CHAPTER 1

Behavior Therapy

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BEHAVIOR THERAPY AND PSYCHOTHERAPY

The term *behavior therapy* has been in use for no more than 30 years, having been introduced to mark a Kuhnian revolution (Barnes, 1982; Kuhn, 1959, 1970, 1974) in the prevailing theories of neurosis (Eysenck, 1959, 1960, 1964). At the time, the prevailing paradigm was a Freudian, psychodynamic one, and it may be argued that since then there has been a paradigm shift of fundamental importance to psychiatry and clinical psychology (Eysenck, 1985, 1987). This paradigm shift from Freud to Pavlov, from psychotherapy to behavior therapy, from emotional insight learning to Pavlovian extinction and deconditioning, is in large part based on the recognition that Freudian theory has essentially failed to produce methods of treatment superior to placebo treatment, or even to no treatment at all (Eysenck, 1952; Hattie, Sharpley, & Rogers, 1984; Prioleau, Mardock, & Brody, 1983; Rachman & Wilson, 1980). Meta-analysis (Shapiro & Shapiro, 1982; Smith, Glass, & Miller, 1980) has been suggested to provide evidence in favor of the effectiveness of psychotherapy, but the method itself has been severely criticized (Eysenck, 1983; Matt & Wittman, 1985; Searles, 1985) and in any case Smith, Glass, and Miller completely failed to show that any of the theories examined (with the exception of behavior therapy) had any *specific* effects, that is, effects traceable to the special theory on which the therapy was based. Furthermore, they failed to compare psychotherapy with placebo treatment, but used placebo treatment instead as one of the 18 treatments examined! Even worse, from the point of view of psychotherapy and psychoanalysis, is the fact that strong negative effects of these types of treatment have been found (Hadley & Strupp, 1976; Strupp, Hadley, & Gomes-Schwartz,1977).

Eysenck (1959) suggested 10 major points on which the new paradigm differs from the old; these are given in Table 1. In spite of many criticisms, it can still be maintained that these points encapsulate the new paradigm, and that work done over the past 30 years has shown that, in the words of Lakatos (1970), it constitutes a

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TABLE 1

Psychotherapy	Behavior therapy
1. Based on inconsistent theory never properly formulated in postulate form.	Based on consistent, properly formulated theory leading to testable deductions.
2. Derived from clinical observations made without necessary control observations or experiments.	Derived from experimental studies specifically designed to test basic theory and deductions made therefrom.
3. Considers symptoms the visible upshot of unconscious causes ("complexes").	Considers symptoms as unadaptive conditioned responses.
4. Regards symptoms as evidence of *repression.*	Regards symptoms as evidence of faulty learning.
5. Believes that symptomatology is determined by defence mechanisms.	Believes that symptomatology is determined by individual differences in conditionability and autonomic lability, as well as accidental environmental circumstances.
6. All treatment of neurotic disorders must be *historically* based.	All treatment of neurotic disorders is concerned with habits existing at *present*; their historical development is largely irrelevant.
7. Cures are achieved by handling the underlying (unconscious) dynamics, not by treating the symptom itself.	Cures are achieved by treating the symptom itself, i.e., by extinguishing unadaptive C.Rs and establishing desirable C.Rs.
8. Interpretation of symptoms, dreams, acts, etc. is an important element of treatment.	Interpretation, even if not completely subjective and erroneous, is irrelevant.
9. Symptomatic treatment leads to the elaboration of new symptoms.	Symptomatic treatment leads to permanent recovery provided autonomic as well as skeletal surplus C.Rs are extinguished.
10. Transference relations are essential for cures of neurotic disorders.	Personal relations are not essential for cures of neurotic disorder, although they may be useful in certain circumstances.

progressive research program or problem shift, whereas the psychoanalytic theory has proved to be a degenerative research program. In this opening chapter, it may be appropriate to discuss some of the criticisms that have been made, some of the advances that have been recorded, and some of the changes in theory that have been suggested.

In recent years there has been an attempt to bring together these two incompatible models of neurotic behavior and treatment (e.g., Goldfried, 1980; Wachtel, 1977), but these attempts have not found favor in the eyes of critics like Franks (1984), Messer and Winokur (1980), and Yates (1983), who saw more clearly the incompatibility of the two approaches. Wolpe (1981) also clearly saw the irreconcilable differences between behavior therapy and psychoanalysis, and argued against the pseudoscientific eclecticism that would result from any attempt to merge them.

THE CONCEPT OF NEUROSIS

We may start with the observation that behavior therapy is intimately related with the concept of neurosis. It attempts to explain the occurrence of neurotic disorders, and it attempts to suggest methods of treatment of neurotic disorders. It thus

inevitably confronts Mowrer's (1948, 1950) paradox, which he identified by pointing, as a central feature of neurosis, to the fact that the self-defeating behavior of the neurotic is self-perpetuating. As he put it: "the neurotic paradox lies in the fact that human behavior is sometimes indefinitely perpetuated despite the fact that it is seriously self-defeating" (Mowrer, 1950, p. 524). The explanation of the Mowrer paradox given by Watson and Rayner (1920) is of course in terms of Pavlovian conditioning; neurotic symptoms are conditioned emotional and skeletal autonomic and behavioral responses that are immune from rational criticism. As I have pointed out elsewhere (Eysenck, 1968), conditioning theory so conceived does not explain why there is no extinction when self-defeating behaviors are found to be self-punishing, and are not reinforced (Kimmel, 1975). Watson's theory, although along the right lines, requires careful restatement in the light of more recent experiments and theoretical developments.

Attempts have been made to find an operant explanation of Mowrer's neurotic paradox (Tryon, 1978). This theory is based on Woods's (1974) taxonomy of instrumental conditioning. His analysis attempts to explain the apparent contradiction of the law of effect by postulating a particular subset of eight binary combinations of response contingencies, where one contingency accelerates response omission whereas the other decelerates response omission. The theory is implausible because of its complexity, because it has no factual support, but mainly because it fails to account for many well-established features of the development of neurotic responses, such as their insidious onset and incrementation through CS-only exposure (Eysenck, 1979).

Mowrer's paradox does not define neurosis, and in recent years there has been an attempt by psychiatrists to get rid of the term altogether, as for example in DSM-III. This official manual of psychiatric diagnosis has been extensively reviewed by Eysenck, Wakefield, and Friedman (1983), who point out that DSM-III is based on no particular empirical evidence, but is merely an attempt to resolve differences between psychiatric schools by committee decisions. What has happened is simply a substitution of many neurotic disorders, often highly correlated, for a general term, neurosis, under which these different neurotic disorders could be subsumed (Gossop, 1981). To say this is not to deny that the term may be difficult to define, or that it is useful to subdivide neurotic disorders into subsets that can be independently classified and diagnosed. Nevertheless, as we shall show, it appears to be true that there is a large number of mental disorders characterized by anxiety and other similar mental/autonomic/behavioral responses to stimuli that are normally unlikely to lead to strong and lasting responses of this type. These strong and lasting emotional responses in turn lead to behaviors (such as obsessive-compulsive handwashing) the purpose of which is to reduce neurotic anxiety (Gossop, 1981). Watson's theory posits that these emotional reactions are produced by Pavlovian conditioning, and may be cured by Pavlovian extinction.

Neurotics have certainly been with us for a very long time (Simms, 1985), and they impose a great stress on society (Simms, 1983). The difficulty introduced by varying levels of neurotic disorder, many of which are never seen by the psychiatrist, has given rise to a very useful model for mental illness in the community, introduced by Goldberg and Huxley (1980). They use the concepts of levels and filters. A large number of people in the community suffer from psychological symptoms in any one year (Level 1). Most of these pass the first filter and seek help from their general

practitioners (Level 2). Many of these the general practitioner recognizes as suffering from psychological symptoms (Level 3), and a quite small number of these are referred to a psychiatrist (Level 4). Of those seen as psychiatric outpatients, even fewer are admitted as inpatients (Level 5). It follows that neurotic disorders are predominantly concentrated in the community and form a smaller proportion of those patients seen by the psychiatrist. At Level 5, when the International Classification of Diseases was applied to all psychiatric inpatients in a District General Hospital Psychiatric Unit, 21% were found to be suffering from neurotic disorders (Zigmond & Simms, 1983). When serial psychiatric outpatient referrals were classified diagnostically (Level 4) 60% were found to have a primary diagnosis of neurosis (Simms & Salmons, 1975). In a large study of diagnosis in a population of 300,000 people in general practice (Level 3), the consultation rate for all neuroses was 75.5 per 1000 per annum for males, and 162.9 for females, which gave a rate of over 90% for neuroses among all psychiatric diagnoses (Office of Population Censuses and Surveys, 1974). It is clear that as we go from the community via general practice to the psychiatric outpatient/inpatient treatment, the number of neurotic patients diminishes, but the severity of their neurotic symptoms increases.

Neuroses are found extremely commonly among inpatients and outpatients of hospital specialities other than psychiatry. For instance, it has been estimated that 15% to 20% of hospital presentations in the opthalmological clinic are for neuroses (Karseras, 1976). It seems certain that neurotic disorders constitute most of the psychiatric illnesses encountered in general practice, but that only a small proportion of these cases is referred to hospital (Kessel, 1960; Kessel & Shepherd, 1962). This large class of sufferers cannot be conjured out of existence by dropping the concept of neuroses.

