positive (success) and negative (failure) events show that there was no significant difference between the groups on the anticipatory electrodermal epochs (i.e. PREL, PRER and PREL-PRER). Nor was there any significant difference found in electrodermal activity measured during the presentation of the left (success) light (DUL) and electrodermal activity measured during the presentation of the right (failure) light (DUR). However, the difference between these measures DUL-DUR was found to be significantly different between the groups. It was found that compared to the H.S. group, the F.F. group has a significantly larger negative bias in their electrodermal responding to signalled success and failure. An examination of the total measured electrodermal epochs (TL, TR and TL-TR) revealed no significant differences between the groups.

Thus hypothesis 2(i) (chapter 7) is partially confirmed on the parameter of bias in electrodermal activity to positive and negative events in that DUL-DUR in the F.F. group was found to be significantly more negative than in the H.S. group. However, the other electrodermal measures did not produce any significant results.

Tables 8.16, 8.17 and 8.18 are concerned with the perceptual sensitivity measures to the left (success signalling) and right (failure signalling) lights.

(ii) Perceptual sensitivity to the left light

The means for the perceptual sensitivity scores to the left light (SENL) are shown in table 8.16.

Table 8.16 SENL scores

Groups	Range	Mean	SD	
H.S.	17.05 - 22.05	18.61	1.36	F = 0.308
F.F.	16.4 - 22.8	18.79	1.79	N.S.

(iii) Perceptual sensitivity to the right light

The means for the perceptual sensitivity scores to the right light (SENL) are shown in table 8.17.

Table 8.17 SENR scores

Groups	Range	Mean	SD	
H.S.	17.50 - 22.10	19.23	1.32	F = 0.385
F.F.	16.00 - 22.9	19.00	1.6	N.S.

(iv) Difference between sensitivity thresholds

Table 8.18 shows the means and range scores for the difference between the measures of perceptual sensitivity to the left (success) light and right (failure) light (SENR - SENL, i.e. SEN diff.).

Table 8.18 SEN diff. scores

Group	Range	Mean	S.D.	
H.S.	(-0.4) - (+2.65)	0.62	0.7	F = 2.76
F.F.	(-2.5) - (+1.50)	0.21	0.49	N.S.

Although tables 8.16 and 8.17 show that the F.F. group had the higher perceptual sensitivity to the left (success) light (SENL) and the lower perceptual sensitivity to the right (failure) light (SENR), the differences between the groups were not significant. Moreover an examination of the bias scores (SENR-SENL) in table 8.18 revealed that the F.F. group had a lower positive bias than the H.S. group. It will be recalled from chapter six that the results on this measure showed that normals tend to have a positive bias and recovered depressives and anxious patients also have a positive bias in perceptual sensitivity, but to a much less marked degree. A similar phenomenon seems to have occurred here with the F.F. group having the lesser positive bias in their perceptual sensitivity scores. However, although this difference did not reach statistical level of significance, it did approach significance. Thus the findings are in the expected direction as predicted by hypothesis 2(i) (chapter 7).

(3) Attitudinal and cognitive

(i) Internal-external scale

The means and F ratio on the internal-external scale (I-E) are shown in table 8.19.

Table 8.19 I-E scores

Group	Range	Mean	SD	
H.S.	1-15	7.61	4.2	F = 6.78
F.F.	4-17	11.56	4.6	$p < 0.05$
				df = 32

It can be seen that, as predicted by hypothesis 3 (i) (chapter 7), the F.F. group had a significantly higher externality score than the H.S. group.

(ii) Test Anxiety Scale

The means and F ratio on the test anxiety scale (TAS) are shown in table 8.20.

Table 8.20 Test Anxiety Scores

Group	Range	Mean	S.D.	
H.S.	4-21	11.89	6.4	F = 12.8
F.F.	10-33	18.65	6.4	$p < 0.01$
				df = 32

As predicted in hypothesis 3(ii) (chapter 7), the F.F. group had a significantly higher level of test anxiety than the H.S. group. In view of the large significant difference between these two groups on the success/failure inventory, this difference in the TAS might have been expected to be even larger as both measures purport to measure the same thing, i.e. fear of failure.

(iii) Beck story completion test

The means and F ratio on negative thinking (BSCT) are given in table 8.21.

Table 8.21 BSCT scores

Group	Range	Mean	S.D.	
H.S.	20-40	29.67	5.9	F = 18.52
F.F.	29-79	45.37	14.15	$p < 0.001$
				df = 32

As predicted by hypothesis 3(iii) (chapter 7), the F.F. group had a significantly higher negative thinking (BSCT) score than the H.S. group.

Tables 8.22, 8.23 and 8.24 are concerned with the performance estimates, judgement discrepancy, expectancy discrepancy and aspiration discrepancy.

(iv) Judgement discrepancy

Table 8.22 shows the means and F ratio for the judgement discrepancy (JD) scores.

Table 8.22 JD scores

Group	Range	Mean	SD	
H.S.	(-4) - (+2)	-0.28	1.53	F = 0.51
F.F.	(-5) - (+5)	-0.44	2.53	N.S.

(v) Expectancy discrepancy

Table 8.22 shows the means and F ratio for the expectancy discrepancy (ED) scores.

Table 8.22 ED scores

Group	Range	Mean	SD	
HS	(0) - (2)	0.44	0.78	F = 3.74
FF	(0) - (1)	0.06	0.25	NS

(vi) Aspiration discrepancy

The means and F ratio for aspiration discrepancy (AD) are shown in table 8.23.

Table 8.23 AD scores

Group	Range	Mean	SD	
HS	(0) - (+10)	4.33	3.23	F = 0.237
FF	(0) - (+6)	3.81	2.97	NS

These performance estimate scores did not show any statistical difference between the groups, although the difference between the groups on expectancy discrepancy only just failed to reach significance.

III. SUMMARY OF RESULTS AND SHORT DISCUSSION

Table 8.24 provides a summary of the results of the between group analyses (Hope of Success versus Fear of Failure).

As in chapter six, the group depicted on the left side in column 2 is the group with the higher score or value on the corresponding parameter in column 1 and the >

sign is used to show this.

The sign — indicates that no significant difference was found for that comparison.

A short discussion of the results follows table 8.24.

Groups compared Variables	Hope of success (H.S.) versus Fear of failure (F.F.)
General and background	
BDI	F.F. > H.S.
GA	F.F. > H.S.
SFTAS	-
SFPS	-
SFVI	-
Attentional and perceptual	
PREL	-
PRER	-
PREL-PRER	-
DUL	-
DUR	-
DUL-DUR	F.F. > H.S. (more negative)
TL	-
TR	-
TL-TR	-
SENL	-
SENR	-

Groups compared Variables	Hope of success (H.S.) versus Fear of failure (F.F.)
SENR-SENL	-
Attitudinal and cognitive	
I-E	F.F. > H.S.
TAS	F.F. > H.S.
BSCT	F.F. > H.S.
JD	-
ED	-
AD	-

Short discussion

Comparisons made between a relatively high success orientated group (H.S.) and a relatively high fear of failure group (F.F.) revealed that on a self-report depression scale (BDI) the F.F. group scored significantly higher than the H.S. group. On a self report anxiety scale (TAS) the F.F. group was also found to score significantly higher than the H.S. group. It is questionable how far these differences reflect a genuine affective difference between the groups or are more a reflection of differential reporting. On the measures of background electrodermal activity during the completion of the test anxiety scale, problem solving and vigilance task, no significant differences were found between the groups.

The attentional and perceptual parameters showed that anticipatory electrodermal activity to both the success (left) light and failure (right) light were not

significantly different between the groups. Moreover, there was no significant difference between the groups on the measure of bias of anticipatory responding. The electrodermal activity measured during the actual presentation of the left (signalling success) light and also during the presentation of the right (signalling failure) light also showed no significant difference between the groups. However, the bias in this electrodermal responding, that is the difference between these two measures, was found to distinguish the groups. The F. F. group was found to have a significantly higher negative bias than the H.S. group to signalled success and failure events. Thus the F. F. group showed a significantly greater tendency to respond (as measured electrodermally) more to a negative event (failure) than to a positive event (success) as compared to the H.S. group. This finding offers some support to the theory on fear of failure motivation (Birney et al., 1969). However, it will be recalled from the results in chapter 6 that this particular electrodermal measure did not differentiate any of the groups. In that study both the depressed and recovered depressed were found to be not significantly different from the anxious and normal control groups.

The total measured electrodermal activity over the anticipatory epochs and during the signal (left and right light) presentation epochs revealed no significant difference between the groups.

The perceptual sensitivity data did not show any significant differences either. However, all the results were in the predicted direction. The differences between the perceptual sensitivity scores showed that the H.S. group had the largest positive bias score and the F. F. group the lowest, this result approached significance. Moreover it might be noted that on this measure the F. F. group appeared to respond in a similar manner to the recovered depressed group.

The attitudinal and cognitive measures indicated that the F.F. group was significantly more external than the H.S. group. The F.F. group was also significantly more test anxious than the H.S. group. However, although this later result was significant at the 1% level, it might have been expected to have been significant at an even higher level of significance in view of the fact that these subjects were chosen for being very different in their relative fear of failure orientation, and that test anxiety has been used as a measure of the strength of fear of failure.

The F.F. group showed a significantly higher level of negative thinking than the H.S. group, but the performance estimation scores did not reveal any significant difference between the groups. However a comment should be made on the observation that in the depressed patients when ill and after recovery, there was a tendency for aspiration discrepancy to be higher than in the normal control group, whereas in the F.F. group the tendency was for aspiration discrepancy to be lower than in the H.S. group. Although not significant it may be of value to examine this issue in more detail with more sensitive tests, since it was suggested in chapter 4 that a predisposing factor in depression might be a high fear of failure allied with a high aspiration level.

CHAPTER NINE

EXPERIMENT 3

In the previous two studies an attempt was made to investigate certain attentional and attitudinal factors considered to be causally related to depression (Beck, 1967, 1976). This work arose from Beck's view that depression results from the emergence and dominance of certain negative, idiosyncratic patterns of thinking; the basic hypothesis being that negative and distorted cognitions produce congruent distorted affective states. It follows from this position that the major factors influencing the predisposition to depression reside in the cognitive, evaluative style of the individual, in contrast to (say) life events (e.g. Brown and Harris, 1978) or a possible biological vulnerability (e.g. Akiskal and McKinney, 1975).

Beck's theory of depression has generated much research which demonstrates the presence of various cognitive distortions in depressed individuals. It remains the case, however, that few studies have used adequate psychiatric controls. Moreover, some of the central cognitive factors in Beck's theory of depression are observable in other psychiatric groups. For example Becker (1974) points out that low self-esteem (negative view of the self) is common to many different psychiatric populations. In addition a negative view of the future (hopelessness) has been shown to have a high correlation with suicidal intent, regardless of diagnosis (Minkoff et al., 1973).

The results of study one revealed that, in comparison with an anxious control group, depressed patients were not significantly different on the measures of locus of control (I - E), fear of failure (TAS) and negative thinking (BSCT), and both groups had significantly higher scores than a normal control group. The absence of differentiating findings between the depressed and anxious groups may justify Blaney's (1975) claim that Beck's model of depression is, in its present form, too flexible to offer a true theoretical model of depression, as its flexibility makes it difficult to refute.

Thus it remains important to examine the role of some of the cognitive factors outlined by Beck for a specific theory of depression. Since Beck argues that individuals are prone to depression because of idiosyncratic evaluative styles, it should be possible to provide evidence of this by comparing depression prone individuals with individuals who have not suffered a specific depressive disorder. On the question of predisposition to depression, it is interesting to note that Brown and Harris (1978) consider Beck's stipulation of inappropriate negative conceptualisations as unnecessary for a theory of depression. They argue that depression may also come from an entirely accurate conceptualisation, 'the 'fault' lying in the environment rather than the person' (p. 83). They also challenge the idea that there is something 'cognitively wrong' with the individual in terms of his attitudes and

schemas before a depression occurs. In their view, such a susceptibility accounts for only a small percentage of depressed individuals.

I. AIMS

The aims of this study are therefore to investigate certain attitudinal and cognitive factors in the light of the above consideration. An attempt is made to examine differences between a group of depression prone individuals (recovered depressed patients) and a group of improved anxious patients without a history of primary depressive disorder.

This study therefore attempts to examine the question of whether depression prone individuals tend to have more negative cognitive attitudes on certain salient measures when compared to a group of improved anxious patients who have not experienced or been treated for a depressive illness.

II. DESIGN

A cross sectional design was adopted for this study. This required that potential subjects be identified from case registers, following which patients would be selected or rejected according to specific diagnostic criteria. In addition to consultant advice, this procedure relied on the confirmation of a diagnosis from patient notes according to criteria described by Feighner et al. (1972).

All subjects selected and agreeing to take part in this study completed a series of self report questionnaires aimed at examining certain cognitive parameters.

III. METHOD

A. SELECTION OF PATIENTS

1. Recovered Depressed Group (R. Dep.)

In the first instance three Consultant Psychiatrists (Dr. R. Devine, Dr. B. Cornes and Dr. C. Roberts) were contacted and the aims of the study explained. These consultants offered names of patients who they felt might be suitable for inclusion in the study. This produced a large number of potential patients who had been treated with a recognised anti-depressant or ECT and/or were being maintained on lithium carbonate. All were regarded as having had, and recovered from, a major depressive illness. The second phase of selection required that patient case notes be examined and only those patients meeting the criteria of Primary Affective Disorder as described by Feighner et al. (1972) were further considered. In addition to the exclusion criteria described by Feighner et al. (op. cit.), Section C, patients over the age of 65 or with a history of mania were also rejected. These various exclusion criteria were responsible for a drastic reduction in the original potential sample.

The third phase of selection required that patients should have been recovered for three months or longer and that their level of self-reported depression as measured by the Beck Depression Inventory be 10 or below. The Beck Depression Inventory

correlates well with objective measures of depression in recovered patients (Carroll et al., 1973; Paykel et al., 1973).

Finally, patients were contacted by the author and asked to attend a one hour session at the Department of Clinical Psychology in Norwich. If this was not possible, the author made an effort to see them following follow-up appointments with the psychiatrist.

Although each criterion resulted in the loss of a number of potential subjects, it was hoped that the final group would reliably consist of patients who had recovered from a true unipolar depressive illness. The original patient sample taken from lists of patients on lithium and consultant recommendations consisted of over 80 cases.

In addition to the wastage due to the first three phases of selection, a number of potential patients refused to co-operate in the project. The final number of patients selected was 15.

2. Improved Anxious Patients

In the first phase of selection four Clinical Psychologists (Mr. D. Castell, Dr. H. Kuna, Mrs. J. Rhodes and Mrs. K. Lawrence) were informed of the aims of the study. These psychologists kindly agreed to examine their records over the past three years and to draw up a list of potential subjects for this group. This provided approximately 55 cases who had been seen by a psychologist primarily for an anxiety problem and had

been treated with a recognised anti-anxiety psychological procedure, occasionally in conjunction with the appropriate drugs. From the lists obtained, case notes were consulted and an effort was made to apply Feighner's et al. (1972) criteria for Anxiety Neurosis and Phobic Neurosis. Only those patients offering a reasonably straight forward picture of either an anxiety neurosis or phobic neurosis were considered further. In addition to Feighner's exclusion criteria, special attention was given to the presence of depressive symptoms and patients who showed any significant depressive symptoms were rejected. In addition, patients who scored above ten on the Beck Depression Inventory when tested were also rejected.

It was considered more appropriate to label these patients as improved anxious patients because, while some patients appear to have completely recovered, other patients complained of minor episodic difficulties. However, no patient was significantly impaired at the time of testing and all were considered to have improved, though some were being seen by a psychologist as follow-up out-patients. From the original sample, 15 patients who met the above criteria were obtained. In the final selection of these patients, an effort was made to match their B.D.I. scores with those in the recovered depressive group.

3. Comparison of the two samples for age and sex

Age

The mean age scores and group statistics of the two groups are given in Tables 9.1 and 9.2. The difference between mean ages was analysed using a two tailed 't' test. Further discussion of this technique is given at the end of the chapter.

Table 9.1 Age Score

Group	Range	Mean	SD
R. Dep.	31 - 64	47.9	10.18
Im. Anx.	24 - 57	35.6	9.22

Table 9.2 Differences in ages

comparison	difference between means	SE diff.	t	p	df
R. Dep. vs Im. Anx.	12.3	3.55	3.46	0.01	28

The results of the 't' test reveal that the Improved Anxious subjects are significantly younger than the recovered depressed subjects. This result is similar to that found in study one. It should also be noted that the mean age of the recovered depressed patients in study one was 46.4 compared to the mean age of recovered depressed patients in this study of 47.9. A 't' test revealed this small difference to be well below significance. Similarly, the mean age of the improved anxious patients in this study (35.6) was very similar to the mean age of the Anxious

subjects of study one (33.7). Again 't' analysis revealed the difference to be far from significant.

It therefore seems, with respect to age, that this study replicates the findings of study one.

Sex

Although sex was not a purposefully manipulated variable the male/female ratio turned out to be identical in each group, the ratio being 6 : 9.

B. MEASURES

Since this study is designed in part to be a replication of study one, many of the measures used in this study are the same as those used in study one. However, one scale has been changed and one further scale added.

The following scales were administered:

1) The Internal-External Scale (I-E) (see pages 148 - 152)
2) The Beck Story Completion Test (BSCT) (see pages 157 - 159)
3) The Success-Failure Inventory (SFI) (see pages 257 - 258).

The Success-Failure Inventory was used in preference to the Test Anxiety Scale as a measure of

fear of failure. This was because the Success-Failure Inventory was designed to be used with normal and psychiatric adult populations (McReynolds and Guevara, 1967), whereas the Test Anxiety Scale, though a more often used measure, is biased towards a student population.

