

A COMPARISON OF PROGRESSIVE RELAXATION, DIAZEPAM, AND PLACEBO
DRUG IN THE REDUCTION OF ANXIETY, AND AS ADJUNCTS IN THE
TREATMENT OF SMALL ANIMAL PHOBICS BY FLOODING

C

Rodney John

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ABSTRACT

A COMPARISON OF PROGRESSIVE RELAXATION, DIAZEPAM, AND PLACEBO DRUG IN THE REDUCTION OF ANXIETY, AND AS ADJUNCTS IN THE TREATMENT OF SMALL ANIMAL PHOBICS BY FLOODING

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The present study investigated two related but separate questions. The first question looked at the comparative efficacy of progressive relaxation, valium, and valium placebo in the reduction of anxiety caused by exposure to a phobic stimulus. The second question investigated, compared the treatment of small animal phobics under one of five conditions; (1) Progressive Relaxation assisted exposure, (2) Valium 5mg assisted exposure, (3) Valium 7.5mg assisted exposure, (4) Valium placebo assisted exposure and (5) Exposure without anti-anxiety adjunct, with a No-Treatment Control condition.

Frontalis muscle tension, pulse rate, systolic blood pressure, diastolic blood pressure and self report were used as the indices of anxiety, while slides of the feared animal (rat, spider or snake) were used as the phobic stimulus. For the first question, the results indicated that all the anti-anxiety adjuncts under different conditions were effective in reducing anxiety. When the question of reduction of phobic behaviour was considered, the results indicated that while all the treatments were effective in reducing phobic avoidance, when compared to the no-treatment control group, indications are that treatment with an anti-anxiety adjunct can enhance this reduction.

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Table of Contents

	Page
Introduction.....	1
Method.....	44
Results.....	52
Discussion.....	106
Reference Notes.....	133
References.....	134
Appendices	
A. Subjective Rating Scale.....	170
B. Self Analysis Form.....	171
C. Fear Inventory.....	177
D. What Are You Afraid Of?.....	182
E. Checklist Item:Behavioural Avoidance Test.....	184
F. Demographic Information.....	185
G. Subjects Instructions 1	186
H. Subjects Instructions 2.....	187
I. Consent Form.....	189
J. Modified Jacobsonian Instructions for Progressive Relaxation.....	190

List of Figures.

	Page
1. Pre-post measures on the Behavioural Avoidance Test across the six groups.....	57
2. Pre-post measures on Self Rating of Phobic Avoidance across the six groups.....	61
3. Pre-post measures on the Fear Survey Schedule across the six groups.....	63
4. Pre-post measures on the What Are You Afraid Of, across the six groups.....	67
5. Pre-post measures on the I.P.A.T. across the six groups.....	68
6. Readings for self report obtained from treatment sessions 1-6 across the five treatment groups.....	72
7. Readings for self report obtained from treatment sessions 1-6 across the five treatment groups.....	74
8. Frontalis E.M.G. data showing the mean scores at four times of reading over six treatment sessions across the five treatment groups.....	81
9. Pulse Rate data showing the mean scores for four times of reading over six treatment sessions across the five treatment groups.....	84
10. Systolic Blood Pressure data showing the mean scores for times of reading over six treatment sessions across the five treatment groups.....	87
11. Systolic Blood Pressure showing the average of four readings over six sessions across the five treatment groups.....	90

List of Tables

Table		Page
1.	Means and Standard Deviations for Screening Data Across Six Groups.....	53
2.	Summary Analysis of Variance Table for Dependent Measures Obtained on Six Groups During Initial Screening Session.....	54
3.	Analysis of Variance (Balanova) on the B.A.T. Pre-Test to Post-Test Data Obtained in the Experiment.....	55
4.	Mean Difference Scores on the Behavioural Avoidance Test Obtained in the Experiment Across the Six Groups, for Pre and Post-Tests.....	58
5.	Analysis of Variance (Balanova) on the Self Rating of Phobic Avoidance, Pre-Test to Post-Test Data Obtained in the Experiment Across the Six Groups.....	60
6.	Analysis of Variance (Balanova) on the Fear Survey Schedule Pre-Test to Post-Test Data Obtained in the Experiment Across the Six Groups..	62
7.	Mean Difference Scores on the Fear Survey Schedule Test Obtained in Experiment Across the Six Groups, for Pre and Post-Tests.....	64
8.	Analysis of Variance (Balanova) on the W.A.Y.A.O. Pre-Test to Post-Test Data Obtained in the Experiment Across the Six Groups.....	66
9.	Analysis of Variance (Balanova) on the I.P.A.T. Scores Obtained in the Experiment Across the Six Groups.....	66
10.	Analysis of Variance (Balanova) on the Self Report Data (Composite Scores) from Sessions 1-6, Across the Five Treatment Groups.....	70
11.	Analysis of Variance (Balanova) on the Self Report Data (Composite Scores) from Sessions 3-6 Across the Five Treatment Groups.....	76