The varied manifestations, symptoms, and correlates of neurotic disorder may interact with the personality dimension of extraversion-introversion to produce extraverted (hysterical) or introverted (dysthymic) disorders (Janet, 1890, 1903; Jung, 1923; Eysenck, 1947). Figure 1 shows the results of a factor analysis of various notations on 700 male neurotics, and Figure 2 shows a similar analysis of symptoms intercorrelated and factor analyzed for a large group of children in a child guidance clinic (Eysenck, 1970a). These data will illustrate the complex of feelings and behaviors constituting the different neurotic disorders. More details concerning neurosis and the personality trait of neuroticism underlying it are given in a later chapter in this book (Genetics and Preparedness), which will also discuss the relative influence of genetic and environmental determinants on neurosis.

The major evidence suggesting that it may be meaningful to postulate the concept of neurosis, as opposed to normality and psychosis, comes from factor analytic studies demonstrating (a) that these three concepts require two dimensions to accommodate the observed relationships, thus making it impossible to postulate, as Freud had done, that psychosis is merely a further development of neurotic illness, and (b) that most neurotic and psychotic disorders are more easily conceived as end points of two different continua, rather than as categorically different from normality (Eysenck, 1970b).

The theory to be outlined here states that the different types of neurotic illness arise through a process of Pavlovian conditioning, and can be eliminated through a

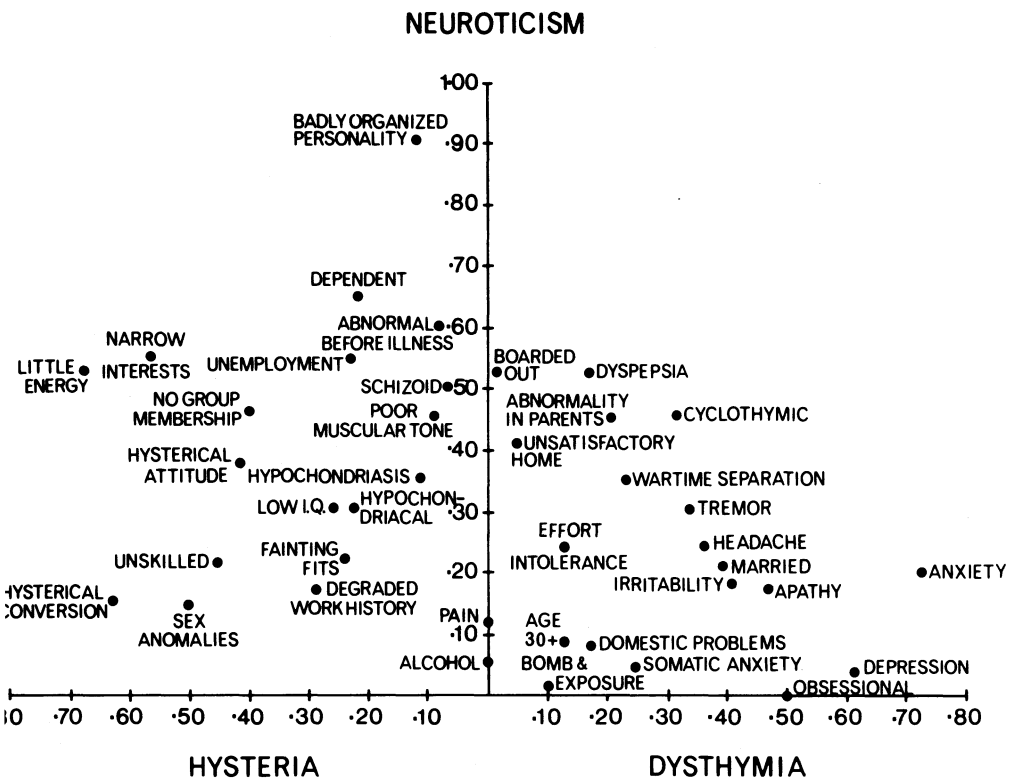

FIGURE 1. Neuroticism as a factor in neurosis, divided into introverted (Dysthymia) and extraverted (Hysteria) behaviors. (From Eysenck, 1947.)

process of Pavlovian extinction. It is often objected that there are certain types of existential problems that would be difficult to understand along these lines, and that would seem difficult to treat by means of behavior therapy. Without wishing to enter into a sustained discussion of the topic, it should be pointed out that it would be unrealistic to assume that all the patients presenting at psychiatric clinics are suffering from either neurotic or psychotic disorders, or a combination of the two. Thus, when psychotic disorders have been eliminated, it would be quite wrong to assume that the remainder must all be suffering from neurotic disorders. Not all anxieties and fears are irrational, and many children and adults presenting in psychiatric hospitals and clinics may require advice and guidance rather than behavior therapy. Similarly, if existential fears, worries, and doubts do not arise from a process of conditioning, along the lines suggested, they would not seem to fall under the general heading of neurosis. The collection of patients normally seen by psychiatrists and clinical psychologists is a relatively arbitrary grouping possessing little in the way of homogeneity. It is scientifically perfectly legitimate to subdivide this heterogeneous total into smaller groups showing that essential uniformity that is required for the elaboration of general laws.

At first sight this argument may seem circular, but in fact it is one that is commonly found in the hard sciences. If we ask whether Euclidian geometry applies

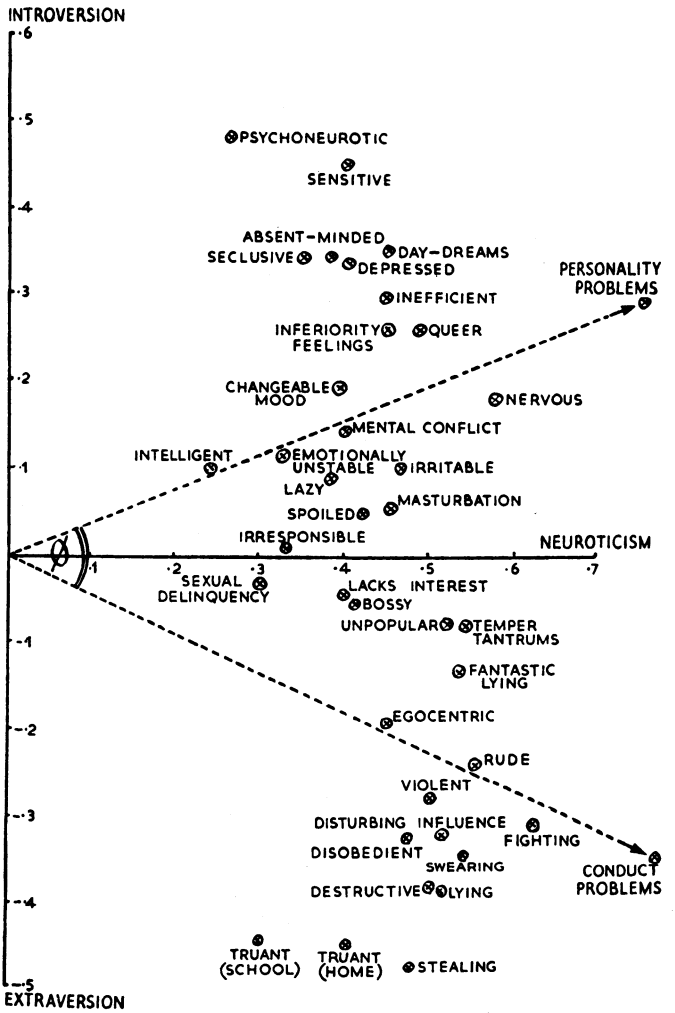

FIGURE 2. Two-factor representation of Ackerson's Correlational Study. Extraverted and introverted forms of neurotic behavior in a group of child guidance clinic children. (From Eysenck, 1970.)

to a particular type of measurement, say of a given part of the earth's surface, we answer that it applies only to planes. If we chose a small part of the earth's surface, say an acre, it is sufficiently close to a plane to make Euclidian geometry applicable. If we chose a larger surface, such as a continent, clearly this is curved, and hence Euclidian geometry does not properly apply. How do we know whether a given surface is or is not a plane? The answer of course is in terms of the application of Euclidian principles; if they apply, it is a plane, if they do not, it is not.

It is also important to remember that in our definition we are dealing with a scientific law, and that such laws always have limits to the conditions under which they apply. Take the law that tells us what the speed of fall S, in metres per second, would be at any point along the path of fall of a body dropped near the earth's surface. The formula of course is $S = 4.43^2h$, where h is the distance that the body

has already fallen, measured in metres. Such a law would not apply to any body whose size, shape, or speed of fall is such that air resistance affects its motion appreciably. In a similar way the application of the general law relating neurosis and conditioning implies limits to the conditions under which it applies, and these should always be borne in mind. They do not constitute a negation of the law, just as little as air resistance implies a negation of the law of falling bodies.

Furthermore, the postulation of classical conditioning and extinction as being the core of the theory of neurosis does not rule out other processes (cognitive, operant conditioning, etc.) as being powerfully involved. Successful treatment along the lines of behavior therapy may produce reactions on the part of spouses, relatives, and others that reward or punish the patient for the improvement in his or her state; this may have positive or negative effects on the success of therapy. These factors, however, are adventitious rather than central, and must be sharply differentiated from the centrally placed factors postulated by the theory. More will be said on this point later on.

There has been a dearth of studies directly investigating the origins of neurotic disorders, and those that have been done have concentrated almost exclusively on phobic disorders (Murray & Foote, 1979; Öst & Hugdahl, 1981; Öhman, Dimberg, & Öst, 1985; Rimm, Janda, Lancaster, Nake, & Dittmar, 1977). The general finding seems to be that in a majority of cases conditioning experiences are remembered. In what is probably the best of these studies, Öst and Hugdahl found that vicarious experiences only accounted for 17%, and instructions/information only for 10%. Similarly Rimm *et al.* found that vicarious experiences accounted for 8% and instructions for 11%. Murray and Foote found a higher proportion of indirect ways of acquiring fears, but they did not use clinical patients with phobias, concentrating on undergraduate students with a fear of snakes. A fair number of subjects in all three studies failed to recollect any specific causal factors. "There is no clear-cut relationship between the ways of acquisition and anxiety components (subjective, behavioral, psychological), nor did the conditioning and indirectly acquired phobias differ in severity." This is clearly an area where much more research is urgently needed, preferably using interview questioning rather than questionnaire data. We shall return to it in a later section in connection with treatment procedures.