4) The Hopelessness Scale (HS)

This scale was added to the existing measures because of the considerable attention the concept of hopelessness has received in recent years. Moreover, this concept plays a central role in the cognitive theory of depression (Beck, 1967, 1976). However, the exact mechanism by which hopelessness and depression are related remains unclear. Melges and Bowlby (1969) point out that hopelessness has relevance to other psychiatric disturbances apart from depression. Moreover, Minkoff et al. (1973) have shown hopelessness to be related to suicidal intent regardless of diagnosis and more recently Wetzel (1976), using this hopelessness scale with a group of depressed patients, has confirmed the positive correlation between hopelessness and seriousness of suicidal intent.

The hopelessness scale (HS) used here was devised by Beck et al. (1974). Although Beck's theory of

depression postulates that hopelessness plays a major role in depression, he is also aware of its relevance to other pathological states. Indeed the authors explain that the HS was devised -

"In order to facilitate the study of hopelessness in various psychopathological groups" (Beck et al, op cit, p. 861).

In view of the attention the concept of hopelessness has received in relation to depression, it seems important to know whether recovered depressed patients retain a residual degree of hopelessness which may continue as a vulnerability factor, and indeed whether they have more negative expectancies on recovery compared to a non-depressed psychiatric control group.

The hopelessness scale utilises 20 true-false items selected to reflect "different facets of the spectrum of negative attitudes about the future and which recurred frequently in patient's verbalisations" (Beck et al., op. cit.).

The scale consists of 11 items scored for a 'true' response and 9 items scored for a 'false' response. An example of an item scored for a true response is:

Item 2 I might as well give up because I can't make things better for myself true/false

An example of an item scored for a false response is

Item 1 I look forward to the future with hope and enthusiasm true/false

From a selected population of 294 hospitalised patients Beck et al. (op. cit.) reported an internal reliability coefficient of 0.90. Using a group of 23 out-patients in general medical practice and a group of 63 hospitalised patients, Beck et al. (op. cit.) found a correlation between clinical ratings of hopelessness and HS scores of 0.74 for the first group and 0.62 for the second group (inter-rater reliability of the two judges was 0.86).

5) Beck Depression Inventory (B.D.I.) (see pages 159 - 161)

This scale was used both as a measure of self-reported depression and as a screening device. Patients with scores of 10 or above were excluded from the study (see pages 159 - 161).

6) General Anxiety (G.A.S.)

The level of general anxiety was measured using the General Anxiety Scale (see pages 161 - 162).

C) TESTING PROCEDURE

Subjects identified as suitable for participation in this study were contacted in one of three ways: (a) by direct referral from a Consultant or Psychologist, (b) by the author's attendance at out-patient appointments, and (c) by letter. For those who could not be seen following an out-patient appointment, a subsequent appointment lasting approximately one hour was arranged at either the Psychology Department or the Bethel (child and family psychiatry) Hospital in Norwich.

In some cases patients agreed to participate in the study only if they could fill the forms in and return them by post. Although not an ideal method of data collection, other recent studies using depressed patients have relied on mailed data (e.g. Parker, 1979). Patients in this situation also received a covering letter thanking them for their help and highlighting the importance of giving only their own personal answers.

D) CONDITIONS OF TESTING

Patients who attended the testing session as arranged were given a room to themselves and presented with the necessary questionnaires. At the end of the session a cup of tea was provided and the subject allowed to ask questions. Only the general principles of the research were explained. It was considered by all those concerned with the

project that patients should receive some information in return for their help.

E) ANALYSIS OF THE DATA

The statistical analysis of the data was concerned with the difference between two means and used the simple 't' test. Alternative methods of analysis were considered but it was decided that an analysis of covariance, using BDI score as covariate, was not warranted in view of the low levels of self-reported depression and the fact that the two groups had closely similar scores.

The formula used was

$$t = \frac{\text{difference between means}}{\text{standard error of the difference between means (SE diff)}} \qquad \text{(two-tailed)}$$

$$\text{SE diff} = \sqrt{\frac{\sigma_1^2}{n_1} + \frac{\sigma_2^2}{n_2}} \qquad \text{(Kerlinger, 1973)}$$

The results are presented in two sections:

1. Difference between recovered depressed patients in study one and study three.

   In view of the consideration that the recovered depressed patients of study one may not have been completely recovered, it is of importance to be aware of any differences between this group and the group chosen for study three. The analysis uses the formulae shown above.

2. Difference between recovered depressed patients (study 3) and improved anxious patients

This comparison constituted the main purpose of the study with the aims discussed at the beginning of the chapter. The analysis used the formula shown above.

F. HYPOTHESES

The main hypotheses of the study are derived from the analysis and review of the literature. This review leads to the following questions and hypotheses.

(a) recovered depressed patients will have lower I-E scores than improved anxious patients, that is they will be more internal.

(b) recovered depressed patients will have higher negative thinking scores (on a scale designed to measure negative attitudes relevant to depression) than improved anxious patients.

(c) recovered depressed patients will have higher fear of failure scores than improved anxious patients.

(d) recovered depressed patients will have higher hopelessness scores than improved anxious patients.

CHAPTER TEN

RESULTS

The results of this work are presented in two parts:

1. Comparison of differences between the recovered depressed group of study one with the recovered depressed patients of study three.
2. Comparison of differences between the recovered depressed group of study three with the improved anxious group of study three.

1. RECOVERED DEPRESSED STUDY ONE (R Dep. s l) VERSUS RECOVERED DEPRESSED STUDY THREE (R Dep s 3)

i) Beck Depression Inventory (BDI)

The mean and standard deviation scores are shown in table 10.1.

Table 10.1 BDI Scores

Groups (n = 15)	Range	Mean	SD
R Dep s l	0 - 35	11.53	11.88
R Dep s 3	0 - 9	3.87	3.27

The results of the 't' test are given in table 10.2.

Table 10.2 Difference between BDI scores

Comparisons	diff between means	t	p	df
R Dep s 1 v R Dep s 3	7.66	2.41	0.05	28

This result demonstrates that the recovered depressed subjects used in study three reported significantly less depression (as measured by the BDI) than those of study one. It should also be noted that Metcalfe and Goldman (1965) quote a mean of 5.4 and standard deviation of 5.8 at the non-depressed level for a British sample using this scale (see page 160). Consequently it seems reasonable to argue that none of the subjects in study three were depressed as measured by this scale.

ii) General Anxiety (G.A.S.)

The mean and standard deviation scores are shown in 10.3.

Table 10.3 G.A.S.

Groups (n = 15)	Range	Mean	SD
R Dep s 1	1 - 13	7.00	4.12
R Dep s 3	1 - 12	6.133	2.92

The results of the 't' test are given in table 10.4.

Table 10.4 Differences between GAS scores

Comparison	diff between means	t	p	df
R Dep s1 v R Dep s3	0.867	0.665	NS	28

This result shows that there is no difference between the level of anxiety, as measured by this scale, in the recovered depressed subjects in study three compared with those of study one.

iii) Internal - External Scale (I - E)

The mean and standard deviation scores are shown in table 10.5.

Table 10.5 I - E scores

Groups (n = 15)	Range	Mean	SD
R Dep s1	4 - 17	10.46	3.7
R Dep s3	2 - 14	7.53	3.23

The results of the 't' test are given in table 10.6.

Table 10.6 Differences between I - E scores

Comparison	diff between means	t	p	df
R Dep s1 v R Dep s3	2.93	2.31	0.05	28

The results of this comparison show that the recovered depressed patients of study three were significantly more internal than those of study one. In view of this finding it was considered useful to compare the I - E scores of the recovered depressed patients in this study (three) (mean 7.53) with those of the normal control subjects used in study one (mean 8.6). This comparison indicated that this recovered depressed group had marginally lower scores (indicating a higher degree of internality) compared with the normal group of study one, but the difference did not approach significance.

iv) Negative thinking (BSCT)

The mean and standard deviation scores are shown in table 10.7.

Table 10.7 BSCT scores

Groups (n = 15)	Range	Mean	SD
R Dep s1	22 - 67	40.2	8.6
R Dep s3	30 - 52	39.67	6.45

The results of the 't' test are given in table 10.8

Table 10.8 Difference between BSCT scores

Comparison	diff. between means	t	p	df
R Dep s1 v R Dep s3	0.53	0.19	NS	28

The result of this comparison show that on the measure of negative thinking the two groups were not significantly different.

II. RECOVERED DEPRESSED (R Dep) VERSUS IMPROVED ANXIOUS (Imp Anx)

(both groups of the third study)

i) Beck Depression Inventory (BDI)

The mean and standard deviation scores are given in table 10.9.

Table 10.9 BDI scores

Groups (n = 15)	Range	Mean	SD
R Dep	0 - 9	3.87	3.27
Imp Anx	0 - 8	3.53	2.95

The results of the 't' test are given in table 10.10.

Table 10.10 Differences between BDI scores

Comparison	diff between means	t	p	df
R Dep v Imp Anx	0.34	0.294	NS	28

The result shows that the groups were very similar in their BDI scores (similar means, range and standard deviation) and in view of normative data reported by Metcalfe and Goldman (1965) neither group was depressed, as measured by the BDI, when tested.

ii) General Anxiety (G.A.S.)

The mean and standard deviation scores are given in table 10.11.

Table 10.11 G.A.S. score

Group (n = 15)	Range	Mean	SD
R Dep	1 - 12	6.13	2.92
Imp Anx	3 - 14	9.4	2.92

The results of the 't' test are given in table 10.12

Table 10.12 Difference between G.A.S. scores

Comparison	diff between means	t	p	df
R Dep v Imp Anx	3.27	3.06	0.01	28

The result shows that the improved anxious group were significantly more anxious as measured by this scale than the recovered depressed group. This is a useful result since previous analysis (tables 10.9 and 10.10) have shown that neither group was significantly depressed, though as shown here differences do exist in the level of self-reported anxiety as measured by this scale.

iii) Internal - External Scale

The mean and standard deviation scores are given in table 10.13.

Table 10.13 I - E Scores

Group (n = 15)	Range	Mean	SD
R Dep	2 - 14	7.53	3.23
Imp Anx	5 - 18	12.67	6.37

The results of the 't' test are given in table 10.14.

Table 10.14 Differences between I - E scores

Comparisons	diff between means	t	p	df
R Dep v Imp Anx	5.14	3.05	0.01	28

The results of this comparison show that the recovered depressed group were significantly more internal than the improved anxious group, thus supporting hypothesis F(a).

iv) Negative thinking (BSCT)

The mean and standard deviation scores are given in table 10.15.

Table 10.15 BSCT scores

Group (n = 15)	Range	mean	SD
R Dep	30 - 52	39.67	6.45
Imp Anx	28 - 72	40.0	10.33

The results of the 't' test are given in table 10.16

Table 10.16 Differences in BSCT scores

Comparison	diff between means	t	p	df
R Dep v Imp Anx	0.33	0.16	NS	28

The result of this comparison shows that these two groups are not significantly different on this measure of negative thinking. Hypothesis F (b) is thus not supported.

v) Success - Failure Inventory (SFI)

The mean and standard deviation scores are given in table 10.17.

Table 10.17 SFI Scores

Group (n = 15)	Range	Mean	SD
R Dep	(-7) - (+12)	5.0	5.58
Imp Anx	(-9) - (+11)	3.33	6.39

The results of the 't' test are given in table 10.18.

Table 10.18 Differences between SFI scores

Comparison	diff between means	t	p	df
R Dep v Imp Anx	2.67	0.763	NS	28

The result demonstrates that although the improved anxious group had a slightly higher fear of failure score (as indicated by a lower hope of success score) this difference did not reach significance. Hypothesis F(c) was, therefore, not supported.

vi) Hopelessness (HS)

The mean and standard deviation scores are given in table 10.19.

Table 10.19 HS scores

Group (n = 15)	Range	Mean	SD
R Dep	1 - 6	3.73	1.33
Improved	1 - 9	3.73	2.76

The results of the 't' test are given in table 10.20.

Table 10.20 Difference between HS scores

Comparison	diff between means	t	p	df
R Dep v Imp Anx	0.0	0.0	NS	28

This result shows that on this measure of hopelessness the two groups were not significantly different - again disconfirming hypothesis F (d).

III. SUMMARY OF RESULTS

The results presented in Chapter ten are summarised in tables 10.21 and 10.22.

1. COMPARISON - R Dep s1 v R Dep s3

Comparing the recovered depressed group of study 1 (R Dep s1) with the recovered depressed group of study 3 (R Dep s 3), the results shown in table 10.21 below were obtained.

Table 10.21 R Dep s1 v R Dep s3

BDI	GAS	I - E	BSCT
R Dep s1 > R Dep s3 (p 0.05)	NS	R Dep s1 > R Dep s3 (p 0.05)	NS

2. COMPARISON OF R DEP v IMP ANX (STUDY THREE)

The summary of the results from the two groups in study 3 are given in Table 10.22 below.

Table 10.22 R. Dep. v Imp Anx

BDI	GAS	I - E	BSCT	SFI	HS
NS	Imp Anx > R Dep ($p < 0.01$)	Imp Anx > R Dep ($p < 0.01$)	NS	NS	NS

Brief discussion

The comparisons between the recovered depressed group of the first study and the one obtained for this study are important. The relatively high residual depression reported by the first study's recovered depressed group did cast doubt on the degree of recovery, in spite of consultant judgement. However, for this study very stringent selection criteria were applied to the choice of subjects, which though resulting in a high degree of wastage did ensure that only truly recovered depressed patients were selected. In addition, bipolar depressives were excluded - which was not the case for the first study. The results show that these patients were significantly less depressed than those in study one. Moreover, they had significantly lower externality scores. Such a finding may indicate that a patient's view of controllability may change over the course of his illness. This finding would support both Seligman's (1975) original formulation of depression and its recent reformulation in terms of attribution theory (Abramson et al., 1978). Strictly speaking Beck's theory is not supported by this finding

since Beck's position would predict that patients with higher depression scores should be more internal and move towards an external position as the depression recedes. This is because Beck has stressed the relationship between the increase in the perception of personal responsibility (control) and depression. In fact, however, as discussed earlier, an attribution of low ability to control could still result in high external scores when depressed and thus make this result compatible with Beck's theory.

Although work with recovered patients is a neglected area of research by cognitive theorists, it remains one of importance. If it is not possible to demonstrate 'cognitive' vulnerability factors in depression prone individuals, then the predictive validity of the theory is weakened. Unfortunately, the differences found in the level of self-reported depression between the two recovered groups does point to a potential source of confusion in this type of research. This confusion could arise from the fact that for the investigation of recovered depressives it is necessary to ensure that (i) patients selected for study did suffer from a genuine depressive disorder and (ii) that they have completely recovered. Taken together these two criteria may bias the type of patient investigated. In the first place, using Feighner's et al. (1972) criteria, patients who had been clearly depressed but who also had a history of obsessional or anxiety neurosis and presented an unclear diagnosis were excluded. In the second place, it may be that those patients who make the best recoveries with physical treatments represent a group of patients sometimes referred to as endogenous, and there remains a very real question mark over whether the cognitive theory of depression does, or should, attempt to provide aetiological explanations of all patients suffering from depression. For

example, cognitive theory has made no attempt to explain what are often dramatic diurnal changes of mood in some depressed patients. It is not uncommon for some severely depressed patients to wake up each morning agitated, full of guilt and feelings of unworthiness, refusing help, food or to talk and yet by evening these symptoms have retreated with an apparent mysterious reduction in negative attitudes and conclusions. In short, some severely depressed patients often seem to unlearn their rather negative conclusions during the course of every day only to wake prematurely the next morning to discover they have returned with force while they slept.

In addition, cognitive theory alone does not adequately explain those depressions which can be substantially shortened and relieved by either ECT or anti-depressant drugs in the absence of any direct or systematic confrontation with the patient's attitudes and conclusions. These remain important issues which will be further discussed in the following chapter. Suffice it to say here that there may exist a conflict arising from the clear necessity to apply objective criteria to patient selection which may result in a sampling bias operating against the paradigm under investigation.

The failure to find differences in negative thinking between the two recovered depressed groups with different levels of self-reported depression is difficult to explain. This is especially so since the scale was originally shown to be a sensitive measure with normal subjects (Weintraub et al., 1974). At the outset of this research in 1974/75, the scale was obtained from Beck and there was reason to believe that it would prove a useful instrument. Unfortunately no subsequent work with this scale has been reported in the literature. Logically

the design of the scale is appropriate as an investigatory instrument of Beck's theory and it is disappointing that the correlation results of study one and those of this study have provided results the interpretation of which is unclear.

The results of this third study comparing a recovered depressed group and an improved anxious group indicate that neither group was significantly depressed when tested. However, the improved anxious group was significantly more anxious than the recovered depressed group. Consequently these two groups can be considered to generate useful data within the aims of the project.

Observation of the results show that on only one out of four possible cognitive and attitudinal measures was a significant difference found between the two groups, this being on the I - E scale. Thus in the absence of reported depressive symptoms it would seem that recovered depressed patients may hold more internal views compared to improved anxious patients. However, on measures of negative thinking, fear of failure and hopelessness, the groups could not be statistically discriminated. At the present time fear of failure has regained the interest of some researchers (Shaver et al., 1978; Shaver, 1979). Moreover, in view of Shaver's demonstration of correlations between a measure similar to the concept of fear of failure and various 'neurotic' symptoms, this concept continues to hold promise. Unfortunately, as Shaver et al. (1978) point out, confusions over measurement abound. Moreover, as chapter four made clear, an individual may 'fear failure' because of an expectation of aversive consequences (punishment) or an expectation of the withdrawal of positive reinforcement. These two sets of expectations need not represent identical attitudes. But it may require an instrument specially designed with this

view in mind to discriminate between them. The success-failure inventory probably does not do this. As yet there is no measure available which does serve this function.

The hopelessness scale did not discriminate the two groups under investigation. Thus there is perhaps some question over whether residual hopelessness can act as a vulnerability factor in recovered depressed patients. Such a result does require considerable caution in interpretation because of the low ratings of hopelessness from both groups. The problem remains, however, that if hopelessness and negative thinking are not cognitive styles which depression-prone individuals exhibit when well, then these variables lose their predictive validity. This may be qualified by the argument Beck (1967) puts forward when he suggests that the negative cognitive style of depression-prone individuals may not be discernable when they are well. If Beck is indeed correct on this point then the aetiological dimension of Beck's theory becomes extremely difficult to test since there is no way in advance to predict who is prone to depression and who is not except perhaps for some individuals whose depression has not yet developed but is imminent. It is because of these difficulties that work with recovered depressed patients is essential for an aetiological theory such as the cognitive theory of depression. It is something of a moot point perhaps but if it were possible to identify individuals who do have a hopeless outlook with low self-esteem and a negative view of the future, would this produce a group of individuals who, either were, or are about to become, pathologically depressed? It is an unresolved empirical question.