CRITICISMS OF BEHAVIOR THERAPY

We may now turn to some of the criticisms that have been made of the conception of behavior therapy here outlined. First, let us consider some points made by Breger and McGaugh (1965), Locke (1971), and London (1972). These and other authors first challenged the theoretical basis of behavior therapy, maintaining that the laws of learning on which behavior therapy was said to be based remained to be established themselves. As they pointed out, fundamental issues, such as the role of mediational events in behavior change, the nature of responses learned, and the limitations of a stimulus-response analysis, had not yet been resolved. They made the point that behavior therapy mistakenly assumed a monolithic learning theory as a basis of behavior therapy as an applied science, but, if learning theory itself had

not succeeded and resolved its major issues, then how could behavior therapy rely on the theory as an established guide (Erwin, 1978)? And in the second place, critics have argued that the principles of learning theory, if any such existed, do not in fact inform the practice of modern behavior therapists. Instead, it is argued that serendipity, nonspecific factors such as suggestibility, the personality of the therapist, etc., are responsible for the success of treatment, if any. These criticisms, which have been frequently repeated, deserve an answer.

It is certainly true that the principles of learning theory are not as firmly established as one would like (Zuriff, 1985), and that indeed the whole doctrine of behaviorism is under assault (Mackenzie, 1977). But of course this is precisely what characterizes a new paradigm. Barnes (1982) comments on the

> perceived inadequacy of a paradigm as it is initially formulated and accepted . . . its crudity, its unsatisfactory predictive power, and its limited scope, which may in some cases amount to but a single application. In agreeing upon a paradigm scientists do not accept the finished product: rather, they agree to accept it as a basis for future work, and to treat as illusory or eliminable all apparent inadequacies and defects. Paradigms are refined and elaborated in normal science. And they are used in the development of further problem-solutions, thus extending the scope of scientific competences and procedures. (p. 46)

Newton's *Principia Mathematica* was dismissed out of hand by French physicists for similar reasons to those adduced by Breger and McGaugh, and his mathematical treatment of the calculus was not made rigorous until 150 years after his death, in Cauchy's *Cours d'Analyse.* If we followed the lugubrious advice of the critics, no scientific advances would ever be possible. The fact that a large number of queries remain as far as the application of learning theory to behavior therapy is concerned, and indeed, as far as the establishment of learning theory itself is involved, cannot be an argument against the new paradigm. Quite the contrary; it is because this is a new paradigm that these problems remain for normal science to settle. Some of the advances made since the days of Breger and McGaugh will be recounted in this volume. A more detailed discussion of the point has been given by Eysenck (1976) in his chapter entitled "Behavior Therapy—Dogma or Applied Science?"

Among the critics of learning theory as a basis of behavior therapy, perhaps the most notable is Wolpe (1976a, b). He argues that behavior therapy is a synthetic construct, and is to be defined in terms of "principles and paradigms" rather than "learning theory." The case is argued in detail by Eaglen (1978), who concludes that

> the development of theories . . . and their careful application to treatment programs is vital for the future development of behavior therapy, and it is only by insisting on a close link between therapy, theory development and research evidence that we can avoid the otherwise inevitable eclipse. (p. 128)

Wolpe's definition encounters the obvious danger that it may seem to encourage the "broad-band" eclecticism that he himself criticizes (Wolpe, 1976a, b).

It is difficult to see how it can seriously be argued that behavior therapists, in developing their methods, do not base themselves on principles of learning and conditioning theory. Wolpe (1958) himself clearly has done so in his development of desensitization therapy, and obviously Watson and Rayner (1920), in suggesting detailed methods of treatment to Jones (1924), had a clear theoretical rationale of Pavlovian extinction in mind. The work done under my direction at the Institute of

Psychiatry (Eysenck & Rachman, 1965; Rachman & Hodgson, 1980) has always been buttressed by reference to theoretical positions in learning theory, and most of the authors who have contributed original material in this field have clearly drawn on the same treasure house. It is not necessary to assert that they all make use of the same basic theories, or make identical deductions, but it would be difficult to find anyone who claimed to have derived his methods without benefit of prior experimental and theoretical work on the principles of learning and conditioning.

This is true even of those who have chosen electicism as a way of life. As Eysenck (1970a) has made clear, their applied work is characterized not by a rejection of theory as such, but rather by an overindulgence in reliance on heterogenous theories that do not form any sensible kind of whole, and are often contradictory, partial, and difficult to integrate in a meaningful manner. Eysenck (1987) gave several examples of the development of methods of behavior therapy following on experimental laboratory work with animals, and the theories associated with that work. Critics seldom examine particular cases in order to try to demonstrate the alleged absence of reliance on theoretical formulations; they make wholesale suggestions without specifying the precise methods of therapy they have in mind as not being influenced by learning theory.

Altogether it seems that psychologists, possibly because they are often derided because their science, as William James suggested, was merely the "hope of a science," tend to take theories and their defects much more seriously than do hard scientists. Take as an example research in cryogenics. As Mendelsohn (1966) pointed out:

> As was inevitable, ever since superconductivity was first discovered, many different theories with explanations have been proposed; roughly at the rate of 2 or 3 per annum, and for the better part of half a century. . . . [E]ventually Felix Bloch, who has done so much for our understanding of electrons in metals, annunciated an axiom of his own which ran: "every theory of super-conductivity can be proved wrong." And for a long time this axion turned out to be the only correct one.

Yet in spite of this unpropitious state of affairs, theories of superconductivity have been used from the beginning to further practical ends, and the results of applied research have been used to disconfirm or improve existing theories.

Wolpe's (1976a, b) argument that because there is no one "modern learning theory," it is meaningless to define behavior therapy in terms of such a theory, and that instead treatment should be based on "principles and paradigms" is clearly one that hard scientists would not accept. We do have more than one theory, as is true of people working on cryogenics, and the correct way to use these is to make differential predictions from different theories, as far as application is concerned, and then study the results in order to choose between different theories. This is the way in which physics and chemistry have advanced so successfully over the last three centuries, and there is no reason to assume that psychology is positioned any differently.

But, it may be objected, is it not true that there are many different behavior therapies, rather than one single behavior therapy? The fact is not to be doubted, but its interpretation would seem to be somewhat different. The theory maintains that all cures of neurotic disorders are based on Pavlovian extinction, hence it makes sense to talk of behavior therapy. However, extinction can be produced along many different lines, but always involving the unreinforced exposure of the conditional

stimulus, either *in vivo*, or else in imagination. Among the best known procedures for producing extinction, we have modeling, desensitization, and flooding with response prevention, as well as many combinations and modification of these. Thus those who insist that there is one behavior therapy, and those who would prefer to talk about different "behavior therapies" are both right; there is one underlying principle on which all behavior (and other) therapies operate, but the application of this principle can take an infinite variety of forms.

CLAIMS OF COGNITIVE BEHAVIOR THERAPISTS

Cognitive psychologists, like Beck (1976), Mahoney (1974), and Meichenbaum (1977) have made strong and increasingly inclusive claims for what they sometimes term cognitive behavior therapy, with the stress more on the cognitive than on the behavioral side. Actually it is very difficult indeed to find any coherent account of theories, deductions, and experiments relevant to the claims made. Allport (1975) characterized the whole field of cognitive psychology in a rather unflattering summary. It is, he maintains, typified by

> an uncritical, or a selective, or frankly cavalier attitude to experimental data; a pervasive atmosphere of special pleading; a curious parochialism in acknowledging even the existence of other workers, and other approaches, to the phenomena under discussion; interpretations of data relying on multiple, arbitrary choice-points; and underlying all else a near vacuum of theoretical structure within which to interrelate different sets of experimental results, or to direct the search for significant new phenomena.

M. W. Eysenck (1984), in his *Handbook of Cognitive Processes*, points out "the extremely diverse and sprawling nature of the current scene" (in cognitive psychology). And he goes on to say that

> at least part of the reason for the growing army marching behind the banner of cognitive psychology is the increased vagueness with which the term is used. Virtually all those interested in perception, learning, memory, language, concept formation, problem solving, or thinking call themselves cognitive psychologists, despite the great diversity of experimental and theoretical approaches to be found in these various areas. (p. 1)

Eysenck finally characterizes cognitive psychology in terms of its "strong reaction against the facile approach of Behaviorism" (p. 2). This no doubt is true, but a reading of the criticism of behaviorism contributed by leading cognitive psychologists suggests that their criticisms are directed at the 1920 reflexological model, rather than the more up-to-date and much more formidable modern neobehaviorism presented, for instance, in Zuriff's (1985) book.

It is one of the sad features of this debate that it seems to be quite tangential to the real claims and issues. There can be no doubt about the real strength of modern neobehaviorism, yet sadly enough cognitivists disregard it completely, and direct their arguments at out-of-date beliefs, no longer held by behaviorists. Equally, behaviorists tend to play down the very real contributions made in many different fields by cognitivists, as outlined by M. W. Eysenck (1984). In so far as cognitive psychologists attempt to introduce cognitive concept into the behaviorist framework, they are simply following in the footsteps of Pavlov, who argued powerfully that

words can be both conditioned stimuli and conditioned responses, with the implication that cognitive events follow the same laws as behavioral events (Ullmann, 1981)

In talking about behavior therapy, we should of course bear in mind that different types of behavior may not covary in time in any precise manner. Lang (1970) endeavored to construct a three-system analysis of fear reactions, and later work by Hodgson and Rachman (1974) and by Rachman and Hodgson (1974) suggests that fear/anxiety reactions could best be regarded as consisting of at least three loosely coupled systems—subjective, behavioral, and physiological (see also Grey, Sartory, & Rachman, 1979). These authors have reviewed the evidence to show that one or more of these systems can be discordant at any particular time, and can change more rapidly or more slowly than the others in response to treatment (desynchrony). Some behaviorists might refuse to recognize the subjective reactions that make up one of the three systems as truly subject to scientific analysis, because of the well-known objection to introspective evidence of most behaviorists (Zuriff, 1985). Indeed, if it be true, as Nisbett and Wilson (1977) maintain, that subjects are sometimes unaware of the existence of a stimulus that importantly influences a response, are unaware of the existence of the response, and are unaware that the stimulus has affected the response, then indeed we might have to follow the restrictionist line of argument. However, as Nisbett and Wilson point out, accurate reports do occur when influential stimuli are salient and are plausible causes of the responses they produce; this would seem to cover the cases of fear/anxiety in neurotic patients, and the changes that take place during therapy.

It might be thought that the strong evidence regarding the primacy of affect (Rachman, 1981; Zajonc, 1980, 1984) would rule out subjective reports as important systems, but surely this is not so. Even though affect may precede cognition, and affective arousal may not always entail prior cognitive appraisal, this does not rule out the existence of such cognitive appraisal, and its importance for the patient.

Recognition of the desynchrony of affect has led to an interesting paradox pointed out by Bandura (1977). As he states,

> on the one hand, explanations of change processes are becoming more cognitive. On the other hand, it is performance based treatments that are proving most powerful in effecting psychological changes. Regardless of the method involved, the treatments implemented through actual performance achieve results consistently superior to those in which fears are eliminated through cognitive representations of threats. (p. 78)

Bandura (1977), goes on to argue that

> the apparent divergence of theory and practice will be reconciled by recognizing that change is mediated through cognitive processes, but the cognitive events are induced and altered most readily by experiences of mastery arising from successful performance. (p. 193)

Rachman and Hodgson (1974) draw the inference that different methods of behavior therapy may be appropriate, depending on which of the three systems is most deviant, and make appropriate suggestions in this respect. They are more willing than most behaviorists to accept the subjective, introspective, mental type of fear reaction as equally important with the behavioral and physiological types of response, and of course cognitive psychologists not only accept this, but emphasize cognitive components, sometimes to the exclusion of physiological and behavioral ones. The

growing stress on cognitive components, mentioned by Bandura, makes it necessary to devote some consideration to their claims.

We may now turn to a consideration of the specific arguments advanced by cognitivists in the field of behavior therapy. The position taken here is essentially that of Wolpe (1978), who argued that cognition is also behavior and is subject to the same law of inevitability as other behavior. Accepting the Lang-Rachman-Hodgson theory of desynchrony, he argues that "overcoming the unadaptive learned habits typified by neurotic behavior requires cognitive, autonomic and motor relearning, according to the indications of behavior analysis" (p. 437). If we can embody cognitive processes within a behavioristic framework, what then is the major contribution of cognitive psychologists?

Marzillier (1980) pointed to three major usages made by cognitive therapists: cognitive events, cognitive processes, and cognitive structures.

> Cognitive events have been readily assimilated into behavior therapy, and, as Beck pointed out, they have been there from the beginning. What has emerged has been the increasing interest in cognitive events as dependent variables, the focus on client's thoughts and images in relationship to their emotional problems. This is an area that cognitive therapy has pioneered, and its techniques and practices are of value to behavior therapists. Much less attention has been paid to cognitive processes in behavior therapy. However, it is evident from recent developments in behavior therapy, that therapists are beginning to focus directly on deficiencies in cognitive processing, using cognitive restructuring and problem-solving methods as part of the behavior approach. Particular attention is drawn to the implications of cognitive appraisal which can be seen as stressing the meaning of events and behavior. In behavioral analysis a concern for meaning should provide greater breadth and sensitivity in the processs of assessment. Finally, cognitive therapists have stressed the need to consider long-term fundamental cognitive change, as a goal of therapy. The term "cognitive structures" has been used but as yet it lacks precise meaning. It is possible for behavior therapists to consider cognitive structures, such as beliefs or attitudes, as these can be linked closely to observable behavior. However, the value of so doing remains to be established. (p. 256)

Are these alleged contributions to behavior therapy real, or are they merely promissory notes drawn against a nonexisting account? Latimer and Sweet (1984) gave a critical review of the evidence concerning cognitive versus behavioral procedures in cognitive-behavior therapy. They mention the increasing emphasis on cognition in psychology and behavior therapy during the past decade, and address the question of whether cognitive therapy is an evolutionary or revolutionary development from behavior therapy. They also critically evaluate the evidence for the efficacy of procedures specific to cognitive therapy. Their conclusions are worth quoting in full.

> Cognitive therapy is an evolutionary rather than a revolutionary development in the field of behaviour therapy. It is unique only in its greater emphasis on one class of behavior—cognitions. Several innovative therapeutic methods have been spawned as a result of this shift and emphasis, but these have not been demonstrated to be efficacious in the treatment of clinical populations. Cognitive therapy as actually practiced usually involves a variety of methods including behavioral procedures of established efficacy. Most of the claims made in support of cognitive therapy are based on studies employing these cognitive-behavioral methods. It remains to be demonstrated either that the new cognitive therapy procedures make a significant contribution to therapeutic outcome or that existing behavioral methods are rendered more effective when conceptualized in cognitive terms. The widespread adoption

> of cognitive treatment procedures is unwarranted on the basis of existing outcome data involving clinical populations. (p. 21)

This paragraph highlights the problem that is posed by the claims made by cognitivists. The main claim of behavior therapy to a higher scientific status than psychotherapy has always been its willingness to attempt to prove its assertions by actual clinical experiments, and to employ empirical comparisons between different types of treatment in order to establish the superiority of one over the others. Cognitive psychologists have reverted to the older practices of psychoanalysts and psychotherapists, all making claims without furnishing proof that these claims are actually justified. By using mixed methods of treatment they make it impossible to distinguish the contribution of cognitive and behavioral variables. Until and unless they bring forward actual experimental proof of the superiority of their methods, it is impossible to concede these claims. By presenting these claims as a "paradigm shift" they suggest a successful revolution in our conception of neurosis and therapy, but there is really no justification for this claim (Eysenck, 1987). Revolutions in science are based on demonstrated effectiveness, not on speculation and theoretical argument unsupported by empirical data. There has been a paradigm shift in this field, but it has been from psychotherapy to behavior therapy. The possibility exists that the theoretical advances noted by Marzillier will result in an actual demonstrable improvement in the rate of recovery of neurotic patients, but until this has been clearly substantiated it would be premature to jump on this particular bandwagon.

'It may be useful, at the end of this section, to note some of the ways neobehaviorism (or what Davey, 1983, a,b, calls dialectical behaviorism) attempts to cope with the complexities of human behavior that are not normally covered by the older forms of behaviorism. First and foremost, as we have seen, it uses words and language as part of a conditioning system (Platonov, 1959; Staats, 1964, 1968). In the second place, we have the work of Levey and Martin (1983) and Martin and Levey (1985) to demonstrate the existence of evaluative conditioning as a process that uses the principles of conditioning in a specifically human context. Third, we have the insistence on the importance of central representations in the conditioning process.

Mackintosh (1984) makes it quite clear how views have changed in recent years as far as learning theory is concerned.

> The view of conditioning as the establishment of new reflexes or the strengthening of S-R connections, a view which dominated Western learning theory for half a century, has gradually given way to a view of conditioning as the acquisition of knowledge about the relationship between events in an animal's environment, knowledge which may not be immediately apparent in any change in behavior at all. When a CS is regularly followed by a reinforcer, animals can be said to learn that the CS signals the reinforcer. This is achieved by the establishment of an association between some central representations of the two. From studies that have altered the value of a reinforcer after conditioning, it is apparent that the representation of the reinforcer associated with the CS must, in at least some cases, itself be available for modification when their value is manipulated. (p. 56)

Such "central representations" are of course cognitive processes as properly defined, and hence modern learning theory incorporates cognitive processes in a most explicit fashion; and it is not clear why cognitive theorists should claim exclusive patronage of such processes, or why they should declare that modern learning theory is incomplete

because it does not take them into account, when clearly it does (Rescorla, 1972). A more detailed discussion of this whole problem is given by Eysenck (1987).

The S-S analysis of conditioning can very easily be integrated with an information-processing paradigm, which reflects the more empirical contributions that cognitive psychology has to make to learning theory (Kanfer & Hagerman, 1985). Reiss (1980) and Bootzin (1985) outline such a theory following Wagner and Rescorla's (1972) information model. These developments are implicit in Tolman' (1948) view of learning theory, and do not present an alternative view to theories of cognition. Altogether, those who oppose cognitive to conditioning theory would seem to commit the logical fallacy technically known as the *unacceptable entymeme*. It presents an argument with one of its stages understood rather than stated, the understood premise being that conditioning theories are of the Watsonian S-R type; this premise is clearly erroneous.

A NEW CONDITIONING MODEL OF NEUROSIS

Watson's conditioning theory has encountered many criticisms, and at first sight these seem fatal to it, certainly in its original form. Some of these will be considered in a later chapter by the present author; others will be considered here. Eysenck (1979, 1982a) has pointed out that Watson's theory (like Freud's) is based on the occurrence of a traumatic fear-producing event constituting the Unconditional Stimulus (UCS), which is followed by fear/pain responses that constitute the (UCR) Unconditional Response. Neutral stimuli accidentally present at the time will become conditioned through contiguity, thus being made into CSs that from then on will evoke CRs similar in nature to the UCR, that is, feelings of fear and pain. These CRs continue indefinitely, unless deconditioned along the lines discussed by Watson and Rayner (1920), and exemplified in the work of Mary Cover Jones (1924). This account raises the following problems.