CHAPTER ELEVEN

DISCUSSION

I. EXPERIMENT ONE

The first study of this thesis set out to examine certain of the hypotheses derived from a cognitive-behavioural approach to depression. It was predicted, on the basis of this approach, that depressed subjects, as compared to a psychiatric control group and a non-psychiatric control group, would show distortions in their attention to and perception of stimuli that signalled positive (success) and negative (failure) events. It was predicted that depressed subjects, compared to the two control groups, would show distortions in their attention and perception in a more negative direction. It was further predicted that on various attitudinal and cognitive measures, such as negative thinking, test anxiety, perceived control over reinforcers, and ability to estimate accurately actual performance on a problem solving task, depressed subjects would show a more negative orientation, than the two control groups. In addition it was predicted that depressed patients would show a smaller expectancy to be able to improve in the problem solving task but would have a higher aspiration level as reflected in a calculated aspiration discrepancy score.

As outlined in the aims of this study (p.138) the work attempted to shed light on four questions which appeared relevant from the review of the literature. It is intended to examine each of these questions in turn in the light of the results of this study. Before proceeding, however, some discussion of the groups themselves is required.

All the patients who took part in this study as depressed patients were diagnosed as suffering from a primary depressive disorder and were clinically depressed at the time of testing. This criterion was met by a diagnosis given by a senior member of the MRC clinical staff. A problem which is common to other psychological studies is that no objective alternative assessment appeared as part of this study. The lack of a clinical training by the author presented problems to such assessment. Moreover many of the patients were given the Present State Examination (Wing et al., 1974) as many also took part in other ongoing studies in the Unit. It was thus considered that although not ideal, the Beck Depression Inventory (Beck et al., 1961) was a sufficient measure of depression for this study, given the experience of the staff responsible for the allocation of patients.

A one-way analysis of variance revealed that the depressed patient group were significantly more depressed than both the anxious group and the normal group, as measured by the Beck Depression Inventory. The same criterion, clinical diagnosis, was also used for the selection of the anxious control subjects. However, although the general anxiety, as measured by the General Anxiety Scale (Sarason, 1972) was slightly higher for the anxious group than the depressed group, the difference was not significant. It is possible that a more comprehensive scale (e.g. the Taylor Manifest Anxiety Scale, Taylor, 1953) would have differentiated the groups better. However, the reason why a shorter scale was presented was outlined in chapter 5. Although the anxious group was only marginally more anxious than the depressed group, they were significantly more anxious and more depressed than the normal control group. Although this is problematic in a study

of this nature, it is questionable whether a non-organic psychiatric control group can be found that provides a useful comparison group for depressive illness and which does not also exhibit some degree of depression. At any event, it seems preferable to have an anxious control group that also has a component of depression rather than an unspecified psychiatric control group as used by DeMonbreun and Craighead (1977) in their cognitive investigation of depression. In their study the unspecified psychiatric control group were not significantly different on depression, anxiety or hostility from a normal group.

Although the self-report levels of anxiety were not significantly different between the depressed and anxious group, the depressed group were found to have a significantly lower level of background electrodermal activity as measured by the mean number of spontaneous fluctuations per minute over the three parts of the experiment, namely test anxiety scale completion, problem solving and light detection. This measure has been shown to be a valid measure of anxiety in several studies (Lader, 1975).

Bearing in mind the disadvantages of not having objective criteria for selection of patients, it should be emphasised that all the clinical staff responsible for the allocation of patients to the different groups had experience of research in the affective disorders and were aware of the aims and needs of the study. Consequently it seems reasonable to argue that the anxious control group consisted of patients who suffered predominantly from anxiety as opposed to patients in the depressed group who suffered predominantly from depression. In this regard the comparisons between the groups can be considered to offer valid comparisons of depression as opposed to anxiety.

A. This study attempted to examine five basic questions. The first of these, as outlined on P. 138, was

In what ways do depressed patients differ from control groups in their attention and perception to positive and negative events?

This question was considered relevant because Beck (1974, 1976) has argued that depression results from distorted cognitive processes. The depressed individual is believed to cognitively distort information from his environment so that he arrives at a negative conclusion. For example, Beck (1976, P.119) argues that the depressed individual "evidently screens out, or fails to integrate, successful experiences that contradict his negative view of himself". In addition, the depressed individual is viewed as being hypersensitive to negative information. As Hammen and Krantz (1976) put it, "it is as if, for example, the (depressed) individual selectively attends only to dysphoric or pessimistic information or selectively interprets events to establish or verify pessimistic hypotheses". (P. 577) Experimentally Lewinsohn et al. (1975) found that depressed students are more sensitive to aversive events (mild electric shock) as measured by electrodermal changes, than non-depressed students. In contrast to Lewinsohn's et al. (op. cit.) study, this study found that depressed patients did not differ from control groups on electrodermal activity to negative (failure) events, but did significantly differ to positive (success) events.

It was found that depressed patients showed a significantly lower tendency to respond to the anticipation of a positive event (in this case the signalling of success) than both control groups. In addition, the depressed group showed a

significantly lower tendency to respond to the actual success event (signalled by the left light) than the anxious group. Analysis of the two epochs, before and during light presentation, also revealed that the depressed group had a significantly lower tendency to respond to a positive event, than did the two control groups. On the other hand, electrodermal activity to the anticipation of and the signalling of failure did not prove to be significantly different among the groups.

The perceptual data which measured perceptual thresholds to stimuli that had been paired with success and failure showed similar results. The depressed group had a significantly higher perceptual threshold to the light that had been paired with success (left light) than the normal control group, although there was no significant difference in perceptual sensitivity thresholds to the light that had been paired with failure.

An examination of the bias in the perceptual thresholds of each group revealed that normal control subjects tended to have a positive bias; that is they had a lower perceptual threshold for the light that had previously been paired with success than for the light that had been paired with failure. On this measure both the depressed and the anxious groups were found to have a significantly lower positive bias than the normal control group.

These results tend to indicate that depressed patients may be under-attentive to positive events, in contrast to the other groups, rather than being over-attentive to negative events. In other words they appear to be less affected and less physiologically aroused by positive events and less perceptually sensitive to stimuli that had been paired with success than normal controls.

These findings are in accordance with the growing literature which tends to show that positive and negative affects do not produce symmetrical effects on cognitive and behavioural parameters; for example, Masters and Furman (1976) found that while positive affect induced the expectation of future positive events, negative affect did not produce the reverse. Mischel et al. (1973) found that selective attention to the self was significantly influenced by success experiences, but subjects experiencing failure did not significantly differ from a control group in their allocation of attention. More recently Isen et al. (1978) found that inducing positive or negative mood by manipulating success or failure at a 'star trek' game, affected the recall of positive material, but the recall of negative material was not affected by this procedure. Similarly Teasdale and Fogarty (1978) found that inducing negative mood affected the recall of pleasant memories but did not affect the recall of unpleasant memories.

Thus, it appears from these studies that positive and negative mood both affect the positive elements of the individual's cognitive processing, while leaving the negative elements relatively unaffected. From this study, using a psychiatric population where the affective state was primary depression, it also appears that depressed subjects are relatively underattentive to positive events rather than 'hypersensitive to negative events' as was suggested earlier, as compared with anxious and normal controls. Such a view seems to have been based partly on the assumption of the symmetrical effect of mood on cognitive and behavioural parameters and partly on an imprecise definition of 'negative'. As will be discussed shortly, it may be necessary to discriminate carefully

between perceptions of loss and calling them negative perceptions and perceptions of threat and calling them negative perceptions also. However, before examining this semantic question it should be pointed out that these findings fit with a number of different theoretical perspectives.

The behavioural formulations of Ferster (1973, 1974), Lazarus (1968) and Lewinsohn (1974, 1975) have all argued that the primary focus for therapy with depressed individuals should be to increase the amount of positive reinforcement that they receive. All these formulations imply that it is a reduction in the positive (and not necessarily an increase in the negative) reinforcers in the environment which produces depression. Thus, their view, which in Ferster's (op. cit.) case is based on animal experiments, is that depression can be investigated, understood and treated solely by manipulation of positive reinforcement schedules. In a similar vein, Costello (1972) has argued that depression results from a loss in reinforcer effectiveness. That is, positive reinforcers lose their positively reinforcing properties, although why this happens is not convincingly explained (Eastman, 1976). Costello's work is based on clinical experience and many agree that the concept of a loss of reinforcer effectiveness fits the depressed individual's apparent general loss of interest in his environment.

Costello's idea that depressed patients suffer from loss of reinforcer effectiveness also fits neatly with some of the neurochemical theories of depression as discussed in chapter 3. These theories have tended to see depression as being caused by a reduction in the efficiency of the reward system so that the registration of positive reinforcement is impaired, i.e. because of

a biological abnormality in the reward system positive reinforcers become ineffective. What the behavioural and these neurochemical theories seem to have in common is the idea that depression results primarily from a reduction or deficit in positive feedback. None of these theories give much consideration to either aversive reinforcement or the punishment areas.

In a sense the cognitive theories of depression have upheld the view that it is distortions in positive feedback which are associated with depression. On the other hand, the terminology of the cognitive model has tended to confuse this issue: for example, Beck (1967) has discussed depression in terms of a negative view of the self, the world and the future, and in terms of negative cognitive distortions, etc. The use of this terminology has implied that mood has symmetrical effects on cognition and behaviour, and this assumption seems to have crept into theorising in the last few years. Thus recently De Monbreun and Craighead (1977) argued that in cognitive theory the depressed patient is seen as being ". . . hypersensitive to negative feedback so that negative feedback is interpreted as being more negative than it is and neutral or ambiguous feedback is frequently perceived as being negative". (P. 312) In fact, in their own study, DeMonbreun and Craighead (op. cit.) found no evidence for this view. Rather they found that it was only in the positive dimension (depressed patients underestimated the amount of positive feedback they had received in a high positive feedback situation) that depressives were different from a psychiatric and a non-psychiatric control group. Neutral feedback was not interpreted more negatively by the depressed patients. Moreover in an earlier study Nelson and Craighead (1977) found that depressed students were quite accurate in their estimation of negative feedback in a high

negative feedback situation. Thus it would seem that a great deal of care is needed in the terminology used to describe and investigate cognitive deficits in depression. It would be more appropriate to describe the cognitive deficits in depression as occurring only in the positive dimension, that is depressed patients are less positive in their expectations, evaluations, and attitudes than non-depressed individuals. Thus it can be seen that there is a growing consensus among behavioural, neurochemical and cognitive theorists of depression who, although disagreeing on the mechanisms involved, do seem to agree that it is a deficit in positive feedback rather than an increase in negative feedback that seems to be the crucial problem in depression.

The problem for cognitive theorists resides partly in the definitions of "negative" and "aversive". For example, an increase in threat appraisal and an increase in loss appraisal are often both referred to as negative appraisals in that they are unpleasant and aversive. Yet the appraisal of loss which Beck (1974, 1976) describes as the appraisal associated with depression) implies an appraisal of a reduction or withdrawal of some positive characteristic within the individual's personal domain. On the other hand, the appraisal of threat implies an appraisal of an increase in the harmful and negative characteristics within the individual's domain. It may often be difficult to separate these two appriasals. The appraisal of failure may be a case in point (this is discussed more fully in the next section). Nevertheless, as the evidence from this study and others is beginning to show, to be 'less positive' in cognitive processing does not necessarily imply a 'more negative' orientation in a strict symmetrical sense. Thus, as Lazarus and Launier (1978) point out,

different appraisals such as threat, loss, harm, danger, etc. may all be aversive, but the cognitive and behavioural repercussions of each appraisal will be quite different. Moreover, Lazarus and Averill (1972) argue that each different emotion should be understood in terms of a specific kind of appraisal. Consequently depression may be more clearly described and investigated by a precise definition of the appraisal believed to be associated with it (i.e. loss), avoiding assumptions of symmetrical effects of these appraisals on other cognitive processes.

In conclusion to this section, it can be said that the results of this investigation of the attention to and perception of positive and negative events in depressed patients show that depressed patients show deficits in their attention to positive events, as measured psychophysiologically, as compared to a psychiatric and normal control groups. They are also less positive in their perceptual sensitivity thresholds compared to a normal group. However, no differences among the groups were found in the attention to and perception of negative events. These results are in accordance with other recent studies of cognitive processes in depression.

B. In what way do depressed patients differ attitudinally from control groups?

The attitudinal and cognitive factors investigated in this study were: perceived control over external reinforcers, level of test anxiety as indicative of level of fear of failure, level of negative thinking and ability to estimate accurately performance on a problem solving task. In addition, perceived ability to improve on the problem solving task and aspiration discrepancy,

reflecting aspiration level on this task, were also investigated. It was predicted that the depressed group would be significantly more external than the normal control group, more test anxious than both control groups and would have a higher level of negative thinking than both control groups. It was further predicted that the depressed patients would show the largest judgement discrepancy, the lowest expectancy discrepancy and the highest aspiration discrepancy following the problem solving task.

The findings, using Rotter's Internal-External Scale, were in accordance with other findings (e.g. Harrow and Ferrante, 1969), in that the depressed group were found to be more external than the normal control group. However, this measure was a long way from differentiating the two ill groups, the anxious and depressed groups, and may thus reflect the fact that this scale is rather unspecific and related to psychopathology in general (Shybut, 1968). This finding may cast some doubt on Seligman's (1975) view that anxiety and depression can be differentiated on the construct of perceived controllability. Moreover, there was no correlation between level of depression as measured by the Beck Depression Inventory and level of externality in the depressed group, Although such a relationship has been found in mildly depressed subjects (e.g. Abramowitz, 1969, Prociuk et al., 1976), this relationship did not occur in this clinically depressed group. However, in both the depressed group and the anxious group, level of externality was highly correlated with level of negative thinking. Thus the tendency to perceive events as being outside one's control may be related to a more general tendency to think negatively in a psychiatric population. In other words, a high external score may reflect a generalised

negative (or less positive) orientation of cognitive processes, rather than a specific tendency to have an external view of reinforcement contingencies. This would seem to be supported by the findings that depressed patients become more internal on recovery (Harrow and Ferrante, 1969), and in this study by the finding that this correlation completely disappears on recovery from depression. Thus, in retrospect, this scale lacks specificity for this type of study and its results are difficult to interpret. A high externality score may also simply be an illness effect, especially as found in depressives.

The study also examined test anxiety (Sarason, 1972). It was considered a relevant measure for two main reasons. Firstly, as suggested in chapter 4, depressed and depression-prone individuals may be prone to a high fear of failure, of which test anxiety has been one of the main measures (Weiner, 1972). Secondly it has been argued that highly test anxious individuals are prone to negative evaluative cognitions such as "maybe I won't succeed", "I'm stupid" etc. (Liebert and Morris, 1967, Sarason, 1972). It was noted that this formulation of test anxiety was similar in some respects to Beck's (1976) view that depressives are also prone to have negative self evaluative cognitions. Moreover, Goldfried and Soboinski (1975) found that the tendency to hold irrational beliefs was correlated with various pencil and paper measures of test and evaluative anxiety. Although these authors were more concerned with emotional arousal in certain situations, their study does add some validity to the view that high test anxiety may be associated with the tendency to hold irrational beliefs and to make negative evaluations in assessment situations.

The results showed that the depressed subjects were significantly higher on this measure than the normal controls but did not differ from the anxious group. Also in both ill groups the level of test anxiety correlated with the level of general anxiety, which makes the results more difficult to interpret. However, in the recovered depressed group, the level of test anxiety was correlated with the level of negative thinking. If negative thinking is an important aetiological factor in depression, then it may be that high fear of failure should also be considered as one, or high negative thinking may entail high fear of failure or vice versa. It is, however, not possible to determine this from correlational studies.

The failure to find a significant difference between the anxious group and depressed group on fear of failure needs some consideration. Firstly, the scale itself may not have been ideal for measuring the concepts considered relevant in this study, especially since this scale concentrates on academic situations. Nevertheless it has been used in many studies investigating fear of failure (Birney et al., 1969), and was considered to be useful for this study in spite of its drawbacks. The other problem concerns the whole concept of fear of failure itself. As Birney et al. (op. cit.) point out, fear of failure can be situational, specific or general. Individuals may be fearful of failing academically but not in social situations. Other individuals may suffer the reverse. Thus, in the depressed individual the concern with failure may be related to a number of personal attributes or just one or two, e.g. failing as a parent. This diversity makes the construct difficult to measure. Nevertheless,

even with these problems, depressed patients were found to be significantly more test anxious than normals.

The other problem that may explain why the anxious and depressed groups did not differ on this measure concerns the question of why some individuals find failure so aversive in some situations. Chapter four attempted to argue that for the depression-prone individual, failure is aversive because of an expected withdrawal (loss) of secondary reinforcers which is seen as contingent on failing: e.g. one fear expressed by one patient in the study was "If I'm not a good mother and cope with the children, my husband won't love me and my own parents won't visit the house". In addition this patient couldn't respect herself unless she thought she was coping well at home. These attitudes, which Beck (1967, 1976) has pointed out, all centre on the common theme of loss if some standard is not met. And, as Beck (1974) says, the patient often cognises the event (e.g. failure) as if it has actually happened. Thus, for the depressed subject, the fear of failure may centre primarily on the theme of loss. In the anxious patient on the other hand, failure may be aversive because of expected punishment. This view of course is speculative but it does seem reasonable to argue that fear of failure can be related to different concerns, the appraisal of subsequent loss being only one. The outcome, however, is that although fear of failure may arise from different learning histories and for different reasons in an anxious-prone as opposed to a depression-prone individual, the level of fear of failure as measured by a scale that does not measure <u>why</u> the individual is fearful of failing, may appear similar in both types of individuals. Thus it would seem useful for

further research in this particular area, not only to examine the fear of failure concept itself but also to examine the question of why individuals are fearful of failing in certain situations.