The first problem is a clinical one. War neuroses often do begin with a traumatic event, such as the person in question being buried alive by an explosion, or coming into contact with death or mutilation of friends and colleagues. However, in civilian neuroses such events are very rare, and in the majority of cases the initiating event is not excessively traumatic, and does not produce an immediate, strong CR. Rather, there appears to be an insidious increase in the anxiety produced by the CS that may take years, or even decades before a full-blown phobia becomes apparent, or a clinical state of anxiety is reached. This is the major clinical objection to the theory.

From an experimental point of view, a second objection is the simple one that on this account extinction should set in almost immediately, making impossible the development of any long-lasting neurosis. Whatever the CS may be, the subject is likely to encounter it quite frequently and without attending reinforcement. This should produce relatively quick extinction of the CR. Let us consider a person suffering from a cat phobia; he or she is likely to encounter cats in nonthreatening situations quite frequently, and each such encounter should foster extinction. The phobia should thus quite soon disappear. The fact that this does not seem to happen is a powerful argument against Watson's theory (Kimmel, 1975).

A third point of importance is that in ordinary Pavlovian conditioning there is

no way in which the CR could be stronger than the UCR. Yet if we look at clinical cases, as mentioned earlier, the initiating conditioning experience often leads to UCRs and CRs that are rather mild; it is only after the insidious development of the neurosis has taken place that the CRs become so strong as to constitute an actual mental illness. Hence in these quite typical cases of neuroses and phobias, the CR becomes much stronger than the original UCR; on ordinary Pavlovian principles this would seem to be impossible.

What these three objections have in common, of course, is a reference to the development of the CR over time, when the subject is exposed a number of times to the CS only, that is, to the CS without simultaneous reinforcement. Classical conditioning theory would expect extinction under these conditions, but what happens in the case of the development of a neurotic illness seems to be the opposite, that is, an incrementation of the CR. To explain this anomaly, Eysenck followed up Grant's (1964) suggestion that there was an important distinction between Pavlovian A and Pavlovian B conditioning, and proposed that the consequences of this distinction are important in regard to extinction. (Eysenck, 1967, 1968, 1976, 1979, 1980, 1982a, b)

Pavlovian A conditioning is exemplified by the textbook example of classical conditioning, that is, salivation on the part of the dog to the sound of a bell that had been repeatedly presented shortly before food was given to the hungry dog. Of the many UCRs presented to the dog (approach to the food, ingestion, etc.), Pavlov chose only to measure one, namely buccal salivation. As Zener (1937) pointed out, it is noteworthy that the CR did not include approach to and attempts to feed upon the bell or other source of the CS. Any approach and reorientation movements were directed to the food source, showing that the CS does not substitute for the UCS, as S-R theorists have often stated. Pavlov maintained that the CS serves as a signal that the food is about to be presented, and this position is also taken by S-S theorists. This approach is now almost universally recognized as being more in line with the facts than the old-fashioned S-R approach (Mackintosh, 1984).

Pavlovian B conditioning is directly linked by Grant (1964) to the Watson and Rayner (1920) experiment, but as he points out, Pavlov has priority. A reference experiment for Pavlovian B conditioning could be that in which an animal is given repeated injections of morphine. The UCR in this case involves severe nausea, profuse secretion of saliva, vomiting, and then profound sleep. After repeated daily injections, Pavlov's dogs were found to show severe nausea and profuse secretion of saliva at the first touch of the experimenter (Pavlov, 1927, p. 35–36).

The major differences between Pavlovian A and B conditioning relate to drive, and degree of similarity between CR and UCR. In Pavlovian A conditioning, no learning takes place unless the subject is in a suitable state of drive, such as hunger in the case of salivary conditioning in dogs. In the case of Pavlovian B conditioning, the UCS provides the drive or motivation. In Watson's theory the UCS clearly provides the drive, making his a case of Pavlovian B conditioning.

In Pavlovian B conditioning, the UCS elicits the complete UCR, whereas in Pavlovian A conditioning the organism emits the UCR of approaching and ingesting the food. Thus in Pavlovian B conditioning the CS appears to act as a (partial) substitute for the UCS, which is not true of Pavlovian A conditioning. Expressed in different terms, we may say that in Pavlovian A conditioning typically the CR and

the UCR are different (salivation as opposed to approach to and ingestion of food), whereas in Pavlovian B conditioning they are similar or identical (nausea, profuse secretion of saliva, vomiting). As Grant points out, many components of the UCR in Pavlovian conditioning "are readily seen as components of the CS which will be evoked by the preparations of the injection after repeated daily morphine injections." (p. 5). A great deal of interoceptive conditioning (Bykov, 1957) and autonomic conditioning (Kimble, 1961) appears to follow the Pavlovian B paradigm.

These differences between Pavlovian A and Pavlovian B conditioning can be used to argue that the consequences of CS-only presentations may be quite different in the two paradigms. (Eysenck, 1976). In Pavlovian A conditioning, it is meaningful for both the subject and experimenter to talk about CS-only presentation as the presentation of the CS that is not followed by the UCS. However, in Pavlovian B conditioning this is difficult to accomplish because the CR, which follows the CS, is for all purposes identical with the UCR. Consequently, the phrase *CS-only presentation* is meaningful for the experimenter, who controls the presentation of the UCS, but not for the subject, who experiences the CR as identical with the UCR. In Pavlovian B conditioning, if it be true that the CS-only condition is not necessarily fulfilled (as far as the subject of the experiment is concerned), then it would seem to follow that the ordinary laws of extinction might not always apply. Although the experimenter has arranged the contingencies in such a way that CS is not followed by UCS, under certain conditions (to be specified later) the CR itself might act as a reinforcement equivalent to the UCR, thus producing not extinction but an increment in the strength of the CR. This incrementation has been called *incubation* and has led to a revised conditioning theory of neurosis (Eysenck, 1968, 1976).

There has been much discussion of the incubation phenomenon, and the large body of research that supports it (Eysenck, 1976, 1979, 1982a); there is no space to review the evidence again here. Incubation is a process that is theoretically intelligible in terms of Pavlovian B conditioning, and experimentally verified by many animals and a few human experiments. We also have both theoretical and practical evidence concerning some of the variables that make for incubation rather than extinction, such as strength of the UCR and CR, duration of exposure of the CS-only, personality, etc. (Eysenck, 1982b).

The general form of the theory of incubation and extinction in neurotic fear reduction is shown in diagrammatic form in Figure 3. It shows on the ordinate the strength of the CR, and on the abscissa the duration of CS-only exposure. Curve A illustrates the decline in fear/anxiety with duration of CS exposure; there is ample evidence from the animal and particularly from the human field (Rachman & Hodgson, 1980, Figure 14.1) to support the general decline over time of the fear/anxiety reaction. The theory states that on this curve there is a critical point. If CS-only exposure stops before this point is reached, that is, while the strength of the CR is above this level, incubation will result. If at termination of CS-only exposure the strength of the CR is below this critical point, extinction will result. Thus duration of exposure is a critical element in deciding whether incubation or extinction is to result from treatment or experiment, and there is much evidence from the clinical field to support this view (Eysenck, 1982a, 1983, 1986; Eysenck & Beech, 1971).

If CS-only exposure is continued long enough to provide an increment of extinction, Curve A will be lowered on the next occasion, as it is indicated by Curve B,

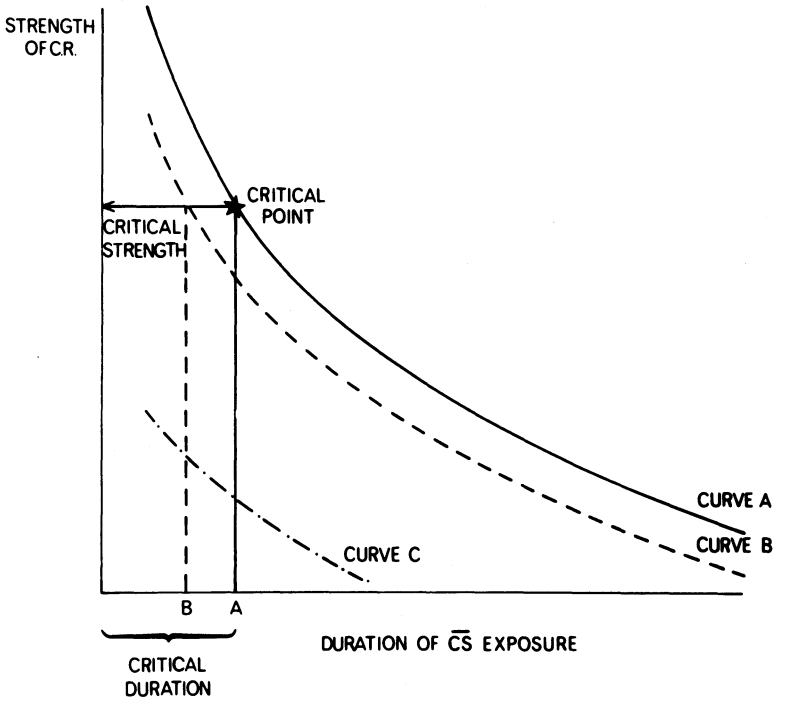

FIGURE 3. Diagram of incubation versus extinction theory.

and subsequent increments of extinction will reduce the whole curve below the critical point, as in Curve C. Curve A indicates a typical sequence of events when flooding with response prevention is used as a therapeutic technique; Curve C indicates the level at which desensitization and modeling proceed.

Strength of the CR and duration of CS-only exposure are not the only critical variables; as we shall see later, personality (and the concentration of peptides and hormones that control both personality and fear/anxiety reactions) also play an important part. Note that the theory is also relevant to the acquisition of fear/anxiety responses; if the original CR exceeds the critical point, then incubation will occur and the final CR will be stronger than the original UCR, an event not contemplated in Pavlov's original theory, but clearly apparent in experimental animal studies, as well as characteristic of the development of human neuroses (Eysenck, 1982a, 1986.)

THE NEUROBIOLOGY OF INCUBATION OF FEAR/ANXIETY*

We can trace the variegated events of extinction and incubation a little further into the biological realm by considering individual differences in levels of neurohormones. The hypothesis developed by Eysenck and Kelley (1987) largely stems from 35 years of animal research that has shown that neurohormones can have a profound modulating influence on resistance to extinction. Considerable experimental and

*This section is paraphrased from a more detailed account by Eysenck and Kelley (1987).

clinical work with humans is consistent with the possibility that it is individual differences in these hormones that mediate the persistence characteristic of disorders such as phobias, and the absence of persistence typical of depression. In relation to the incubation concept, Eysenck and Kelley argue that individual differences in levels of peptides, such as ACTH, allow the fear-producing CSs to increase dramatically in excitatory strength, or to decrease and extinguish, depending on the hormone and the duration of CS exposure. The literature suggests that hormonal mediation of incubation is a reliable phenomenon. At the level of psychological processes, it is suggested that incubation occurs by hormones influencing mechanisms of attention so as to produce changes in CS associability or in the absolute capacity of a CS to have inhibitory or excitatory strength. At the level of psychological treatment of neurosis, this model predicts that an intervention strategy involving both hormones and conditioning may have more impact than manipulation of only one of these factors.

Apart from peripheral endocrine functions, hormones are present in the CNS and affect emotions by the modulation of activity in the limbic system. Patients with panic attacks, for instance, have limbic abnormalities (Reiman, Raichle, Butler, Herscovitch, & Robins, 1984), and it is known that the behavioral effects of hormones are dependent on the integrity of limbic structures (de Wied & Jolles, 1982; Van Wimersma Greidanus *et al.*, 1983). In addition, hormone-induced changes in hippocampal theta occur that show some correspondence with anxiety-related behavioral outcomes (Gray, 1982; Urban, 1984; Urban & de Wied, 1978). This modulation of limbic activity is the balanced outcome of many hormones. The hormones have precedence in the course of evolution, and are of at least equal importance as the better-studied neurotransmitters (Iverson, 1984; Krieger, 1983; Le Roith, Shiloach, & Roth, 1982).

It may be useful to begin our discussion with a mention of some of the findings that suggest a relationship between neurohormones and neurosis. Redmond (1981) and Hall (1979) have demonstrated that there are similarities in the symptoms of anxiety neuroses and withdrawal from opiate addiction, an observation consistent with the finding that there is a strong negative correlation (-0.67) between levels of trait neuroticism and opioid peptides in the cerebrospinal fluid (Öst & Hugdahl, 1981). This correlation is even higher (-0.91) when a measure of state anxiety is employed; from this relationship, and the well-established relationship between analgesia and CNS opioids it is possible to argue that low levels of opioids in the brain of neurotics may make them more susceptible to incubation effects. This point, and the literature relevant to it, are both dealt with in much more detail by Eysenck and Kelley (1987).

Another important peptide hormone is adrenocorticotrophin (ACTH). Whereas opioids dampen neuronal excitation, cholinergic and noradrenergic turnover rates, and behavioral performance in aversive conditioning, ACTH has the opposite effect (Bertolini and Gessa, 1981; Chorney & Redmond, 1983; Markey & Sze, 1984; Redmond, and Huang, 1979; Redmond and Krystal, 1984). In the ACTH-mediated incubation effects, which we will discuss in detail shortly, the opioids have a competitive affinity with ACTH for the same receptors. If we block these receptors with the opioid antagonist, naloxone, ACTH (and also vasopressin) loses its capacity to

induce incubation effects or prolong extinction (Concannon, Riccio, Maloney, & McKelvey, 1980; Concannon, Riccio, & McKelvey, 1980; De Vito & Brush, 1984). This reciprocal relationship between ACTH and the opioids, in conjunction with the negative correlation between CSF opioids and anxiety, suggests that ACTH may play an active role in the occurrence of incubation effects.

A tie between experimentally produced changes in emotionality (defecation) as a trait, and the capacity for stress-induced changes in ACTH levels has been recently demonstrated by Armario, Castellanos, & Balasch (1984). This observation can be combined with the findings of Morley (1977), who showed that emotional animals are more likely to show incubation effects. This is also consistent with the suggestion of Eysenck (1979, 1982a) that incubation effects are likely to be stronger in subjects high on neuroticism (N) and introversion (I). The reasons for this suggestion can be deduced from the nature of these two major personality dimensions (Eysenck & Eysenck, 1985), and need not be detailed here. We will now turn to direct experimental evidence that hormones such as ACTH can modulate incubation.

In a series of aversive conditioning studies by Riccio and his students, ACTH or epinephrine injections (which increases ACTH in the rat) were given to rats prior to a one-minute presentation of the CS during a forced-exposure trial following acquisition training. This procedure repeatedly resulted in a large permanent increase in fear of the CS when animals were tested 24 hours later for resistance to extinction without an injection. Mere presentation of the CS or elevation of ACTH levels alone did not produce such effects (Haroutunian & Riccio, 1977, 1979). Kelley (in press) provided an additional control. In this experiment rats were first given three .5 ma foot shocks during two direct placements on the black side of a shuttle-box with a closed guillotine door, and never shocked during two placements on the white side. In the second phase, the different groups of rats were reexposed to the black side and given either (a) a prior .02 mg injection of epinephrine or (b) saline, or (c) an epinephrine injection 5 hours later. The latency to cross from the white to the black side 24 hours later was found to be several-fold longer in the groups given an epinephrine injection shortly before reexposure. The findings thus demonstrate that contiguity between the presence of the fear cue and high levels of hormones is required to produce incubation effects in the rat. The importance of this contiguity has also been demonstrated by other investigators (Righter, Elbertse, & van Riezen, 1975; Weinberger, Gold, & Sternberg, 1984). Whereas ACTH released by acute exogenous injections is one possible explanation of this, it is also possible that epinephrine itself is important (Borrell, De Kloet, Versteeg, & Bohus, 1983; McGaugh, 1983).

The capacity for ACTH to produce incubation effects is supported by an extensive body of evidence from many laboratories showing that ACTH will enhance resistance to extinction. This occurs with a variety of aversive conditioning procedures and with ACTH (4–10), which has CNS but no peripheral endocrine properties (de Wied & Jolles, 1982). There is also evidence that physical levels of ACTH can have a modulatory effect on extinction (Bohas, Endrocrozi, Kissak, Fekete, & de Weid, 1970; Pagano & Lovely, 1972; Van Wimersma Gredanus *et al.*, 1977, 1983). Although these properties of ACTH have also been observed in Pavlovian A conditioning, they are more readily observed with Pavlovian B conditioning. Consistent with this is the well-established observation that sexual behavior is the other motivational system

where ACTH has a robust effect (Bertolini, Fratta, Eena, Munladr, & Serra, 1981, 1984; de Wied & Jolles, 1982). Eysenck (1982a) suggested that sexual drives are the equivalent on the appetitive side to anxiety on the aversive side for the production of incubation effects (i.e., are mediated by Pavlovian B conditioning).

These findings are consistent with other open-field research suggesting that injections of ACTH or its releasing factor (CRF) may be "anxiogenic" in rats (Britton & Britton, 1981, 1982; File & Vellucci, 1978). Some support for this hypothesis also comes from studies on the effect of CRF in rhesus monkeys (Kalin *et al*, 1983a, b); however, these "anxiogenic" properties of CRF and ACTH are only seen in situations that are already fearful. For instance, in the study by Haroutunian and Riccio (1979), exposure to one side of a novel shuttle-box contagious with an ACTH injection was not itself sufficient to produce later spatial avoidance of that side of the apparatus; thus it would appear that an ACTH injection is not, by itself, an aversive UCS. Considerable evidence suggests that the action of ACTH is on the CS, not the UCS or UCR. When ACTH levels are increased by adrenalectomy, the immediate behavioral responses to foot shock (flinch, jerk, vocalization) are not increased (Borrell *et al.*, 1983). In addition, although reduced open-field ambulation is sometimes observed after adrenalectomy, this is not influenced by injections of dexamethasone that should reduce the ACTH levels. Similarly, effects on exploratory behavior are not reliably found after injections with ACTH 4–10 (Bohus *et al.*, 1982). The open-field apparatus has been shown to be a potent releaser of fear (Blanchard, Kelley, & Blanchard, 1974) but that might be dependent upon the strain of rats used, which would account for some of the ambiguity in the open-field findings with ACTH (Eysenck & Broadhurst, 1964). Finally, the results of experiments with humans also suggest that injections of CRF or ACTH are not themselves anxiogenic (Beckwith & Sandman, 1978, 1982; Gold *et al.*, 1984). In contrast, the anxiogenic properties of the ACTH in rats are readily observed when a CS for fear is present; then, as we have just seen, ACTH enhances the excitatory properties of the cue.