There is a further important question that fear of failure research in depression should concern itself with. This question concerns the criteria by which an individual judges himself to have failed. As Bandura (1977) points out, if individuals judge themselves by unrealistic standards and aspirations then failure is often inevitable with a consequent increase in distress.

> "Dysfunctional self-evaluative systems figure prominently in some forms of psychopathology by activating excessive self punishment or creating self-produced distress that motivates various defensive reactions. Many seekers of psychotherapy are talented and free of anxiety, but they experience considerable personal distress stemming from excessively high standards of self evaluation and unfavourable comparisons with models noted for extraordinary achievements." (Bandura, 1977, P. 140)

Bandura (op. cit.) goes on to argue that attempts to maintain old standards in the face of loss of ability through age or physical injury can further be the source of self-devaluation and distress. Thus the criteria by which an individual judges himself to have failed demands consideration in fear of failure research. In this study it was found that aspiration discrepancy (the score derived from subtracting the individual's estimate performance score on the problem solving task from his 'pleased and satisfied' score) was found to correlate highly with level of depression ($r = 0.61$) and general

anxiety (r = 0.57) in the recovered depressives. It was only for this group that these correlations were found. This result may offer some support for Beck's (1974, 1976) and Bandura's (1977) view that the discrepancy between actual performance and aspired-to performance can be a source of distress in certain individuals of which the depression-prone individual is likely to be one.

Before leaving this aspect of the discussion one negative result should be mentioned. It will be recalled that Birney et al. (1969) have argued that the fear of failure person is likely to be sensitive to cues signalling failure. The results from the attentional and perceptual investigation in success and failure events found no difference between the groups in their attention to and perception of failure events. In other words, the depressed group appears no more sensitive to failure cues than the two control groups. Thus it would seem that subsequent research in this area should address itself to the questions of (a) why do individuals find failure aversive in some situations? and (b) by what criteria does the individual judge himself to have failed?

Another cognitive factor (using a self-report questionnaire) investigated in this study was the level of negative thinking. This was measured by administering the Beck Story Completion Test (BSCT). This questionnaire was provided by Beck. This scale was designed to examine various negative and exaggerated cognitions that are believed to be operative in depressed individuals (Weintraub et al., 1974). The scale was designed to measure cognitions centering on (a) expectations of discomfort, (b) expectations of failure, (c) negative perception of interpersonal relationships and (d)

negative perceptions of self. In this study the sub-divisions were not examined separately but rather as a 'cognitive total' (Weintraub et al., op. cit.).

The findings indicate that depressed subjects are significantly more negative in their thinking than normal controls. However the depressed group were not significantly different on this measure compared to the anxious control group. Moreover some of the highest scores on this scale were found in the anxious control group (table 6.36, P.224). In addition this scale was found to correlate with both general anxiety and level of depression in the anxious group, and with general anxiety in the normal group. However, neither anxiety nor depression correlated with BSCT scores in the depressed group.

As far as the author is aware this is the only study to have used this scale with both a clinically depressed group and a clinically anxious group. Thus, although the relationship between depressed mood and negative thinking has been demonstrated in a normal group (Weintraub et al., 1974), this study does not support this finding in a clinically depressed group. A clear interpretation of this finding is difficult because, as suggested earlier, any relationship that might exist between negative thinking and mood may be distorted by other factors such as biological changes which may occur in clinically ill patients. However in view of the correlations of this scale with general anxiety, it must be said that the specificity of this scale to depression is dubious. Consequently it is felt that the use of this scale with clinically depressed patients is very limited, although subsequent research may wish to examine the possibility that anxious patients and depressed patients can

be discriminated by breaking down the scale into its sub-classes. Unfortunately, this research did not do this.

In the recovered depressed group, the level of negative thinking did correlate with test anxiety as was discussed on p.202 . If negative thinking is aetiologically important in depression, then the question of why some individuals tend to have more negative thoughts than others needs to be answered. In this regard part of the answer may lie in the fact that those individuals with a high fear of failure may have higher expectations of loss following failure. However, as mentioned earlier, the relationship between these two concepts is not clear at this point.

The perception of failure may be inevitable due to the pursuit of unrealistic standards and aspirations. The performance estimate scores which were concerned with the estimate of performance on a problem solving task, the perceived ability to improve on the task and the level of aspiration discrepancy did not provide any significant results between the depressed and normal groups. This study examined discrepancy scores rather than raw scores because it was felt that such data were more useful and that judgement discrepancy, expectancy discrepancy and aspiration discrepancy were more directly relevant to the theoretical axioms of the cognitive model of depression. In retrospect, the failure to obtain significant results on these measures may be attributable to two factors. With the experiment being intended to examine attentional and perceptual sensitivities to success and failure events, the design was required to be symmetrical. As a result some of the questions that could be answered correctly may have been easily distinguishable from the questions

that could not, although only two subjects actually seemed to be aware of this difference and many subjects thought they could have answered more questions given more time. Secondly, the actual number of questions that could be answered correctly was ten. This may have been too small a number for significant differences on the various discrepancy scores to appear. In other more recent studies (e.g. Nelson and Craighead, 1977), many more trials have been used. However, since this study required subjects to complete a number of questionnaires following the experimental session, it was felt that the time taken on the problem solving task could not be expanded. Thus the conflict of aims did not maximise the required characteristics for this aspect of the study. Nevertheless, the results were in the predicted direction although not reaching significance.

Although the results on the discrepancy scores did not reveal significant results between the normal and depressed groups, the anxious group were found to have a significantly higher aspiration discrepancy score than the normal group. Thus the specificity of this concept to depression may also be questionable. What is required is more research that examines these constructs in depression but also matches them with an anxious control group. Future research might profitably adapt the Nelson and Craighead (1977) design with the aim of examining more closely various expectations of success, perceived ability to improve and the possible mismatch between the subjects' perceived actual performance and aspired-to performance. But in view of these results some relevant control group such as an anxious group is required before any conclusions can be reached as to the specificity of the findings to depression.

C. How specific to depression are the cognitive and attitudinal distortions which have been implicated by previous research?

The point just made at the end of the previous section clearly has a bearing on this issue. Moreover the level of externality and the level of test anxiety were found to be almost identical in the anxious group and the depressed group. In addition, the scores on the negative thinking questionnaire, which was designed for measuring depressive thought content, revealed that the anxious group scored marginally higher than the depressed group on this measure. It should be recalled that the anxious group had a moderately high depression score (17.06 on the BDI). Thus all these three measures would appear to be as equally relevant for anxiety as they are for depression. Moreover, negative thinking was found to have high correlations with anxiety in both the anxious group and the normal group.

As mentioned earlier, the various discrepancy scores following the problem solving task also failed to distinguish the anxious group from the depressed group.

From these results it would appear that caution should be exercised in interpreting results which claim to show specific cognitive deficits in depression, in the absence of a suitable control group.

It should be pointed out, however, that on the attentional measures to positive and negative events, the depressed group were clearly distinguishable from the anxious control group. On all the measures of attentional sensitivity to positive events (i.e. over the anticipatory, signalled and combined epochs), the depressed group was found to be significantly less attentive than the anxious

control group. On the perceptual measures, however, no significant differences emerged.

In view of these findings it might be suggested that the attitudinal and cognitive measures, while differentiating the anxious group and depressed group from the normal group, are not specific enough to differentiate emotionally ill individuals, even though the characteristics of each disorder are different. Thus, as in the case of fear of failure, the question of why the individual finds failure aversive may be more important than overall fear of failure levels, and so with the other measures. The issue which may differentiate anxiety and depression may not be so much the absolute level of scoring on a certain scale (e.g. I-E or BSCT) but rather the reasons behind such scoring. It is possible that the difference between anxiety and depression may lie not so much in one group thinking more negatively than the other or one group being higher in fear of failure than the other group, but rather each may have different reasons for thinking negatively or being fearful of failure. The only factor that does seem to differentiate anxiety from depression is that anxious subjects are not 'less positive' than normals in the way depressed patients appear to be. Thus, as Beck (1974, 1976) points out, it is perhaps the appraisal of loss (the withdrawal or devaluation of some positive attribute or reinforcer) that distinguishes the depressed patients from other psychopathologica groups. It must be emphasised then that research should attempt to go behind the scenes, as it were, to discover the various types of appraisal that lead to various psychopathological response patterns. Although the absolute level of negative thinking, fear of failure or externality may appear the same in different pathological subjects, the appraisals (of loss, harm, threat, etc.) may be quite

different. Moreover it would seem essential to have adequate controls. It must, however, be pointed out that anxiety may be the most difficult condition to differentiate from depression, as depressed patients seem invariably to have a high level of anxiety and in this study at least the anxious subjects had a high level of reported depression.

D. 1. In what way do depressed patients change in their attention and attitudes after recovery?

2. How similar to the normal control group do recovered patients become?

These two related questions centre on the issue of whether it is possible to consider recovered depressed patients more vulnerable to depression by being significantly different from normal control subjects on certain of the parameters measured. Unfortunately the results do not provide any clear-cut answers to these questions.

The level of depression was significantly lower on recovery from depression as clearly expected. However, the recovered depressives' level of depression did not significantly differ from the anxious control group and was significantly higher than the normal control group. The level of general anxiety was also significantly lower on recovery from depression and was lower than in the anxious control group. Nevertheless it was still significantly higher than the normal control group. This problem of a significantly higher level of depression and anxiety in the recovered depressives as compared to the normal control group requires consideration. Firstly, as mentioned in chapter 5, recovered depressed subjects could not be excluded from this study on the basis of their Beck Depression Inventory scores since clinical diagnosis was the criterion for

selection. When a patient did score over ten on the Beck Depression Inventory, the clinician in charge of the patient's treatment was notified. In all cases the clinician confirmed his original diagnosis. Although one woman scored 35 on the BDI, the research consultant in charge of her treatment was very sure that she had recovered from her depressive illness and was fit to return home. He felt that this high BDI score was not a true reflection of her affective state. Thus the problem of not having an objective assessment criterion by which patients would be considered recovered has proved an unfortunate disadvantage. Much depends on the reliability of the clinician's judgement. However it must be borne in mind that in psychiatric research, clinical judgement is often used as the external validation criterion and Kreitman et al. (1961) have demonstrated that psychiatric diagnoses are more reliable than some critics would lead one to believe.

The background level of spontaneous fluctuations during the three experimental sessions of test anxiety scale completion, problem solving and light detection did not significantly change on recovery from depression. However, some change had occurred, so that the recovered depressed subjects did not significantly differ from the normal group on these measures (when ill the background level of spontaneous fluctuations during the test anxiety scale completion and during the problem solving task was found to be significantly lower than the normal group). The recovered depressed group, as did the normal group, had a significantly lower level of background spontaneous fluctuations during the light detection (vigilance) task compared to the anxious group.

The attentional and perceptual parameters showed that there was no significant difference in the scores of depressed patients when ill and after recovery. However a sufficient change had occurred to remove some of the significant differences that had been found between the depressed patients when ill, and the two control groups.

Depressed patients when ill were found to be less attentive as measured by an electrodermal measure to positive (success) events, compared to the normal and anxious control groups. On recovery, this significant difference had disappeared. The data show that recovered depressed subjects do not differ from normal or anxious control subjects. An examination of the changes that did occur on recovery from depression (table 6.66) showed that the largest changes occurred on the electrodermal activity to the positive events. This finding tends to support the view that the depressed subjects when ill were indeed less attentive to positive events compared to the two control groups, rather than the difference being due to the lower level of background electroderma activity.

When ill, the depressed patients were found to have a significantly higher detection threshold to the left light (success) than normal subjects. On recovery from depression, no significant difference between the groups was found on this measure. However, as on the attentional measures, this change was not sufficient to produce a significant difference between the depressive's left light sensitivity threshold when ill and after recovery. Moreover, an examination of the bias in perceptual sensitivity thresholds (obtained by subtracting

the perceptual sensitivity threshold to the left light from the perceptual sensitivity threshold to the right light) revealed that the recovered depressives still had a 'less positive' bias in their perceptual sensitivity thresholds than the normal control group, and were not significantly different from the anxious control group. It may be then, that although the depression-prone individual does not see events negatively, he nevertheless may tend to see them less positively than normal controls.

The results on the changes in the attitudinal and cognitive parameters on recovery from depression are difficult to interpret. Externality, which it had been predicted would be reduced on recovery from depression (i.e. depressed individuals would become more internal on recovery), was found to move in the predicted direction but not significantly so. The idea that recovered depressives would be more internal than their normal counterparts does not seem to be supported by this finding. However, because this scale may lack the required specificity, the implications of this finding are not clear.

Test anxiety, used in this study as an indirect measure of fear of failure, was found to have been reduced on recovery from depression, but not significantly so. However, although higher than the normal controls', the recovered depressives' test anxiety scores did not differ significantly from normal level. This result also poses problems of interpretation. It may be that the level of fear of failure in the depression-prone individual lies in the upper part of the normal range. However, as the depression-prone individual engages in certain achievement behaviours, i.e. attempting to reach certain internalised goals and standards, he becomes sensitised to the possible loss of secondary reinforcers that may be considered contingent on non achievement of these

standards. The perception of loss as the consequence of failure is aversive and may sensitise the individual to these very consequences. This sensitisation to the consequences of failing to achieve various standards may reduce the individual's expectation of success and may lead to negative thinking. That is, as the individual becomes sensitised to the consequences of failing, he begins to cognise the event as if it has actually happened, and experiences a sense of loss. Indeed Beck (1974, 1976) does make the point that depression-prone individuals often construe 'hypothesised loss' as an actual event and can experience a lowering of mood as a result.

The results obtained from the Beck Story Completion Test which measures negative thinking revealed that on recovery from depression, depression-prone individuals did 'think' less negatively than when ill and were not significantly different on this measure than their normal counterparts. In fact, this was the only attitudinal or cognitive measure used in this study that did significantly change on recovery from depression. This result lends support to the view that negative thinking as measured by this scale is related to depression (though other results suggest it is also related to anxiety). However, although negative thinking may be causally related to a lowering of mood, two points should be borne in mind. Firstly, although negative thinking may be causally related to mood, depressive illness probably involves dysfunctions at a number of different levels besides the cognitive level (Akiskal and McKinney, 1975). This fact may explain the finding that while negative thinking was found to be significantly lower in recovered depressed subjects, the level of negative thinking and the level of self-rated depression did not correlate in either the depressed or recovered

depressed subjects. The failure to find a correlation between negative thinking and depression in the recovered depressives is a little more problematic. However, it must be pointed out that the Beck Story Completion Test examines only how the individual cognises certain events as actually happening. No measure is given of how the individual would 'like' the outcome of the event to be. In other words, it does not provide a measure of the mismatch between the individual's level of aspiration and perceived actual performance. However, this may be a crucial factor in the whole concept of negative thinking in depression. In other words, it is not the level of negative thinking itself which is directly related to mood, but rather the extent to which the individual sees himself as falling short of his aspired-to level of attainments. The correlations between both the level of depression and the level of anxiety with aspiration discrepancy, as found (only) in the recovered depressed subjects may provide some support for this view.

Secondly, the finding that the recovered depressed level of negative thinking did not significantly differ from the normal control group's indicates that the depression-prone individual when well does not think any more negatively than normals. Thus, if an increase in the level of negative thinking is responsible for the lowering of mood, some other factor must be responsible for triggering negative thinking. As just described, one possibility may be a sensitisation to failure. On the other hand, Beck's (1967) view is that the negative cognitive triad, which is presumably responsible for negative thinking or is the manifestation of it, becomes dominant in the individual's thought process, when he experiences events in his environment that are similar to those that

lead to the formation of the triad, in the individual's childhood.

Whether this 'sensitisation to the consequences of failure' idea or the more 'historic reoccurring events' view is more useful is for future research to decide. The point is, however, that negative thinking, as measured by this scale, is not a stable component. Consequently, some further factor(s) must be sought which help to explain what triggers an increase in negative thinking, and why some individuals appear more vulnerable to this than others.

As already discussed, the judgement discrepancy, expectancy discrepancy and aspiration discrepancy scores did not differentiate the depressed, the anxious and normal groups. In addition, on recovery from depression, no significant change was found to have occurred. Thus these results do not merit further discussion except to point out that the recovered depressed subjects still had a marginally higher aspiration discrepancy score than the normal control group, though not significantly higher. However, in the recovered depressed, aspiration discrepancy, reflecting a mismatch between the individual's perceived performance and aspired-to performance on a problem solving task, was found to be correlated with level of depression and level of anxiety. Consequently it is suggested that these discrepancy parameters merit further investigation but in a more expansive design.

II. EXPERIMENT TWO

The second study of this thesis examined the difference between a fear of failure group and a high hope of success group on the parameters measured in the

previous study. The main issue of interest was whether or not high fear of failure subjects could be considered to be more vulnerable to depression than low fear of failure subjects. The reasons why such might be the case can be listed as follows:

1. As Birney et al. (1969) point out, the history of fear of failure as a personality trait has its roots in such problems as anxiety, guilt, shame and feelings of inferiority. More recently McReynolds and Guevara (1967) have shown that schizophrenics and mixed neurotics had a higher level of fear of failure than normals. Thus there is a link in the literature of the fear of failure concept with psychopathology.

2. With regard to depression, Cohen et al. (1954) and Becker (1960) argued that manic depressives have high value achievement; and high value achievement, it has been argued, "shows itself primarily as a fear of being unsuccessful" (McClelland et al. (1953).

3. Weiner (1972) has pointed out that subjects identified as having a high fear of failure have been found to make internal stable attributions for failure, i.e. they blame failure on some internal attribute such as lack of ability. Beck (1967, 1976) has also argued that depression-prone individuals tend to make similar attributions, that is they blame negative events on some personal attribute.

4. Later work by Weiner et al. (1978) has shown that the tendency to blame internal attributes (lack of ability) for failure may well be associated with depression.