Another possible explanation is that incubation effects are mediated by selective attention: ACTH and other neuromodulators of anxiety may enhance the capacity of a CS to show an increment in the level of excitatory strength by influencing its associability. Unfortunately, the effects of ACTH and other peptides on blocking and overshadowing—indexes of selective attention in rats—have not as yet been investigated (Mackintosh, 1984); thus direct evidence for this possibility awaits testing. Nevertheless, Beckwith and Sandman (1978, 1982), using reversal learning and intra- and extradimensional shift experiments, have argued that ACTH influences selective attention. These older behavioral assays for selective attention, however, are subject to alternative interpretations (Mackintosh, 1974; Sutherland & Mackintosh, 1971).

This is not the place to continue the detailed discussion of the action of hormones, which could be extended to vasopressin and cortisol (Eysenck & Kelley, 1987), all of which have been shown to influence conditioning and extinction behavior. The main point of this section has been merely to point to the importance of extending the field of search for causes or mechanisms to that of peptides and other neurohormonal factors that have been clearly related in numerous experimental studies to fearful, neurotic types of behavior, conditioning, and learning, and also incubation.

It seems likely that a better understanding of the mediation of Pavlovian conditioning and extinction can be obtained by looking more deeply into the biology of the organism, rather than treating it merely as a black box, as behaviorists are wont to do (Zuriff, 1985.)

IS EXPOSURE A NECESSARY CONDITION FOR FEAR REDUCTION?

The essence of Watson's theory, both in its original form, and as amended by the writer, is the notion that neuroses are the product of Pavlovian conditioning, and cures are mediated by Pavlovian extinction. We have noted in previous sections that the concept of Pavlovian conditioning has changed very much from the original simple connection between primitive sensory impressions and muscle twitches, to the much more sophisticated type of S-S conditioning that forms the basis of neobehaviorism and of dialectical behaviorism (Davey, 1983a, b; Mackintosh, 1984.) In this section we will attempt to look at some of the unifying properties of the theory, and go on to confront the question raised by de Silva and Rachman (1981) of whether exposure is really a necessary condition for fear reduction. We will also consider some of theoretical implications raised by this question for the relation between cognitive and behavioral theories.

Eysenck (1980, 1983, 1985) has suggested that this (modified) conditioning theory of neurosis can explain the major facts that are known about the treatment of neurotic illnesses. These may be summarized as follows. (a) Spontaneous remission, that is, fear reduction without any form of psychiatric treatment, is a fairly regular and very important factor in the improvement or cure of people suffering from neurotic illnesses. (b) Placebo treatment is a very successful means of securing fear/anxiety reduction in neurotic patients. (c) Nonspecific psychotherapeutic intervention is as successful as placebo treatment, and possible slightly more so than spontaneous remission, in securing reduction of fear/anxiety reactions in neurotic patients. (The term *nonspecific* is meant to denote the fact that regardless of the theoretical basis of the therapies in question, they are equally successful, suggesting the lack of relevance of the specific theories on which they may be based.) (d) Psychoanalysis on the whole is no more successful than all other methods of psychotherapy, and may be less so. (e) Psychoanalysis specifically has been found to have frequent negative treatment effects, that is, it increases rather than reduces fear/anxiety reactions (Strupp *et al.*, 1977.)

It is possible to explain all these effects in terms of exposure to the unreinforced CS. To begin with spontaneous remission, it is well known that people suffering from neurotic disorders, but unable to obtain psychiatric treatment, will seek out various ways of alleviating their distress, usually by discussing their problems with parents, priests, friends, or other people whose friendly advice they feel able to count upon. In such discussions they inevitably bring up the problems which confront them, and discuss in detail, often in some sort of hierarchical order, the fears and anxieties, and their causes, that they have encountered. Thus the relevant CSs, in the weakened form demanded by desensitization theory, are encountered in a relaxed setting in which a friendly listener provides additional relaxation. The presence of a friendly

listener is vital according to Zillmann's (1978, 1979, 1984) three-factor theory of emotion, and in particular the excitation-transfer paradigm to which it has given rise. This paradigm applies to potentially unrelated successive emotional reactions, and to emotional reactions solicited by simultaneously present, yet potentially unrelated stimuli.

> In the former case, the paradigm projects the intensification of any emotional reaction that is evoked during the presence of residual sympathetic excitation from antecedent reactions—with some specifiable exceptions. In the latter, it projects the intensification of any emotional reaction by sympathetic excitation due to stimuli other than those that elicited the emotional reaction proper. The paradigm is applicable to all emotional reactions associated with sympathetic dominance in their excitatory component. (Zillmann, 1984, p. 148)

Zillmann's three-factor theory distinguishes between the dispositional, excitatory, and experiental components of emotional behavior, in which the excitatory activity of emotions that are characterized by sympathetic dominance in the autonomic nervous system is largely nonspecific, and hence capable of being subject to Zillmann's additive law.

What is posited, then, is that the relaxing effects of the presence of the friendly observer/listener summates *negatively* with the relatively slight sympathetic arousal produced in the "patient" by his evocation of the feared material, and thus assists in the general desensitization that is the outcome of this mode of exposure. It thus reduces the strength of the CR, as in Figure 3, making extinction more likely, and incubation less so. Clearly the effects will be much less clear-cut and marked for spontaneous remission than it is for properly planned desensitization at the hands of an experienced behavior therapist, because the contingencies are not planned, and are only accidentally likely to be optimal; nevertheless, the general combination of stimuli is similar, even if their sequence is nonoptimal, and consequently reduction in fear/anxiety behavior is to be expected.

Much the same is true of placebo treatments in so far as these usually implicate the evocation of material giving rise to few anxiety reactions in the patients, under relatively relaxing conditions. This is also true of most psychotherapies, where the presence of a friendly therapist, evoking fear/anxiety related material, is very likely to lead to a general situation the effects of which should be similar to desensitization. Thus the theory may account for spontaneous remission, placebo effects, and also the nonspecific effects of the various psychotherapies.

Psychoanalysis presents a particular difficulty because of the established fact that it often leads to negative emotional reactions, that is, to what we would call incubation rather than extinction of fear/anxiety reactions (Strupp *et al.*, 1977). The reasons for this are probably to be found in the well-known fact that psychoanalysts are instructed not to act in such a way as to appear helpful, warm, relaxing, and generally friendly, but rather are expected to preserve a remote and neutral presence, and to act only in an interpretative rather than an advisory manner. If we again use Zillmann's excitation-transfer paradigm, this failure to provide a helpful, relaxing atmosphere would increase the general level of fear/anxiety of the patient, thus leading to incubation rather than extinction of anxiety (Eysenck, 1982a). Individual accounts of these effects strongly suggest the accuracy of this view (Sutherland, 1976; York, 1966). A simple exposure theory therefore seems to be capable, in combination with

Zillmann's excitation-transfer theory, to explain the observed phenomena to a reasonable degree.

We must now turn to the question of whether exposure is a necessary, as well as a sufficient condition for fear/anxiety reduction. De Silva and Rachman (1981) defined the term *exposure* as

> the exposure of the subject to the fear-evoking stimulus, either in real life (*in vivo* exposure) or in fantasy (imaginal exposure). Imaginal exposure . . . consists in planned, sustained and repetitive evocations of images/image sequences of the stimulae in question. Mere thoughts or fleeting images do not constitute imaginal exposure in this sense. Indeed, it is worth noting that subjects who lack the ability to conjure up and maintain detailed and vivid imagery are usually excluded from therapies involving imaginal exposure. (p. 227)

(See Wolpe, 1973, pp. 136–137, Rimm & Masters, 1979, pp. 45–46.)

De Silva and Rachman (1981) argue that

> while in many circumstances exposure may be a *sufficient* condition for fear-reduction, there is no good reason to suppose that exposure is a *necessary* condition for success. Fear reduction that takes place in the absence of such exposures undermines the assumption that exposure is a *necessary* condition. (p. 22)

De Silva and Rachman admit that

> all of the examples [they give] are open to criticisms of one sort or another and we shall draw attention to these, but remain confident that in due course when the controlled experimental analyses are completed, the results will confirm our claim that fear can be reduced even in the absence of exposure. (p. 228)

This quotation from de Silva and Rachman makes it clear that there is no proper experimental evidence for their claim, but merely anecdotal evidence, and it will be shown that even that is not fatal to the interpretation of exposure as a necessary condition for fear/reduction.

The argument begins with a reference to Rachman's (1978) suggestion that there are at least three pathways to the acquisition of fear. The three pathways are (a) direct aversive experiences; (b) vicarious acquisition of fear;* (c) the fears that are induced by the transmission of information. De Silva and Rachman attempt to use the same arguments that were brought to bear in analyzing the acquisition of fear to the question of reduction.

> It is argued that fears can be reduced by direct experiences (such as desensitization), by vicarious exposure to the therapeutic model, and—most important for the present argument—by the transmission of information. It seems to us to be indisputable that a person's fear reactions can be weakened or even eliminated by giving him the information that the fear stimulus or the surrounding circumstances are not dangerous. (p. 228)

De Silva and Rachman refer to "studies in which fearful subjects were successfully treated with cognitive-based techniques that did not include contact with the fear stimuli" (e.g., Meichenbaum, Gilmore, & Fedoravicius, 1971; Weiss, Nelson, & Odom, 1975; Weissberg, 1977). The same point is made by Bandura (1977), implicit

*Vicarious acquisition of fear, and its complement in therapy, modeling, are not necessarily inexplicable in terms of conditioning theory, as Baer and Teguchi (1985) have shown.

in whose theory is the denial of the claim that exposure is a necessary condition for behavior change such as reduction in fear.

I think it is essential to posit the existence of two continua which are involved in fear/anxiety. The first continuum ranges from the postulation of reasonable as opposed to unreasonable fears. It is reasonable to be afraid of torture, disease, or injury, in circumstances that are likely to result in any of these consequences. It is unreasonable to be afraid of spiders (in countries where there are no poisonous ones), of contamination (when quite innocent objects are being touched), or of many other typically neurotic fear/producing stimuli. These are not two classes of stimuli, because intermediate ones can be adduced. To what extent is fear of an atomic war realistic, to what extent neurotic? Fear of dogs, cats, and even squirrels can to some degree be justified in Europe where these animals may be infected with rabies.