5. Beck (1976) has argued that the attitudes and rules that produce a

vulnerability to depression are rules such as: "In order to be happy, I have to be successful in whatever I undertake"; "To be happy I must be accepted (liked, admired) by all people at all times"; "If I'm not on top, I'm a flop"; "If I make a mistake, it means I'm inept", etc. It was felt that many of these rules centred on the aversity of failure and would produce a high fear of failure in the individual who adopted them. Thus, it was considered that high fear of failure individuals may well hold some of the attitudes described by Beck (1976) as being vulnerability factors to depression.

For all these reasons it was considered that fear of failure individuals may be more prone to mood disorder than non-fear of failure subjects. Having already obtained discriminating results on a number of parameters for a depressed and recovered depressed population, it was decided to examine high fear of failure subjects in relation to these parameters.

Subjects for this study were obtained from a normal population, chosen on the basis of their answers to the Success-Failure Inventory (McReynolds and Guevara, 1967) and allocated to either a hope of success group (H.S.) or a fear of failure group (F.F.), as described in chapter seven. The Success-Failure Inventory (SFI) was used as a method of selecting subjects in preference to the better known Test Anxiety Scale (Sarason, 1972) because the SFI provides a measure of both the hope of success and fear of failure motivation. The Test Anxiety Scale on the other hand only provides a measure of the fear of failure motivation and is more indirect (Birney et al., 1969). For this study a measure of both hope of success and fear of failure was required.

As described in chapter seven, the experimental procedure and measures

used in this study were the same as those used in the first study.

This study set out to examine three main questions as described in chapter seven. The first of these questions was:

A. Do high fear of failure subjects compared to high hope of success subjects differ on their ratings of depression and anxiety?

A one way analysis of variance showed that the F. F. group rated themselves significantly higher on the Beck Depression Inventory than the H.S. group. However, the mean BDI score for the F. F. group only reached six which is not indicative of depression. On the other hand, an examination of the range of scoring in each group (0-3 for the H.S. and 0-19 for the F. F.) does indicate that some of the F. F. group might have been considered to be mildly depressed (five subjects in this group scored above ten). Thus it would seem reasonable to conclude that some of the individuals in the F. F. did experience low mood at the time of testing, whereas this was not the case for the H.S. group. Consequently it can be argued that the strength of the fear of failure motive may be associated with low mood in some individuals. This association is further supported by the finding that fear of failure correlated with the Beck depression scores in the F. F. group. Moreover, this correlation held for both measures of the fear of failure, namely the SFI and the TAS.

The highest correlations with level of depression in the F. F. group was found with negative thinking and general anxiety. Thus in this group, in which some members could be considered mildly depressed, the association between negative thinking and mood was strongly confirmed. This is in line with Weintraub et al.'s (1974) finding, although it should be stressed that here,

as in their study, the subjects were drawn from a normal population and, as discussed in the previous section, this relationship does not seem to occur with a clinically depressed group.

The level of general anxiety was also found to be significantly higher in the F. F. group compared to the H. S. group and again a few of the subjects in the F. F. group scored quite highly on this scale. Thus, not only level of depression but also general anxiety seem to be higher in fear of failure subjects than it is in hope of success subjects.

As found in the previous study, general anxiety correlated highly with negative thinking, again casting some doubt on the specificity of negative thinking (as measured by the Beck Story Completion Test) to depression. The highest correlation, however, was found between the level of general anxiety and level of depression. Somewhat surprisingly, in the fear of failure group the level of general anxiety did not correlate with either test anxiety of the other measure of fear of failure, the SFI. Thus in the F. F. group, fear of failure seems more associated with depression than anxiety, although the level of general anxiety was higher in this group than in the H. S. group.

In the H. S. group, test anxiety correlated with level of depression but not with level of general anxiety. However, the low level of both depression and anxiety in this group may make this finding spurious.

This result seems to support the view that having a high fear of failure does make the individual (at least in some cases) more vulnerable to low mood. It should be added that some of the subjects in the F. F. group told the author that they did occasionally feel quite depressed, whereas none of the subjects

in the H.S. group offered this information.

The background level of electrodermal activity during the completion of the test anxiety scale, the problem solving task and the light detection task was not significantly different between the groups.

The second question this research examined was

B. How do high fear of failure subjects differ from high hope of success subjects on the parameters of attention and perception to positive and negative events, and on the various attitudinal and cognitive measures as used in this study?

According to the theory of fear of failure, high fear of failure subjects should be more attentive to failure cues than high hope of success subjects (Birney et al., 1969). Although the results were in the predicted direction, the attentional parameters yielded only one significant result. It was found that both groups showed more electrodermal spontaneous fluctuations to the right (signalling failure) light (DUR) compared to the left (signalling success) light (DUL). However, the F.F. group had a significantly greater negative bias than the H.S. group. Thus, although the groups did not differ on their electrodermal responses to either a failure event alone, or a success event alone, the F.F. group did show the greater negative bias when the two scores were subtracted (DUL-DUR).

The perceptual sensitivity threshold measures showed that unlike the electrodermal measures to signalled success and failure, both groups had a slightly lower detection threshold for the left light (which had signalled success) than for the right light (which had signalled failure). However, the H.S. group

had a larger positive bias than the F.F. group, the difference approaching significance. Thus, in the absence of significnt results on these measures, no firm conclusions can be drawn.

As in chapter six, the attitudinal and cognitive factors investigated in this study were perceived control over external reinforcers, level of test anxiety, level of negative thinking and ability to estimate accurately performance on a problem solving task. In addition, perceived ability to improve on the problem solving task and aspiration discrepancy, reflecting a mismatch between actual estimated performance and aspired-to performance were examined.

The results, using Rotter's (1966) Internal-External Scale, revealed that the F.F. group were significantly more external than the H.S. group. The level of externality was found to correlate with level of fear of failure as measured by the S.F.I. In addition the level of externality correlated with negative thinking (this correlation was also found for the depressed group and anxious group in the previous study). Thus it would appear that the higher the level of fear of failure the greater the lack of perceived control over reinforcers. This would seem predictable since if an individual does have a high fear of failure motivation, at least part of this fear is probably related to the perception that success or failure is outside his control. An individual who perceives himself as having complete control over reinforcers may be expected to have less fear of failure. On the other hand, this conclusion is complicated by the fact that, as Weiner (1972) points out, the I-E scale confounds the dimensions of stable/unstable with internal/external. Thus, it may be that it is precisely because an individual perceives the events as

being controlled by himself (i.e. the future is in his hands) that he fears the consequences failure, i.e. he only has himself to blame. As a result, although the tendency towards externality has been associated with depression and anxiety (Harrow and Ferrante, 1969), and other psychopathological factors (Lefcourt, 1976), the implications of these findings and the results reported in this study are not straightforward.

The results using the test anxiety scale (Sarason, 1972) revealed that the F.F. group had a significantly higher level of test anxiety than the H.S. group. Thus, if test anxiety does reflect the tendency to make negative self evaluations (Sarason, 1972, Liebert and Morries, 1967, Meichenbaum, 1977), the F.F. subjects would appear to be more prone to such evaluations than H.S. subjects. Moreover, Goldfried and Saboinski (1975) found that the tendency to hold irrational beliefs was associated with test and evaluative anxiety. Both these factors, the tendency to make negative self evaluations and the tendency to hold irrational beliefs, have been argued to be important factors in depression. Thus it is important to point out that in two groups chosen to differ in these relative orientations to approach and avoid success and failure, the level of test anxiety correlated with the Beck depression inventory scores. However, in view of the low BDI scores in the H.S. group no clear inference can be made. On the other hand, in the F.F. group, level of depression correlated both with level of test anxiety and fear of failure (as measured by the SFI). In view of these findings it does seem reasonable to suggest that test anxiety and fear of failure may, in certain individuals, be contributing factors to low mood.

In addition to these findings, one negative finding of this study gives cause

for concern. It was found that the correlations for the two groups combined revealed a significant correlation between test anxiety and the level of fear of failure as measured by the SFI, indicating that these two measures are related. However, separate group correlations did not reveal any significant correlations between these two measures. Since both scales are considered to be measures of fear of failure, the absence of a significant correlation between them, especially in a high fear of failure group, is very disconcerting. This finding strikes at the core of the problem in fear of failure research: that is that the measures that are used in this type of research may not necessarily be measuring the same thing. Weinstein (1969), for example, in his correlational study, found very low correlations between the various measures of achievement motivation and fear of failure.

The Beck Story Completion Test, which was designed as a measure of negative thinking, revealed that the F. F. group had significantly higher negative thinking scores than the H.S. group. Moreover in the F. F. group, level of negative thinking was very highly correlated with level of depression ($r = 0.70$). This finding would lend weight to Weintraub's et al. (1974) and Beck's (1974, 1976) contention that negative thinking is an important determinant of low mood. However, the correlation between negative thinking and general anxiety was also found to be high in the F. F. group indicating again that negative thinking as measured by this scale is not specific to depression.

Level of negative thinking was found to be correlated with fear of failure as measured by the SFI, in the F. F. group. In the H.S. group the SFI also

correlated with negative thinking indicating that the lower the hope of success orientation, in a high hope of success group, the higher the level of negative thinking. It seems then that fear of failure as measured by the SFI is related to negative thinking. For individuals who have a high fear of failure there appears a greater tendency to imagine negative outcomes in certain hypothesised situations as depicted by the Beck story completion test. On the other hand, the test anxiety scale was found to correlate with depression in the F. F. group. This finding again tends to indicate that the two scales are measuring different things. The pattern of correlations, however, (the SFI correlating with negative thinking, and the TAS correlating with level of depression in the F. F. group) suggests that both measures (the SFI and the TAS) play some contributory role to low mood in some individuals. Whether these individuals felt threatened by failure, or anticipated loss following failure cannot be determined by this study. Nevertheless, the results do offer support for the view that a high fear of failure can influence negative thinking and low mood.

On the parameters of judgement discrepancy, expectancy discrepancy and aspiration discrepancy, no significant differences were found between the two groups. As suggested earlier, however, it may well be worth examining these variables in a more expansive design.

The last question of this study is the most difficult to answer.

C. Are fear of failure subjects more vulnerable to depression than hope of success subjects?

In general, the answer to this question seems to be that fear of failure subjects are indeed more vulnerable to depression than hope of success subjects. F. F.

subjects were found to have higher Beck depression inventory scores and, although a mean score of six is not indicative of depression, five members of this group did score over ten. F.F. subjects had a high externality score and various studies (e.g. Harrow and Ferrante, 1969, Prociuk et al., 1976) have found a relationship between depression and externality. F.F. subjects were found to have high test anxiety which in theory may be related to a proneness to depression and in the previous study test anxiety was found to be higher in depressed patients than in normals. F.F. subjects were found to have a higher negative thinking score than H.S. subjects. Again in theory this construct is related to a proneness to depression, and in the previous study depressed subjects were found to have a higher level of negative thinking. Thus the evidence does suggest that F.F. subjects are more likely to suffer from low mood than low fear of failure subjects. On the other hand there is reason to be cautious of this interpretation. Firstly F.F. subjects did not appear to be underattentive to positive events compared to hope of success subjects, as measured by electrodermal activity. Yet this was one measure which did separate the depressed patients from the normal and anxious controls in the previous study. Moreover all the results in this study which found F.F. subjects to have higher externality, higher test anxiety and higher negative thinking than the H.S. group could be equally well interpreted as indicating that F.F. subjects are more vulnerable to anxiety than H.S. subjects. None of these findings can be

interpreted as showing that F.F. subjects are specifically more vulnerable to depression rather than anxiety. Nevertheless, these results do offer some support to the view that fear of failure may make individuals more vulnerable to more negative affect in general. The different affective consequences of having a high fear of failure may be teased out by examining the reasons behind the individual's 'fear of failure'. The prediction from the work discussed here is that those who fear failure because of a perceived loss of secondary reinforcers (e.g. love, social approval and also loss of self-esteem) and who tend to construe 'hypothesised failure' as an actual event would be more vulnerable to depression. On the other hand those who fear failure because of threat of punishment as considered by Birney et al., (1969) and who are concerned more with the avoidance of failure by avoiding situations where failure is seen as possible, may be more vulnerable to anxiety. However, this is a hypothesis which has not been tested in this work and thus remains an open question. Moreover the correlational results tend to show that general anxiety and level of depression are highly correlated in F.F. subjects. Thus it may be that both the appraisal of loss and of threat as consequences in a failure situation are important. Alternatively, it may be that as long as the failure is perceived as a threat and potentially avoidable, then anxiety may be the more probable affect. However, once the individual begins to construe the 'hypothesised' or potential failure as actually having happened, depression may be more likely. These speculations are for future research. From this study, though, it would seem that a high fear of failure does merit further investigation since there

does seem a tendency for high fear of failure subjects to be more prone to low mood than high hope of success subjects. In the light of the interest now shown by cognitive psychologists in depression, the high fear of failure subject may prove to be one subject who though vulnerable to depression and anxiety, may not actually be ill. The possible reasons for this vulnerability have already been explained. However to go back to the question posed at the beginning of this section, it would appear that the fear of failure subjects scored very like recovered depressed subjects on most measures.

To end up the discussion, it must be pointed out that there is an implication in this study and other psychological studies that a vulnerability to low mood is also a vulnerability to affective illness which one should be extremely cautious about. This issue is taken up in the conclusion.

III. EXPERIMENT THREE

This study set out to investigate cognitive and attitudinal parameters in recovered depressed patients. In cognitive theory, work with recovered depressed patients has been a neglected area of research. Yet, cognitive theory makes strong claims to providing an aetiological theory of depression.

A. Comparison of the two recovered depressed groups

Analysis following the third study found that differences existed between a group of recovered depressed patients selected by Consultant judgement and a similar group selected by more objective criteria. These differences are manifest on the level of self-reported depression and the locus of control measures. The first question which needs to be asked in

view of the higher BDI levels of the former group is (a) did the Consultants make poor decisions on their patients' recovery and/or (b) are the patient groups different. The answer to the first question is somewhat controversial and is probably unanswerable. The answer to the second is much more serious from a research point of view.

Consideration of the selection of patients in the two studies reveals major sources of difference. In the first study the sample of recovered depressed patients was dictated by the sample of depressed patients selected; they were the same patients on follow-up. The use of a follow up design presents problems since, while it may be possible to offer a definite diagnosis when the patient is ill, it is not always possible to predict treatment response or outcome. In other words patients with a diagnosis of 'depressive illness' cannot all be expected to improve uniformly or to the same degree within a limited time. Much can depend on a host of factors outside clinical control including pre-morbid personality, marital problems, economic difficulties and social support. Also of importance is the type of depression the patient suffered from. It is well known that some physical treatments work well with some types of depression but for some of the less severe depressions, are perhaps, less effective (Paykel and Rowan, 1979). In any event the follow up of the patients in Study One revealed different degrees of recovery. Some patients were very well indeed while others, believed to be over their 'illness', were still rather unhappy people with a fair degree of dissatisfaction with their lives. Continuation with physical treatment may often be considered necessary to prevent relapse (Paykel et al., 1975) but it is unlikely to bring

any further improvement for such people. Paradoxically it may be that cognitive therapy is especially appropriate for this sort of patient who does continue to express doubts about himself and the future in the absence of any direct attempt at attitude change. Yet these patients would be rejected in a study of recovered depressed patients because of the presence of 'residual depression'. In other words it seems that on an intra-individual basis, depression is considered to be dimensional. That is mild depression following a depressive illness is regarded as a continuation of the 'disease'. This is illustrated by a case in which a young lady (34) was presented as a potential subject for study three. Unfortunately after her illness had receded her marital situation worsened. Although there was at that time no question over her diagnosis of recovery (she was back at work and had been functioning well for about seven months), she was understandably worried about her future and the outcome of these difficulties. She scored 16 on the BDI and was rejected as a recovered depressed patient suitable for the study. Such a patient may not have been rejected in study one because rightly or wrongly the Consultant would have regarded her as 'recovered from her illness' though still unhappy with life. Thus it can be seen that the patients in the two groups may be slightly different. Only those patients who demonstrated complete recovery with no residual depressed mood were considered suitable for this study. In addition the patients of study three were not seen when depressed, thus allowing for much wider selection. Therefore the question remains whether the patients in the two groups represent slightly different depressed populations

because of the different criteria used for selection.

In the brief discussion following the presentation of the results of study three, some consideration was given to this issue. It was pointed out that diurnal variation in the symptom intensity of some patients together with the efficacy of physical treatments of depression for certain patients are not adequately accommodated by cognitive theory. In addition there is some cause for concern over the early data used to develop the cognitive approach. For example, Loeb et al. (1964) investigated performance effects on depression and reported that depressed patients assigned to a high performance condition rated themselves as happier and more self-confident following the experimental manipulation than those in a low performance condition. However, the patients used in this study were undiagnosed (except for the absence of any organic problem) and patients were assigned to a 'high depressed' condition on the basis of a score of 16 or above on the BDI alone. The authors point out, however, that ". . . the process mediating the effects of relatively stable affective predispositions upon social judgement may well be different from those mediating the impact of more temporary moods . . ." (P. 615).

In a subsequent study Loeb et al. (1971) reported that in a group of depressed outpatients the experience of success significantly improved performance. Beck (1974) argues from such results that ". . . the finding that the depressed patient reacts positively to tangible evidence of successful or superior performance is most important . . . This tendency to exaggerate the evaluated aspects of situations and to overgeneralise in a

positive direction after success offers obvious clues to the therapeutic management of depression . . ." (P. 19).

It seems that the distinction between patients who are depressed and patients who have a depressive illness has been overlooked in later theoretical writings. Thus early investigations of the cognitive model do not necessarily provide the type of evidence required for a model of depressive illness. In contrast, recent work has concentrated on the important question of whether therapy based on the cognitive paradigm provides an effective treatment of depression. Here the concern that the treatment be applied to a specific group of patients rather than those who are just depressed has been met by applying careful selection criteria (Spitzer et al., 1978). The use of the unipolar - bipolar distinction is the one favoured so that recent studies (Rush et al., 1977) have only selected patients who satisfy the criteria for primary unipolar depression.

However, the use of such criteria does not prove that cognitive therapy is suitable for all depressions. Indeed one of the sub-categories (major endogenous unipolar depression) has a "lack of reactivity to environmental changes (once depressed doesn't feel better, even temporarily, when something good happens) as one of its signs. Consequently, observed changes in mood following success or failure manipulations would presumably mitigate against such a patient receiving this diagnosis. In other words, Beck's (1974) argument that patients can generalise from success experiences may say little except that such patients may not be good representatives of

the endogenous type depressions.

This issue is controversial and yet on theoretical grounds requires consideration. This is especially so if work with recovered depressed patients is to advance. It also raises the question of whether endogenous depressive types should be rejected from the cognitive model. At present the answer would seem to be no, but this may be because most of the theoretical work has (a) failed to specify the presence of depressive illness rather than depressive mood, (b) has tended to use depressed outpatients or worse depressed students, (c) has assumed that depressed mood and depressive illness are on a continuous dimension, (d) has assumed that people diagnosed as primarily depressed represent a reasonably homogenous group of patients, (e) has often used the BDI as a diagnostic scale. There is now sufficient understanding of depression to be clear that depression is a tremendously heterogeneous disorder and that many of the above assumptions and procedures are in error (Depue and Monroe, 1978, Kendell, 1975, 1976, Paykel and Rowan, 1979, Akiskal et al., 1978). Even biochemically depression presents itself in great diversity (Maas, 1975, Iversen, 1979). Therefore it seems reasonable that if selection criteria bias the sample chosen for study, this may be an unfair test of cognitive theory whose error is an overgeneralisation of its position rather than incorrect axioms for relevant cases. The only observation the author could make on this point is that in reading a large number of case notes and having seen a number of patients, some of whom were rejected, an impression is left that those patients who did take part in this study tended to come from those depressives

whose symptoms included early morning wakening, significant agitation or retardation, required hospitalisation when ill, who responded well to ECT and on the whole tended to be in the 40-60 age range. This impression cannot, of course, be regarded as anything more than that. These patients may well fit the endogenous type of patients described by Raskin and Cook (1976), thus highlighting again the possibility that these patients do not necessarily represent depressive illness in general. Nevertheless, as both Becker and Schuckit (1978) and Rush et al. (1978) point out "the job that remains is to determine who responds best to what" (P. 109). Moreover, if selection criteria do introduce bias into the type of recovered depressed patient selected, this may introduce serious difficulties in testing some of the premises of the cognitive model.

B. Comparison of Recovered Depressed Patients with Improved Anxious Patients

The discussion above has clear implications for the third study. Indeed the possibility that selection criteria can bias the selection of recovered depressed patients raises questions over the value of the comparisons made here. However, two points should be borne in mind, (a) the concerns voiced above are made with hindsight and were not immediately obvious at the beginning of the Study, (b) in cognitive theory as it stands at present, one recovered depressed group is as good as any other (excluding bipolar depressives). Thus, as a test of certain aspects of the cognitive theory of depression, this work remains valid.

The major findings of this study were that, in the absence of depressed mood in both groups but with the improved anxious group being more anxious that the depressed group, the improved anxious patients were significantly more external than the recovered depressed patients. What is interesting here is that in depressed patients, as demonstrated in Study one and in this Study, there is a tendency for lower depression scores to be associated with lower external scores in group data. However, for the improved anxious patients there is some indirect evidence that externality may be more stable trait which does not change as 'anxiety' recedes. This can be seen by considering the fact that the anxious group of Study one (I-E score M = 12.4, SD = 2.9) and the improved anxious group of Study three (I-E score M = 12.67, SD = 6.37) are very similar. On the other hand a 't' test reveals that the improved anxious group anxiety scores (M = 9.4, SD = 2.92) are significantly lower than those of the anxiety group used in Study one (M = 12.35, SD = 2.83). In addition, of course, the patients used in Study three were either discharged or receiving routine follow up consultations only. All were functioning reasonably well in the community. Thus there are reasonable grounds for arguing that the level of anxiety in the two control groups was different. By similar observations the BDI scores are lower for the improved anxious subjects of Study three, than for the anxious group of Study one (sig. $p < 0.01$). While the relation between externality and anxiety is not directly related to the aims of this Study, it is worth mentioning in passing that relatively speaking, externality appears a much more changeable measure in depressives than it does in

anxious patients. While the evidence is not clear on this issue, it is possible that there are at least two ways such data could be interpreted:

(a) for some individuals (depressives) changes in perceived controllability follows a change in mood. For other patients (e.g. anxiety prone) it constitutes a relatively stable trait that may act as a vulnerability factor. If patients are tested when ill, both groups may have high externality scores and it is only as patients recover that differences in perceived controllability emerge.

(b) perceived controllability is related more to neuroticism than any particular disorder. Those suffering from endogenous type disorders will show variable I-E scores while those suffering from the more neurotic type disorders will show a relatively stable tendency towards a negative view of their own perceived control.

Both these possibilities are untested. The matter is further complicated by the fact that some researchers have found the I-E scale an unsuitable measure of perceived controllability in certain situations (Miller and Seligman, 1973). Moreover, Rotter (1975) has suggested that locus of control may be significantly influenced by situational factors and has also raised the question of there being more than one type of 'external'. Consequently the issue of whether locus of control is a unidimensional or multi-dimensional construct is not yet decided. The extent to which this measure of locus of control confounds attributional processes (Weiner, 1972) adds to the difficulty of interpretation and the possibility that the I-E scale has a mood set built into it (Lamont, 1972) presents

still further problems. Consequently the observations noted above are of interest but interpretation can only be a matter of speculation in the present state of knowledge.

The failure to find significant differences on the other three cognitive measures (negative thinking, fear of failure and hopelessness) is also difficult to explain. It might be argued that the low level of psychopathology in both groups acted in opposition to differences emerging. However, this argument seems invalid in the light of the aims of the study and Beck's (1967, 1976) strong claim to offering an aetiological theory of depression. It must be remembered, for example, that in Beck's model the major differences that discriminate normal from pathological emotional states are cognitive variables. Although in later work, Beck and Kovacs (1978) acknowledge multi component processes, the role of the biological parameters of depression are largely ignored and considered unnecessary for understanding such a discrimination. Thus the question of psychological cognitive vulnerability which lies at the heart of the aetiological theory is of crucial importance. Beck (1976) presents a strong argument in favour of regarding such vulnerability as a direct result of there being a particular set of attitudes, expectations and attributions in the depression prone individual. While there is little doubt that such factors are important in depression, it is yet to be demonstrated that those variables postulated by Beck are more than just correlates of mood change and do indeed constitute important causal factors of depression. Nevertheless the negative findings reported here

cannot be taken as a refutation of the cognitive position. Indeed Beck (1967) himself suggests that cognitive vulnerability factors may not be discernible in recovered individuals. If Beck is correct, then the attitudes, expectations and attributions outlined by him are unstable cognitive components only becoming prominent in response to certain life events to which the individual has become 'sensitised'. While this would seem a reasonable position, it makes the axioms of the theory almost untestable at least in the absence of any qualifying additions to the theory. Indeed some writers (e.g. Brown and Harris, 1978) argue that Beck's model of depression needs to be supplemented with an understanding of the interaction between life events and cognitive processes. Furthermore, there is evidence which suggests that depressed patients view some life events as more stressful than controls (Schless et al., 1974), that depressed patients confronting life events are more likely to relapse (Paykel and Tanner, 1976) and that life events and the presence or absence of social support are important variables in psychological impairment (Andrews et al., 1978, Wartteit, 1979). Recently Rahe and Author (1978) have suggested that it is not only the interaction between attitudes, coping styles and life events that require consideration. They argue that illness (both functional and organic) requires an understanding of the interactions between the perception of a life situation, psychological defences, the psychophysiological response, response management, illness behaviour and illness measurement. They suggest that the activity at each

can attenuate or worsen the impact of specific life events. Their model seems especially relevant to depression and brings us back to the idea that it is not enough to understand functioning at one level but rather we need to study the interactions that make up the total response pattern of the individual. This would include the importance of feedback mechanisms between psychological and biological systems which is an element absent from cognitive theory.

It is against this background that the results of this study need to be set. If these results serve any function, it is to point out perhaps that a simple investigation of only cognitive variables is insufficient by itself to pinpoint predictive vulnerability factors. The idea that depression prone individuals are sensitised to certain events, at the very least, points out that these events are an important requirement of the cognitive model. It does, however, remain an open question as to how damaging these findings are to the cognitive theory of depression. As will be discussed in the final discussion, the efficacy of cognitive therapy does not imply a completely accurate theory.

Nevertheless, since negative thinking, fear of failure and hopelessness were far from reaching significance, this does suggest that the model is indeed too flexible to offer an explanation of depression (Blaney, 1975). We will need to sharpen up our concepts considerably and develop more specific instruments of measurement. The scales used here may simply be too crude to measure what are, in fact, very subtle differences. The answer does not lie in large samples for if the instruments used are insensitive we

will move no nearer to eliciting the crucial elements by simply increasing the number of patients seen. Indeed these results suggest that even with larger samples no differences would have emerged. It is also questionable as to how valuable significant differences are which require large samples to demonstrate them and which show a high degree of intra-group variability.

Though disappointing, these results and discussion serve the useful function of pointing out that (a) there is a need for greater specificity in what is to be measured, (b) work with recovered depressed patients is an essential component of any aetiological theory, (c) that the heterogeneity of depression may present problems for unicomponent models of depression, (d) when investigating recovered depressed patients, selection criteria may introduce an element of bias to the type of patient selected, (f) that the attitudes and cognitive variables presented as important for a model of depression may lack specificity when compared to other psychiatric groups.

IV. CONCLUSION

Depression is one of the oldest psychiatric disturbances known to man and is still one of the most common problems presenting for treatment. Its long history has reflected the changing philosophical outlooks adopted by investigating physicians. Yet in spite of its history, there remain many unresolved difficulties and confusions. Zilboorg and Henry (1941) believe that psychiatry tended to view man's mental state as capable of dissection. This gave the appearance of validity to explanations of depression which approached the disorder in terms of disturbances in the affective field or in the mood regulating areas of the brain. The advent and success of the biological treatments of

depression have perhaps reinforced such conceptualisations. On the other hand there have always been those who have opposed such demarcated views of psychopathology (e.g. A. Meyer, 1866-1950). The need to include cultural, sociological and psychological factors in any theory of depression that claims to be comprehensive has long been advocated both by the early psychobiologists and more recently by Akiskal and McKinney (1973, 1975). In a sense investigations of the role of life events in illness (e.g. Brown and Harris, 1978, Rahe and Author, 1978) would have found favour with the old psychobiologists. In addition, the influence of thinking, imagining, remembering and feeling have been shown to be interactive with mood states rather than just separate or consequent functions (Beck, 1967, 1976, Mandler, 1975, Lazarus, 1966). Thus the idea that thinking rather than biochemistry may be the cause of depression has gained both logical appeal and advocates. Unfortunately, though appealing, this psychological versus biological view also carries some subtle dualistic assumptions. This dualism reflects the two principal modalities of data collection. These are subjective reports and chemical/physical measurements which are often assumed to measure different phenomena, respectively learned and non-learned. The learned/non-learned distinction is clearly an important empirical question upon which the value of psychotherapy depends. But the unsupported implication that subjective experience depends on learned processes and psychical measurements on non-learned processes needs very cautious consideration. Unfortunately this difficulty has not been seriously considered by cognitive theorists. A genuinely interactive approach would hold that there are no subjective experiences without

underlying physiological processes and conversely that subjective experience is one attribute of some physiological process. The possibility that subjective experience and physiological responses can both be either learned or non-learned (or some combination) requires careful attention. A hint of this difficulty appears in the literature relating to the finding that some stimuli induce fear responses more easily than others, relatively independently of their actual potential harm (Seligman, 1971, Rachman, 1979). Moreover, Lacey (1967) demonstrated that there is significant inter-person variability in physiological responding to the same set of stimuli. It is outside the scope of this work to discuss these issues further. However, it should be emphasised that in a truly interactive analysis of depression the learned/non-learned and psychological/biological dimensions are potential sources of confusion and certainly not to be used interchangeably as cognitive theory tends to do.

A slightly different but related problem centres on the issue of symptom development. For Beck (1967) all symptoms of depression, even those considered to have a physiological basis, arise from the emergence and dominance of the negative cognitive triad within an individual's cognitive system. So bold a claim does not have much empirical support and it is difficult to imagine how this position could be tested. It must be remembered that Beck, unlike Seligman (1975), does not attempt a multi-component approach which allows for certain symptoms to arise from dysfunctional biological systems regardless of their primary cause. It does seem unreasonable to some, however, that sleep disturbance, constipation, retardation/agitation, loss of energy and appetite do not

require some clear understanding of internal mechanisms over and above those of cognition. More so since the effectiveness of physical treatments is well proved. For example, Paykel et al. (1975) investigating the effects of amitriptyline compared with psychotherapy found that early withdrawal of amitriptyline resulted in a marked return of symptoms. Symptoms were about one third less for patients on this drug during the maintenance period compared to psychotherapy and placebo. These authors argued that the symptoms showing the most consistent change with amitriptyline were guilt and anxiety and "other feelings of hopelessness . . . somatic complaints and insomnia" (P. 76). These patients also showed less fatigue and loss of energy. Interestingly the effects of the drug on mood "showed only weak effects". Such findings may suggest that amitriptyline had most influence on those symptoms having some physiological basis, with hopelessness decreasing as a result of increased energy and a consequent increase in feelings of being able to cope. It was also found that psychotherapy did not significantly affect symptoms but rather produced its main effects on social adjustment. Such a finding is confirmed by Weissman et al. (1974). It may be that it is symptom development rather than social deterioration that leads to hospitalisation (Tanner et al., 1975). However, some caution should be exercised in regarding symptoms and social adjustment as independent which seems an implication of the above work.

Where do such concerns leave the cognitive theory of depression? To answer this question it is necessary to consider the cognitive position from its most recent development which has been in therapy (Beck et al., 1978) rather than

theory. Rush et al. (1977) found cognitive therapy to be a superior treatment in a controlled comparative study with pharmacotherapy. Although this study has been criticised on selection and design grounds (Becker and Schuckit, 1978), there is little doubt that it is often referred to enthusiastically in the cognitive literature and has done much to advance the case for cognitive therapy. However, there is considerable danger in arguing backwards, as some do, by saying that the efficacy of the derived therapy proves the theory. Actually cognitive theorists are not alone in this sort of backward reasoning. The biochemical theories of depression are in part based on such reasoning. For example it may be that the future research will show that the monoamines, which constitute only a tiny percentage of neurochemical activity, are something of a side issue in depression and that some other hormone or neurochemical substance or interaction of substances yet to be discovered, constitutes the real biological substrate of depression. This is not to decry the value of theory but rather to underline the potential errors in backward reasoning. From the cognitive therapy perspective, in common with biochemical treatments, the treatments are not very specific. In the first place the cognitive therapy outlined by Beck et al. (1978) is more correctly described as cognitive-behavioural therapy Indeed Beck is more than happy to accept the eclectic nature of his therapy and readily accepts its heavy reliance on behavioural techniques. It is something of an open empirical question whether pure cognitive therapy devoid of behavioural procedures would prove very effective. Bandura's (1977, 1978) analysis of the mechanisms of behaviour and cognitive change would perhaps cast doubt on a positive outcome since he argues that "efficacy expectations" and

other cognitive variables are more readily changed by changing behaviour directly. This creates something of a problem for the theory concerning the primary and aetiological significance of cognition versus behaviour. Is it possible, for example, that some individuals do think negatively but continue to behave in an outgoing and coping manner and only if and when this behaviour begins to decline will they become ill? If this is so then learning theorists would indeed have cause to argue that behaviour is the pivot of change and therefore it is behaviour which requires analysis and the therapeutic thrust. Thus, as Blaney (1975) has argued, Beck's model lacks specificity. Many different cognitive and behavioural perspectives are easily accommodated within the the rapy whilst the theory stresses one dimension of change, that being cognition.

Secondly, as mentioned earlier, even if cognitive treatment does produce promising results this does not demonstrate the necessity or adequacy of the cognitive approach. For example, learning theory as an explanation of the development of fears and phobias is now under serious attack (Rachman, 1979). Nevertheless, there is good evidence to suggest that correctly applied treatments derived directly from learning theory can be very effective (Matthews, 1978, Marks, 1979).

Thirdly, there is no study which has demonstrated the specificity of attitudinal patterns in depression. Even the therapy for depression has many similarities to the cognitive-behavioural treatment of anxiety, which can include the daily recording and rational evaluation of "irrational thoughts" (Goldfried

and Goldfried, 1975, P. 101), sometimes thought to be specific to Beck's treatment of depression approach. In the work presented here there was also a failure to demonstrate any specific attitudinal or cognitive parameter able to discriminate improved anxious from recovered depressed patients. Although externality may be such a candidate, its interpretation is at present not sufficiently clear. It is also disappointing to report that in the history of the cognitive theory of emotional disorders there is not one study which has clearly demonstrated with the use of adequate controls good evidence for setting psychiatric patients apart on the basis of attitudes and cognitions alone, though this is a claim of cognitive theory (Beck, 1976). There are other aspects of the specificity problem which produce difficulties for cognitive theory. If cognitive and attitudinal factors are reasonably disorder specific, then presumably these disorders should be reasonably stable over time. In fact, Kendell (1974) found that 24.4% of patients diagnosed as having an anxiety state were at a subsequent admission diagnosed as having a depressive illness. The change in diagnosis in the opposite direction was only 2.39%. In fact Kendell's results show that in the case of an initial diagnosis of anxiety state, personality disorder and dementia, the most likely subsequent change in a diagnosis was to a depressive illness. Two things stand out here. Firstly, the relatively high instability of certain disorders and secondly the dominance of a depressive illness as a new diagnosis rather than changes in diagnosis being equally likely for each diagnostic category. Certainly, as Kendell (op. cit.) points out, the change in diagnosis of depression $\longrightarrow$ anxiety compared to the anxiety $\longrightarrow$ depression are strikingly dissimilar. It is not the concern here to discuss why this should

be but rather to consider what such results mean for cognitive theory.