Again, fears may be acquired through cognitive learning (as when we are told that another person is suffering from an infectious disease, or that we can be burnt by fire, or that lions and tigers are dangerous animals), or fears can be conditioned through Pavlovian B conditioning. This too is a continuum rather than an either/or type of classification, because cognitive and conditioning methods of fear acquisition may be active in any particular instance. Thus the obsessive-compulsive patient's fear of contamination is partly due to what he has been taught cognitively about the danger of bacteria, and is partly acquired through a process of conditioning. Note the demonstration by Öhman, Dimberg, and Öst (1985) that prepared CSs are very difficult to extinguish by cognitive means, as compared with nonprepared CSs.

As Figure 4 shows, we thus have four categories of fears. Some rational fears are learned, as are some irrational fears; indeed, it is likely that there is some contribution by cognitive learning in all fears. Similarly, it seems likely that even rational fears may have some slight, and often a considerable basis in Pavlovian conditioning. That this must be true is obvious from a consideration that the very data given

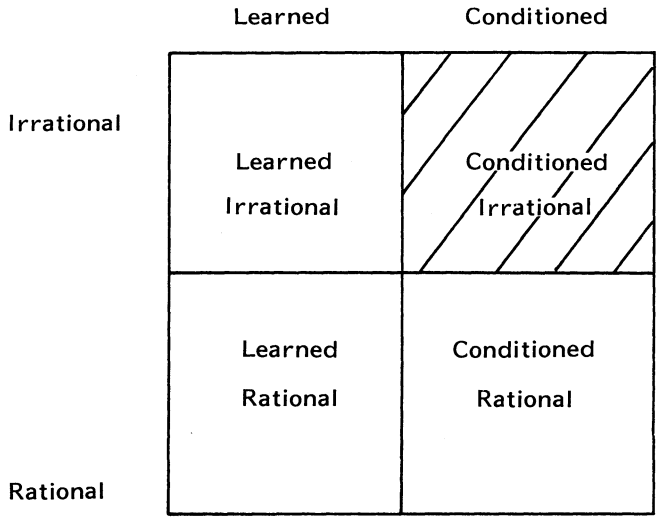

FIGURE 4. Different combinations of learned and conditioned, irrational and rational fears.

us by perception are acquired and organized in terms of conditioning (Taylor, 1962).

In this two-dimensional framework, neurosis is primarily concerned with conditioned irrational fears and anxieties, but the fact that we are dealing with continua means that we will hardly ever encounter a pure example, suggesting the possibility that part of these fears may be acquired through learning, and hence in part extinguished through unlearning. In that sense De Silva and Rachman are undoubtedly correct, but this does not detract in any way from the theory here maintained, namely that specifically neurotic fears are reduced only through exposure. Consider an example. This concerns a Council employee who painted the white lines in the middle of the road. He was hit from behind by a car and injured; he developed fears which were eliminated by behavior therapy (desensitization) to enable him to go back to his job. The success of the therapy was short-lived, however, as he was run down again by another car. He was subjected to desensitization a second time, again successfully, but was finally run over again a third time! We considered it unethical to continue the treatment, as clearly his fears were only part conditioned and irrational, but in large measure also learned and rational. This combination is not unusual, and may be particularly applicable to the studies adduced by De Silva and Rachman as supporting their view.

A proper experimental study of the hypothesis that exposure is not necessary for fear/anxiety reduction, in order to contradict the theory here advocated, would have to show that it dealt with conditioned irrational fears, and did not capitalize on the learned and rational parts of those fears. It is not entirely irrational to be afraid of public speaking or snakes, and hence it seems that the examples given by De Silva and Rachman are contaminated, so that the possible fear reduction through learning may only have affected the learned rational part of the total fear. The position taken by De Silva and Rachman is not necessarily false, but the evidence supporting it is rather insufficient.

The same is true of another example given by them, namely a study by Marks, Gelder, and Edwards (1968) using hypnosis. The hypnotic treatment contained no element of exposure to the feared stimuli, but it did contain relaxation and other general fear/anxiety reducing elements, and in line with Zillmann's theory, outlined earlier, this might reduce the level of anxiety below the critical points (Eysenck, 1982).

One further point that remains to be discussed is the degree to which cognitive factors are opposed to a behavioristic theory of the kind here adopted. Such an opposition would certainly exist in relation to the old-fashioned S-R type of theory adopted by Watson, but not in relation to the more modern S-S type theory (Mackintosh, 1984). The relationship is between stimulus and stimulus, and where, as Pavlov emphasized, words can be used as conditioned stimuli as well as conditioned responses, there is no contradiction involved in explaining results such as those of Wilson (1968), who showed that conditioned responses to a given stimulus could be reversed by suitable instruction (see also Bridger & Mandel, 1964). An even earlier example is Miller's (1935) demonstration that the psychological response could be conditioned to a cognitive stimulus. Miller administered electric shocks to subjects when the letter *T* but not the number *4* was read out aloud, and then instructed

subjects to think *T* and *4*, alternately, in a series of trials. Galvanic skin responses occurred when the subject thought *T*, but not when they thought *4*.

To say all this is not to suggest that exposure is indeed necessary for fear/anxiety reduction, even in the limited sense of conditioned irrational fear/anxiety reduction. It is merely maintained that the evidence against this view is not very strong, and does not take into account considerations that may be vital in assessing the relevance of the studies quoted by the critics. Admittedly it will be difficult to conduct experiments that can establish the reduction of such fears without exposure, but this difficulty should not be allowed to permit the suggestion that the deed had already been done. As far as the existing evidence is concerned, it is robustly in favor of the view that exposure is much the most important, and may be a necessary condition for fear/anxiety reduction. This conclusion may not apply to the same extent to learned fears, whether irrational or rational; much research remains to be done to clear up this particular question.

SUMMARY AND CONCLUSIONS

It may be useful to repeat what is, and what is not, asserted in this chapter. It is asserted that learning theory, and particular modern principles of conditioning and extinction, are basic to the acquisition and treatment of neurotic disorders. It is not asserted that in particular cases other factors may not be of considerable importance, and may be used to help or hinder the development of a proper treatment procedure. However, the principles of conditioning are fundamental in any viable theory of neurosis.

It is not asserted that cognitive therapy (e.g., Hoffman, 1984) constitutes a separate, antagonistic framework of theory and practice contrasted with behavior therapy and the underlying theory of conditioning. It is asserted that modern learning theory, as outlined for instance in the chapter by Dickinson in this volume, takes into account cognitive processes and principles, and combines these in a meaningful manner within learning theory. Information processing (Foa & Kozak, 1986) is an essential part of modern learning theory, as so defined, and does not require us to posit a separate cognitive psychology, separate and apart from theories of learning.

It is not asserted that all human behavior can be reduced to principles derived from animal behavior; no such complete reductionism is intended. It is asserted, however, that certain types of behavior, particularly neurotic behaviors, do find a very close analogue in animal behavior, and that hence the study of conditioning and learning in animals is of fundamental importance for an understanding of the processes mediating the acquisition of neurotic behavior in humans, and its extinction. The Rachman and Hodgson studies of obsessions and compulsions (1980) would seem to establish the correctness of this view once and for all.

It is not asserted that Watsonian theories of neurosis and treatment, or the Hull-Spence type of learning theory, should govern our thinking about the relationship between conditioning and neurosis. It is asserted that these writers laid the foundation for a better understanding of the acquisition of neurotic disorders, and

pioneered an understanding of the principles on which any treatment must be based. Our theories may be in error, but as Francis Bacon remarked, "truth comes out of error more readily than out of confusion."

It is not only our general theory of learning and conditioning that has changed dramatically from an S-R to an S-S theory (or better still, to an S-S-R theory!), but also certain specific applications of learning theory to neurosis. Concepts such as that of incubation of fear seem to be essential if we are to map the facts of neurosis onto the theories of conditioning and learning. It will be a long time before this is done in such a way as to satisfy all our theoretical and practical demands, but already the fit is better than to any other existing theory.

How, in fact, shall we evaluate a theory? To quote Mao Tse Tung: "The only standard by which truth can be assessed is in its practical results." It has been noted in many discussions of the effects of psychotherapy that "all have won, and all must have prizes," a conclusion typical of the Alice in Wonderland state of affairs prevailing in psychotherapeutic research. If indeed all different psychotherapeutic methods, as well as placebo methods, work equally well, then clearly none of the specific theories giving rise to these many different methods of psychotherapy can have any specific value; such effects as are seen must be due either to nonspecific factors, such as suggestion, prestige, friendly human interaction, etc., or as suggested in this chapter, to unintended but nevertheless present Pavlovian extinction. The fact that behavior therapy is surely more effective than psychotherapy or placebo treatment in many cases is clear evidence that specific factors are involved here, and hence that there must be some measure of truth to the principles on which behavior therapy is based.

If that be so, we may perhaps here quote Kurt Lewin's famous saying: "Nothing is more practical than a good theory." Progress in behavior therapy depends crucially on improving and updating our theories in the light of ongoing research, using these theories to improve our method of treatment and checking their efficacy against the effects of treatment. Treatment may be regarded as an extension of laboratory research. We cannot test our theories concerning strong emotions very easily in contrived laboratory settings, for ethical and humane reasons. Neurotic fears present us with a testing bed for predictions derived from our theories. This reciprocal process, laboratory helping clinic, and clinic assisting laboratory, is perhaps the most important outcome of the advance of the behavior therapy movement.

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