Some cognitive theorists might argue that there is no reason why one individual cannot have more than one "set" of negative attitudes and thus be vulnerable to more than one disorder. This, on the surface, seems reasonable but this suggests that different cognitive schemas become dominant at different times - How many negative schemas do people have? Is there a "healthy one", a "depressed one" and an "anxious one" in some individuals - if so were they developed at different stages of life or at the same time? Why is it that people tend to move from a diagnosis of anxiety to depression but hardly ever the other way round? The implication seems to be that some disorders are not nearly so neatly packaged with their attitudinal and cognitive styles as Beck (1976) suggests. In fact, there seems to be a good deal of overlap and a lack of stability between some disorders. The view that each disorder has a clearly identifiable cognitive style has not only not been demonstrated but is highly misleading.

Having discussed the problems of the cognitive specificity of various disorders, it is important to recall an earlier discussion on specificity of terms. In the early part of this chapter it was argued that the term "negative" has been used too loosely and has been used to describe both the presentation of something aversive and the withdrawal of something positive. The results of study one demonstrated that depressed patients were not hypersensitive to negative events (e.g. failure) compared to two control groups, although loose use of cognitive terminology has certainly suggested this to be an important

factor in depression (De Monbreum and Craighead, 1977). In fact it was found that rather than a hypersensitivity to negative events there was a hypo-sensitivity to positive events. The importance and evidence for making this distinction has already been discussed. However, further cause for separating the potential two meanings of "negative" comes from more recent evidence (Mischel, 1979). Mischel argues that while it is possible that depressed persons do have an unrealistically negative view of self, it is also possible that this view reflects a realistic observation of a lack of competence. Mischel correctly points out that a decision about whether self perceptions are distortions or not requires more than just subjective observation. Rather a comparison between self rating and observer rating is needed. According to Mischel such work has recently been carried out in collaboration with Lewinsohn et al. (1980). Mischel reports that although the depressed subjects rated themselves as less competent than non-depressed controls, they were initially more realistic in their self perceptions than were controls. Mischel (1979) discussed the findings:

> ". . . specifically, the controls perceived themselves more positively than others saw them, whereas the depressed saw themselves as they were seen. Non depressed people may thus be characterised as having a halo, or illusory glow, that involves an unrealistic self-enhancement in which one sees oneself more positively and less negatively than others see one. Clearly if social reality is defined by the extent of agreement with objective observers, the depressed were the most realistic in their self-perceptions, and the controls were engaged in self-enhancing distortions" (P. 752).

This interesting twist as to who is actually distorted in their thinking processes, normals or depressed subjects, cannot be fully appraised in the absence of the published study. Clearly, as has been mentioned before, it is yet to be demonstrated whether such findings have any relevancc to depressive illness or are simply correlates of mild low mood. Nevertheless, such work does point to the importance of being precise in the concepts used. In other words, terms like "distortion" or "negative", need to be carefully defined. For example, we need to know whether depressed individuals see themselves as having bad characteristics or rather an absence of positive ones. In the same vein the concept of hopelessness does not necessarily imply a perception of the future as full of obstacles and changes, but may be one where nothing happens; it is empty, devoid of any significant positive or enjoyable event.

The discussion so far has taken a critical look at the cognitive theory as an explanation of depressive illness. At the heart of this criticism has been the fact that depression is too heterogenous a disorder to allow for such narrow theorising. But while the model can be attacked from this perspective, the realisation that cognitive factors play a major role in emotional disturbances, psychosomatics, and possibly organic illnesses is gaining a considerable number of advocates. For example, according to Bowers and Kelly (1979) the ability of the immune T-cell and B-cell systems may be affected by the presence of corticosteroids. These in turn are stress responsive but are greatly influenced by perceived coping ability and various psychological

defences (Mason, 1975). That is, as Bowers and Kelly (1979) point out:

> " . . . when a situation is perceived as threatening, and/or when psychological defences are unable to contain the emotional reactions to the perceived stress, the person's biological defences may be diminished as well, making him/her potentially more vulnerable to infectious diseases as well as cancer" (P. 493).

At present such work is in its infancy and it remains to be seen whether such speculations prove warranted or mere over-enthusiasm. At any rate the study of the relationship between stress hormones, immune systems and various diseases is but one example of the great potential of interactive studies. Cognitive theory has an important part to play in this work. Beck's (1976) reports of the effects of automatic thoughts and Meichenbaum's (1977) description of self talk in terms of Luria's model of learning, constitute major advances. Such work and ideas have certainly re-directed therapeutic attention to those self-evaluative mechanisms which patients may be unaware of. To demonstrate interactions between such internal evaluative mechanisms, coping and defending, physiological change and illness, is the challenge of the future. For each group the role is to develop precision of concepts and measurement. So for example cognitive theorists will need to discriminate between (say) a perception of the withdrawal of positive elements in one's domain and the introduction of aversive elements or dangers. They may well have different physiological correlates. Also we can ask to what extent some individuals have particular hormone profiles which predispose them to respond

'cognitively' in one way rather than another to certain events or stimuli? We know enough of drug activity to be able to say that changes in physiological functioning can produce changes in outlook, cognitive and attitudinal responses.

The point is that it is to systems of interaction in depression that attention should be turned in model building. On the other hand, it is not necessary to provide a comprehensive model of depression explaining aetiology, course and outcome, for cognitive based ideas and therapies to prove of immense value. Indeed the idea that maladaptive ideas and beliefs can produce and sustain maladaptive behaviour and affective states is well conceded. As Bandura (1977) argues, the pursuit of unrealistic goals and standards can be a potent source of stress when the individual fails again and again.

> "Some people whose accomplishments bring them a sense of failure resort to alcoholic self-anaesthetisation; others escape into grandiose ideation where they achieve in fantasy what is unattainable in reality; many renounce pursuits having self-evaluative implications and gravitate to groups embracing anti-achievement norms; others protect themselves from self condemnation for their self alleged faults by imputing persecutory schemes; and tragically still others are driven by relentless self disparagement to suicide" (P. 141-142).

Thus it seems reasonable to conclude that cognitive factors are immensely powerful variables within the matrix of systems governing health and illness. On the other hand, it is illuminating to recall that life events were also thought

to hold great promise as illness producing variables. Unfortunately, though important, it is now realised that the life events most often investigated (Rahe and Holmes, 1967), have a rather low correlation with illness. In fact, the vast majority of people experiencing life events do not become physically or psychiatrically ill. We have yet to discover whether the same is true of cognitive or behaviour variables. We do not yet know what percentage of those individuals with poor social skills, negative evaluative attitudes and a pessimistic view of the future are, or become, 'ill'. This has not been investigated although study 2 was a move towards such an investigation. If it is possible for people to hold such attitudes without becoming ill, then one must go beyond just thinking to understand depressive illness.

In conclusion, it can be suggested that this thesis has produced evidence in support of the following tentative possibilities.

(1) It is perhaps better to consider depressed patients as being hypo-sensitive to positive events rather than hypersensitive to negative events.

(2) Depressed and anxious patients are more difficult to separate on attitudinal and cognitive measures than is implied by the theory. This may reflect an insensitivity in the measuring instruments used or a significant degree of overlap in the cognitive variables affecting these two disorders. This may invalidate the idea that some disorders have specific identifiable cognitive styles which can discriminate one disorder from another and which provides an aetiological basis.

(3) Recovered depressed and improved anxious patients also proved difficult to separate on cognitive and attitudinal measures. In addition to the consideration given in (2) this may be due to the low psychopathology of both groups or the fact that the negative schemas are not discernible in people who are well. If the latter is the case then the aetiological part of the cognitive theory of depression is extremely difficult to test.

(4) Work with recovered depressed individuals must play an important part in cognitive research. At the same time, however, there may be some conflict and sampling bias introduced into the research by stringent selection criteria. The author sees this as a potentially major problem in working with recovered patients. This difficulty seems especially troublesome with the use of mood self-rating scales, e.g. the BDI.

(5) The review chapters and recent discussion point out the importance for future 'models' of depression to attempt to be interactive and multi-component in their approach. Nevertheless therapy based on the cognitive paradigm can be of immense value even though the theory may still need considerable development and refinement.

We are beginning to elucidate the components of depression. The task of fitting all our pieces of data together remains the most exciting challenge for the future.

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APPENDIX A

MEASURES

A. ATTITUDINAL AND COGNITIVE MEASURES

1. Internal-External Scale (I-E)

(Please circle either "a" or "b" in each numbered pair as the statement which you accept more strongly. Please answer all questions.)

1. a. Children get into trouble because their parents punish them too much
   b. The trouble with most children nowadays is that their parents are too easy with them.
2. a. Many of the unhappy things in people's lives are partly due to bad luck
   b. People's misfortunes result from the mistakes they make.
3. a. One of the major reasons why we have war is because people don't take enough interest in politics.
   b. There will always be wars, no matter how hard people try to prevent them.
4. a. In the long run people get the respect they deserve in this world
   b. Unfortunately, an individual's worth often passes unrecognised no matter how hard he tries.
5. a. The idea that teachers are unfair to students is nonsense
   b. Most students don't realise the extent to which their marks are influenced by accidental happenings.
6. a. Without the right luck one cannot be an effective leader
   b. Capable people who fail to become leaders have not taken advantage of their opportunities.
7. a. No matter how hard you try some people just don't like you
   b. People who can't get others to like them don't understand how to get along with others.
8. a. Heredity plays the major role in determining one's personality

b. It is one's experiences in life which determine what they're like.

9. a. I have often found that what is going to happen will happen.

b. Trusting to fate has never turned out as well for me as making a decision to take a definite course of action.

10. a. In the case of the well prepared student there is rarely if ever such a thing as an unfair exam.

b. Many times exam questions tend to be so unrelated to course work that studying is really useless.

11. a. Becoming a success is a matter of hard work, luck has little or nothing to do with it.

b. Getting a good job depends mainly on being in the right place at the right time.

12. a. The man in the street can have an influence in government decisions.

b. This world is run by the few people in power, and there is not much the man in the street can do about it.

13. a. When I make plans, I am almost certain that I can make them work

b. It is not always wise to plan too far ahead because many things turn out to be a matter of good or bad fortune anyhow.

14. a. There are certain people who are just no good

b. There is some good in everybody.

15. a. In my case getting what I want has little or nothing to do with luck

b. Many times we might just as well decide what to do by flipping a coin.

16. a. Who gets to be the boss often depends on who was lucky enough to be in the right place first

b. Getting people to do the right thing depends upon ability, luck has little or nothing to do with it.

17. a. As far as world affairs are concerned, most of us are the victims of forces we can neither understand, nor control

    b. By taking an active part in political and social affairs the people can control world events.

18. a. Most people don't realise the extent to which their lives are controlled by accidental happenings

    b. There really is no such thing as "luck".

19. a. One should always be willing to admit mistakes

    b. It is usually best to cover up one's mistakes.

20. a. It is hard to know whether or not a person really likes you

    b. How many friends you have depends upon how nice a person you are.

21. a. In the long run the bad things that happen to us are balanced by the good ones

    b. Most misfortunes are the result of lack of ability, ignorance, laziness or all three.

22. a. With enough effort we can wipe out political corruption

    b. It is difficult for people to have much control over the things politicians do in office

23. a. Sometimes I can't understand how teachers arrive at the marks they giv

    b. There is a direct connection between how hard I study and the marks I get.

24. a. A good leader expects people to decide for themselves what they should

    b. A good leader makes it clear everybody knows what their jobs are.

25. a. Many times I feel I have little influence over the things that happen to m

    b. It is impossible for me to believe that chance or luck plays an important role in my life.

26. a. People are lonely because they don't try to be friendly

    b. There's not much use in trying too hard to please people, if they like you, they like you.

27. a. There is too much emphasis on athletics in secondary school

    b. Team sports are an excellent way to build character.

28. a. What happens to me is my own doing

    b. Sometimes I feel that I don't have enough control over the direction my life is taking

29. a. Most of the time I can't understand why politicians behave the way they c

    b. In the long run the people are responsible for bad government on a national as well as on a local level.

2. Test Anxiety Scale

(Only the second adapted version of this scale was used in this study.)

(a) Sarason's (1972) Test Anxiety Scale

1. While taking an important exam I find myself thinking of how much brighter the other students are than I am
2. If I were to take an intelligence test, I would worry a great deal before taking it
3. If I knew I was going to take an intelligence test, I would feel confident and relaxed beforehand.
4. While taking an important examination I perspire a great deal.
5. During course examinations I find myself thinking of things unrelated the actual course material
6. I get to feel very panicky when I have to take a surprise exam
7. During tests I find myself thinking of the consequences of failing
8. After important tests I am frequently so tense that my stomach gets upset.
9. I freeze up on things like intelligence tests and final exams
10. Getting a good grade on one test doesn't seem to increase my confidence on the second
11. I sometimes feel my heart beating very fast during important tests.
12. After taking a test I always feel I could have done better than I actually did
13. I usually get depressed after taking a test
14. I have an uneasy, upset feeling before taking a final examination
15. When taking a test my emotional feelings do not interfere with my performance.

16. During a course examination I frequently get so nervous that I forget facts I really know
17. I seem to defeat myself while working on important tests
18. The harder I work at taking a test or studying for one, the more confused I get
19. As soon as an exam is over I try to stop worrying about it, but I just can't
20. During exams I sometimes wonder if I'll ever get through college
21. I would rather write a paper than take an examination for my grade in a course
22. I wish examinations did not bother me so much
23. I think I could do much better on tests if I could take them alone and not feel pressured by a time limit
24. Thinking about the grade I may get in a course interferes with my studying and my performance on tests
25. If examinations could be done away with I think I would actually learn more
26. On exams I take the attitude, "If I don't know it now there's no point worrying about it".
27. I really don't see why some people get so upset about tests.
28. Thoughts of doing poorly interfere with my performance on tests
29. I don't study any harder for final exams than for the rest of my course work
30. Even when I'm well prepared for a test, I feel very anxious about it.
31. I don't enjoy eating before an important test

32. Before an important examination I find my hands or arms trembling

33. I seldom feel the need for "cramming" before an exam

34. The University ought to recognise that some students are more nervous than others about tests and that this affects their performa

35. It seems to me that examination periods ought not to be made the tense situations which they are

36. I start feeling very uneasy just before getting a test paper back

37. I dread courses where the professor has the habit of giving "pop" quizzes.

(b) Adapted Test Anxiety Scale

1. In situations where I have to take a test with other people, I find myself thinking of how much brighter the other people are than I am True/Fals
2. If I were to take an intelligence test, I would worry a great deal before taking it True/Fals
3. If I knew I was going to be asked to take an intelligence test, I would feel confident and relaxed beforehand True/Fals
4. While performing an important test I tend to perspire a great deal True/Fals
5. During important tests I find myself thinking of things unrelated to the actual test material True/Fals
6. I feel panicky when I am asked to take a surprise test True/Fals
7. During important tests I find myself thinking of the consequences of failing True/Fals
8. After important tests I am frequently so tense that my stomach gets upset True/Fals
9. I freeze up under conditions of I.Q. testing and assessments True/Fals
10. Doing well on one test or assessment does not seem to increase my confidence on other tests and assessments True/Fals
11. I sometimes feel my heart beating very fast during important tests True/Fals
12. After taking a test I always feel I could have done better than I actually did True/Fals

13.	I usually get depressed after taking a test	True/False
14.	I have an uneasy, upset feeling before taking an important test	True/False
15.	During important assessment situations I frequently get so nervous that my memory lets me down	True/False
16.	When taking a test, my emotional feelings do not interfere with my performance	True/False
17.	I seem to defeat myself while working on important tests	True/False
18.	The harder I try on important tests the more confused I tend to become	True/False
19.	As soon as an important test or assessment is over I try to stop worrying about it, but I just can't	True/False
20.	During important tests and in assessment situations I sometimes wonder if I am performing adequately	True/False
21.	I would rather show my abilities in other ways to that of taking written tests	True/False
22.	I wish important tests did not bother me so much	True/False
23.	I think I could do much better on tests if I could take them alone and not feel pressured by a time limit	True/False
24.	Thinking about how well or badly I will do in assessment situations interferes with my performance	True/False
25.	If tests could be done away with I think I could more easily show my ability	True/False
26.	On important tests I take the attitude, "If I don't know the answer there is no point in worrying about it".	True/False

27. I really don't see why some people get so upset about tests True/False

28. Thoughts of doing poorly on important tests and assessments interfere with my performance True/False

29. I don't go out of my way to study before important tests True/False

30. Even when I am prepared for an important test, I still feel very anxious about it True/False

31. I don't enjoy eating before an important test True/False

32. Before an important test I find my hands trembling True/False

33. I am confident of my ability before taking an important test or assessment True/False

34. Establishments like the hospital ought to recognise that some people are more nervous than others about tests and that this affects their performance True/False

35. It seems to me that test conditions ought not to be made the tense situations which they are True/False

36. I start feeling very uneasy just before I find out how well or poorly I have done on an important test or assessment True/False

37. I dread occasions where I know it is likely that I may be asked to take a test or be put in an assessment situation with very little warning True/False

3. Beck Story Conpletion Test

Directions: Read the following short paragraphs and try to envision yourself as the main character in each. If you have never been in the precise situation, nonetheless try to imagine yourself in it. At the end of each of the paragraphs you are to complete the story by circling one number from each of the four groups of sentences. The completions are concerned with such things as the outcome of the story and your perceptions. If you have completed these paragraphs previously, remembe that there is no need to complete them the same way each time. It is also not necessary to make the four responses consistent with one another. There are, of course, no right or wrong answers; we are only interested in the different ways people view situations. Please answer all questions and work rapidly.

Paragraph: In our school athletics teams, a big event was always the fencing matches. Once I was on the team in the championships. This was to be my first time fencing in a team championships. We practised especially hard before the event because we really wanted to win.

Group A

1. I was confident that I'd perform well
2. It would have surprised me if we hadn't won; we were all too good
3. I knew beforehand that our team was relatively weak
4. I had a sinking feeling that I would fail the team.

Group B

1. We were decisively beaten
2. The team won the championships
3. The team lost to another school
4. For the third year in succession, we won.

Group C

1. The other team-members went out of their way to give me encouragement and practice before my first big match
2. Some of my team-mates were hesitant to help me practise for the event
3. The other members wished me good luck before the match
4. I didn't get along with the other members in the team and practice was a pretty lonely process.

Group D

1. I was not one of the top members of our team
2. My poor performance convinced me to give up fencing
3. I was an outstanding fencer
4. I was proud to be a member of the winning team.

Paragraph: About 2 or 3 years ago I wanted desperately to see my brother again after 2 years' separation. I worked hard at my job in order to earn the money to make the trip to London to meet him. In the final week I rarely had time to do anything else.

Group A

1. I felt hopeful that I would earn enough money to make my plans possible
2. Nothing could have kept me from accomplishing my plans

3. I doubted that my plans would go as smoothly as possible

4. I was certain that my plans would fall through

Group B

1. Just my luck that on the big day there was an emergency at work which prevented the trip

2. I met my brother in London and we spent a few days together

3. Something came up before the trip, so it had to be postponed for a while

4. The reunion was extremely pleasurable for both of us.

Group C

1. The boss offered me a loan to help me out

2. The boss worked me pretty hard even though I had a lot on my mind

3. The boss let me work a little overtime to earn more money

4. At the last minute the boss said if I left town I might lose my job.

Group D

1. I was rather inept in my handling of the trip

2. It was probably my fault that things worked out badly

3. I handled the trip with great skill and forethought

4. My handling of the trip was competent.

Paragraph: I remember the time I was about 10 years old and all the kids in the neighbourhood had planned for weeks for a party to be held in my back yard. Everyone was terribly excited - the night before I went to bed early.

Group A

1. I looked forward to the next day with confidence

2. I was absolutely confident that I would perform my duties well the next day

3. I was fearful that the next day I would forget something
4. All night I tossed and turned - I had an uncomfortable foreboding about the next day

Group B

1. The next day it rained - all our plans failed
2. Everything went fairly well, except for the normal minor mishaps
3. Unfortunately all the party events did not come off as we'd hoped
4. The next day the party was a great success.

Group C

1. The other kids praised me for a superlative job
2. The other kids didn't think I was all that good
3. We all worked well together - the other kids assured me I did okay
4. A few of the kids seemed to feel that I was being obnoxious, bossy and incompetent

Group D

1. I was really stupid and forgot to bring the balloons we needed
2. What an utterly ridiculous performance I gave
3. I was quite pleased at how I performed
4. I guess I did pretty well in my part.

Paragraph: One night I went to a party with my roommate and I saw an attractive girl/boy across the room. I thought I would like to get to know her/him but since I was leaving town in a few days, I didn't have much time. I approached her/him and started talking to her/him.

Group A

1. I expected she'd/he'd enjoy talking to me
2. I had thought she/he would probably accept a date
3. I was apprehensive beforehand that she/he would find me uninteresting
4. I was really fearful that she/he might put me down.

Group B

1. Soon after we met she/he left the party for a date
2. After a short talk she/he accepted a date with me for the next night
3. Soon she/he started talking to someone else
4. I wound up talking to her/him for quite a while, and it was the start of a wonderful relationship.

Group C

1. My roommate knew the girl/boy from before and helped me to get introduced to her/him
2. My roommate suggested that I wasn't her/his type
3. My roommate encouraged me to go up and talk to her/him
4. My roommate commented that I didn't have much of a chance with her/him.

Group D

1. I'm not extremely adept socially with girls/boys
2. I guess I didn't have a chance with her/him
3. I handle myself quite well with girls/boys
4. It's good I'm at ease with girls/boys

Paragraph: For spring vacation this year some friends and I are planning a trip to Cornwall. This will be my only real break this year.

Group A

1. I expect the trip will work out well
2. I'm sure the trip will be really fantastic
3. No doubt the trip will have something go wrong with it
4. I expect the trip will be a flop

Group B

1. I'm sure I'll look back on the trip as a bad experience
2. I'm sure I'll look back on the trip as a pretty good thing
3. I'm sure I'll look back on the trip as not that good an experience
4. I'm sure I'll look back on the trip as a great experience.

Group C

1. My friends will give me a lot of help in planning and packing
2. My friends will probably not be of much assistance
3. My friends will chip in and help with the plans
4. My friends will probably not help me with the trip at all.

Group D

1. I don't really deserve this trip
2. I'm the last person in the world to deserve a good trip
3. I deserve a good trip every now and then
4. I guess I have the trip coming to me.

Paragraph: One birthday when I was a child I gave a party and invited my friends to my house to play games and eat lost of cake and ice-cream. This wa to be a gala affair, so I couldn't think of anything else for a week.

Group A

1. I expected everything to go fairly well
2. I expected the party to be a great success
3. I doubted everything would be as good as I hoped
4. I sort of expected that the party would flop.

Group B

1. The party was actually a pretty miserable affair
2. The party was fun after all
3. The party didn't quite come off too well
4. The party was the high point of the year

Group C

1. My guests were extremely pleased with me and my plans
2. The guests seemed pretty uninterested in me and the party activities
3. The guests acted as if they liked the activities and liked being with me
4. The guests were totally uninterested in the activities and in me.

Group D

1. I felt unsuccessful during the party
2. I sure felt like a flop at that party
3. I felt very satisfied and successful at my birthday party
4. I felt reasonably satisfied and successful at the party

Paragraph: Four years ago I took part in an important speech competition. My school English teacher had nominated me, so I was eager to do a good job. My family was also looking forward to my performance.

Group A

1. I knew I could do a pretty good job with my speech
2. I was pretty sure I could win the first prize.

3. I felt beforehand that I wasn't going to perform too well

4. I really expected to perform pretty poorly.

Group B

1. I did a terrible job with my speech

2. I did a good job with my speech and won an award

3. My performance was pretty mediocre.

4. I walked away with the statewide championship.

Group C

1. Before the competition my teacher coached me every night for a week

2. My teacher was too busy to give me much advice beforehand

3. My teacher helped me by reading my speech over a few times

4. My teacher gave me no help at all in preparation for the competition

Group D

1. I felt pretty nervous and inept during the competition

2. I felt that I was doing a terrible job with my speech and that it was my fault

3. I felt very confident giving the speech

4. I felt that I was doing a decent job during the speech.

Paragraph: Recently I applied to get on to a limited course at my local college. Because it was a popular course, all applicants were required to be interviewed and to submit a written application. I knew one of the sectionmen in the course.

Group A

1. I felt I had a good chance of getting in

2. I was confident that I would be admitted into the course

3. I felt my chances of entry were slim.

4. I felt that I had no real chance of getting in.

Group B

1. I found out soon after the interview that I did not get into the course
2. After some delay I was told that I was in the course
3. I didn't get into the course, but the instructor put my name on the list for next year
4. During the interview the instructor said I was definitely in the course.

Group C

1. The section-man I knew previously put in a good word for me with the instructor
2. This section-man gave me no advice or support
3. This section-man encouraged me to apply
4. This section-man suggested that I need not bother to apply.

Group D

1. During the interview I felt I wasn't coming across too well.
2. I felt that my handling of the interview was quite poor.
3. During the interview I was confident and felt extremely articulate
4. During the interview I felt pretty successful.

Paragraph: Later this year I hope to be able to purchase a car if things go well. I've never bought or owned a car before, but by that time I should be able to afford an inexpensive model.

Group A

1. I expect my plans to buy a car will turn out okay.
2. I expect my plans will work out extremely well.
3. I have a feeling something will go wrong with my plans to buy the car
4. I expect my plans will fail - maybe I won't have enough money after all.

Group B

1. I will look back at the purchase plans as a total mistake
2. I will look back at the plans as moderately successful
3. I will look back at the plans as less than totally satisfactory
4. I will look back at the plans as totally satisfactory.

Group C

1. The car dealer will probably be of invaluable assistance to me
2. I suppose the dealer will give me very little help
3. The dealer will probably be helpful in such a purchase
4. I expect that the dealer will be of no help to me whatsoever.

Group D

1. I don't really merit having my own car
2. There's no way I deserve to get a personal car
3. I'm entitled to own a car of my own
4. I suppose I've earned that car.

Paragraph: Last summer I did some research work in an office in my own town The job was totally new to me, and I was to work closely with some knowledgeab people in the field.

Group A

1. I looked forward to the job as a good job
2. I expected the job to be highly challenging and exciting
3. Secretly I foresaw a dull and uninteresting job for the summer
4. I expected the job to be a complete waste for me.

Group B

1. The job turned out to be dull and stifling for me
2. My job actually was interesting, if not inspiring
3. By and large my work was routine and uneventful
4. The research was fascinating, valuable work.

Group C

1. My co-workers really liked me and admired my work
2. I did not get close to my co-workers, and they didn't really use my work
3. The other people in the office seemed by and large to accept my work
4. My co-workers were generally critical and disapproving of my research efforts.

Group D

1. I can't say that my research efforts were in any way novel or important
2. I did not work I considered good that summer
3. I feel that I make an outstanding contribution to the research
4. I did a competent job.

B. GENERAL AND BACKGROUND MEASURES

1. Beck Depression Inventory

Here are some statements regarding the way people feel or think. The statements are grouped in 21 sections from A to U. One statement must be chosen from each section. You are requested to put a circle round the number of the statement which fits you best at the moment.

A. 0 I do not feel sad

1 I feel blue or sad

2a I am blue or sad all the time and I can't snap out of it

2b I am so sad or unhappy that it is very painful

3 I am so sad or unhappy that I can't stand it.

B. 0 I am not particularly pessimistic or discouraged about the future

1a I feel discouraged about the future

2a I feel I have nothing to look forward to

2b I feel that I won't ever get over my troubles

3 I feel that the future is hopeless and that things cannot improve.

C. 0 I do not feel like a failure

1 I feel that I have failed more than the average

2a As I look back on my life all I can see is a lot of failures

3 I feel I am a complete failure as a person (parent, husband, wife)

D. 0 I am not particularly dissatisfied

1a I feel bored most of the time

1b I don't enjoy things the way I used to

2 I don't get satisfaction out of anything any more

3 I am dissatisfied with everything.

E 0 I don't feel particularly guilty

1 I feel bad or unworthy a good part of the time

2a I feel quite guilty

2b I feel bad or unworthy practically all the time now

3 I feel as though I am very bad or worthless.

F 0 I don't feel I am being punished

1 I have a feeling that something bad may happen to me

2 I feel I am being punished or will be punished

3a I feel I deserve to be punished

3b I want to be punished.

G 0 I don't feel disappointed in myself

1a I am disappointed in myself

1b I don't like myself

2 I am disgusted with myself

3 I hate myself

H 0 I don't feel that I am worse than anybody else

1 I am very critical of myself for my weaknesses, or mistakes

2a I blame myself for everything that goes wrong

2b I feel I have many bad faults

I 0 I don't have any thoughts of harming myself

1 I have thoughts of harming myself but I would not carry them out

2a I feel I would be better off dead

2b I have definite plans about committing suicide

2c I feel my family would be better off if I were dead

3 I would kill myself if I could

J 0 I don't cry any more than usual

1 I cry more now than I used to

2 I cry all the time now. I can't stop it.

3 I used to be able to cry but now I can't cry at all even though I want to

K 0 I am no more irritated now than I ever am

1 I get annoyed or irritated more easily than I used to

2 I feel irritated all the time

3 I don't get irritated at all at the things that used to irritate me

L 0 I have not lost interest in other people

1 I am less interested in other people now than I used to be

2 I have lost most of my interest in other people and have little feeling for them

3 I have lost all my interest in other people and don't care about them at all

M 0 I make decisions about as well as ever

1 I am less sure of myself now and try to put off making decisions

2 I can't make decisions any more without help

3 I can't make decisions at all any more

N 0 I don't feel any worse than I used to

1 I am worried that I am looking old or unattractive

2 I feel that there are permanent changes in my appearance and they make me look unattractive

3 I feel that I am ugly or repulsive looking

O 0 I can work about as well as before

1a It takes extra effort to get started at doing something

1b I don't work as well as I used to

2 I have to push myself very hard to do anything

3 I can't do any work at all

P 0 I can sleep as well as usual

1 I wake up more tired in the morning than I used to

2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep

3 I wake up early every day and can't get more than 5 hours' sleep

Q 0 I don't get any more tired than usual

1 I get tired more easily than I used to

2 I get tired from doing anything

3 I get too tired to do anything

R 0 My appetite is no worse than usual

1 My appetite is not as good as it used to be

2 My appetite is much worse now

3 I have no appetite at all any more

S 0 I haven't lost much weight, if any, lately

1 I have lost more than 5 pounds

2 I have lost more than 10 pounds

3 I have lost more than 15 pounds

T 0 I am no more concerned about my health than usual

1 I am concerned about aches and pains or upset stomach or constipation or other unpleasant feelings in my body

2 I am so concerned with how I feel or what I feel that it's hard to think of much else

3 I am completely absorbed in what I feel

U 0 I have not noticed any recent change in my interest in sex

1 I am less interested in sex than I used to be

2 I am much less interested in sex now

3 I have lost interest in sex completely

2. General Anxiety Scale

The following statements describe how people may feel in general situations. Read each statement carefully. If the statement describes how you feel, circle the word true (i.e. (true), at the side of the questionnaire. If it does not describe how you feel, circle the word false (i.e. (false) at the side of the questionnaire. There are no right or wrong answers, and there is no time limit so you may proceed at your own pace.

1.	I freeze up in a competitive situation	True/Fals
2.	I worry about my mental health more than most people do	True/Fals
3.	I worry about my social adjustment more than most people do	True/Fals
4.	I am a highly strung person	True/Fals
5.	I wish I could be as happy as other people appear to be	True/Fals
6.	I don't seem to be able to control worrying about something even when I know there is no basis for it	True/Fals
7.	I practically never blush	True/Fals
8.	When I have to talk to a group I get very anxious	True/Fals
9.	I am usually calm and not easily upset	True/Fals
10.	I perspire a lot when I am with a group of strangers	True/Fals
11.	I sometime s get so excited that I find it hard to get to sleep	True/Fals
12.	When I go to the doctor I worry that he will tell me something is wrong with me	True/Fals
13.	I am inclined to take things hard	True/Fals
14.	I have had periods in which I have lost sleep over worry	True/Fals

15. I have been afraid of things or people that I know could not hurt me True/False

16. I am easily embarrassed True/False

17. I have periods of such restlessness that I cannot sit long in a chair True/False

Success-Failure Inventory

Items in Success-Failure Inventory (SFI) showing keys for success attainment (S) and failure avoidance (FA)

	Keying	
	True	False
I have a tendency to give up easily when I meet difficult problems	FA	S
I like to try out new ideas even if they turn out later to have been a total waste of time	S	FA
I am ambitious	S	FA
It's better to stick by what you have than to try new things you don't really know about	FA	S
Failure is not a disgrace when one has tried his best		FA
It is better to be an observer than a participant because one learns more and gets into less trouble	FA	S
Success is too short lived for a person to sacrifice much to attain it		S
One of my primary or major aims in life is to accomplish something that would make people proud of me	S	
Any man who is able and willing to work hard has a good chance of succeeding		FA
I would rather remain free from commitments to others than risk serious disappointment or failure later	FA	
When I am in a group or organisation, I like to be appointed or selected for office	S	FA

	True	False
I have a very strong desire to be a success in the world	S	
I like to follow routines and avoid risks	FA	S
Great ambitiousness usually brings great accomplishments	S	
I don't like to work on a problem unless there is the possibility of coming out with a clear cut and unambiguous answer		S
I dislike failure so much that I abstain from participating in competitive situations	FA	S
I sometimes keep on at a thing until others lose patience with me	S	
I used to like it very much when one of my essays was read to the class in school	S	FA
I keep out of trouble at all costs	FA	
I enjoy competitive situations	S	FA
It is better never to expect too much; in that way you are rarely disappointed	FA	S
I like to avoid responsibilities and obligations	FA	S

Hopelessness Scale

Directions

Here are some statements about the way you see the future. Read each statement carefully. If the statement describes how you think about the future, circle the word true at the side of the questionnaire. If it does not describe how you think about the future, circle the word false at the side of the questionnaire.

1.	I look forward to the future with hope and enthusiasm	True	False
2.	I might as well give up because I can't make things better for myself	True	False
3.	When things are going badly I am helped by knowing that they can't stay that way for ever	True	False
4.	I can't imagine what my life would be like in 10 years	True	False
5.	I have enough time to accomplish the things I most want to do	True	False
6.	In the future I expect to succeed in what concerns me most	True	False
7.	My future seems dark to me	True	False
8.	I expect to get more of the good things in life than the average person	True	False
9.	I just don't get the breaks, and there's no reason to believe I will in the future	True	False
10.	My past experiences have prepared me well for my future	True	False

11.	All I can see ahead of me is unplesantness rather than pleasantness	True	False
12.	I don't expect to get what I really want	True	False
13.	When I look ahead to the future I expect that I will be happier than I am now	True	False
14.	Things just don't work out the way I want them to	True	False
15.	I have great faith in the future	True	False
16.	I never get what I want so it is foolish to want anything	True	False
17.	It is very unlikely that I will get any real satisfaction in the future	True	False
18.	The future seems vague and uncertain to me	True	False
19.	I can look forward to more good times than bad times	True	False
20.	There's no use in really trying to get something I want because I probably won't get it	True	False

APPENDIX B

PROBLEM SOLVING TEST

Problem solving questions

1.	E E E E E	Solvable
2.	X Y Z A B	Solvable
3.	F G L N S	Unsolvable
4.	K L M N O	Solvable
5.	E J K G L	Unsolvable
6.	W K J F E	Unsolvable
7.	T U V W X	Solvable
8.	U S T W V	Unsolvable
9.	B G J N F	Unsolvable
10.	D E F G H	Solvable
11.	N O P Q R	Solvable
12.	R S Q U T	Unsolvable
13.	T T T T T	Solvable
14.	B D F H J	Solvable
15.	Q B K G T	Unsolvable
16.	D G K J I	Unsolvable
17.	R S R S R	Solvable
18.	O R S U V	Unsolvable
19.	C D E F G	Solvable
20.	D E B A F	Unsolvable