Table	Page
12. Mean Self Report Scores Obtained in Experiment Across the Five Treatment Groups Over the First Six Treatment Sessions.....	78
13. Mean of Self Report Scores at Time of Reading Obtained in Experiment Across Five Treatment Groups During Sessions 3-6.....	78
14. Analysis of Variance (Balanova) on the Frontalis E.M.G. Data (Composite Scores) Obtained in the Experiment.....	80
15. Analysis of Variance (Balanova) on the Pulse Rate Data (Composite Scores) Obtained in the Experiment.....	82
16. Analysis of Variance (Balanova) on the Systolic Blood Pressure Data (Composite Scores) Obtained in the Experiment.....	86
17. Mean Systolic Blood Pressure Scores Obtained Across Five Treatment Groups for Time of Reading Obtained in Sessions 1-6.....	88
18. Analysis of Variance (Balanova) on the Diastolic Blood Pressure Data (Composite Scores) Obtained in the Experiment.....	92
19. Analysis of Variance (Balanova) on the Self Report (Pre-Post) Data Obtained in Session 7.....	94
20. Analysis of Variance (Balanova) on the Frontalis E.M.G. Data Obtained in Session 7.....	94
21. Analysis of Variance (Balanova) on the Pulse Rate (Pre-Post) Data Obtained in Session 7.....	95
22. Analysis of Variance (Balanova) on the Systolic Blood Pressure (Pre-Post) Data Obtained in Session 7.....	95
23. Analysis of Variance (Balanova) on the Diastolic Blood Pressure (Pre-Post) Data Obtained in Session 7.....	96

cont'd

Table	Page
24. Mean Pre-Post Scores on Frontalis E.M.G., Pulse Rate and Systolic Blood Pressure Obtained in Experiment During Session 7.....	97
25. Analysis of Variance (Balanova) on the Self Report Data Obtained in Session 8.....	99
26. Analysis of Variance (Balanova) on the Frontalis E.M.G. (Pre-Post) Data Obtained in Session 8.....	99
27. Analysis of Variance (Balanova) on the Pulse Rate (Pre-Post) Data Obtained in Session 8.....	100
28. Analysis of Variance (Balanova) on the Systolic Blood Pressure (Pre-Post) Data Obtained in Session 8.....	100
29. Analysis of Variance (Balanova) on the Diastolic Blood Pressure (Pre-Post) Data Obtained in Session 8.....	101
30. Mean Difference Scores on Frontalis E.M.G., Pulse Rate and Systolic Blood Pressure Obtained in Experiment During Session 8.....	102
31. Correlations Between Subjective Ratings of Anxiety and Four Autonomic Measures from Screening and Post-Test Data Obtained Across Fifty Four Subjects in Experiment	104
32. Correlations Between Subjective Ratings of Anxiety and Autonomic Indices Across Six Groups for Post-Test Data.....	105

HISTORICAL CONTEXT

Anxiety as a fundamental human emotion has been studied with increasing regularity in both the psychological and psychiatric literature since the 1890's (Spielberger, 1972). Since the late 1950's, it has been extensively investigated experimentally (Spielberger & Diaz-Guerrero, 1976); nevertheless, there is still little agreement among theoreticians as to the nature of the phenomenon, and no single acceptable definition of anxiety exists.

Historically, there have been many different theoretical approaches to the study of anxiety. Three of the more important are the Psychoanalytic approach, the Physiological approach, and the Learning Theory approach. A brief summary of these three approaches will be outlined below, followed by a subsequent section in which all are evaluated critically.

Psychoanalytic Approach. Freud (1926, 1936) was one of the first major theorists to tackle the question of anxiety systematically (Gilliland, 1979). He saw the following as the three primary attributes of anxiety: (1) a specific unpleasurable quality; (2) efferent or discharge phenomena, and (3) the perception of these (Freud, 1936). It should be noted that this notion of anxiety is quite similar to that of some modern theorists e.g. Spielberger, 1976).

To Freud, anxiety could arise from two sources: the first

involuntary and automatic is a reaction to a situation that presented a danger to the psychic economy, (for an extended treatment see Freud, 1936); the second is anxiety produced by the ego when such a situation merely threatened (Freud, 1936). These led to what he termed "true-anxiety" - anxiety in regard to a known danger, and "neurotic-anxiety" - anxiety in regard to a danger which is not known. While his theoretical formulations were certainly important, Freud's major contribution lay in the fact that his was one of the first attempts to elucidate the meaning of anxiety within the context of psychological theory (Spielberger, 1972). This formulation was elaborated further by Rank (1929), Fenichel (1945) and other psycho-analytic theorists.

Physiological Approaches. Physiological theorizing on anxiety can be traced to Canon's repudiation of the James-Lange theory of emotion. For James (1884) and Lange (1922), the experience of physiological phenomena was said to be synonymous with emotion. Canon (1927, 1929), however cast doubt on the James-Lange Theory by demonstrating that animals surgically deprived of all autonomic reactivity were still capable of manifesting emotional behaviour. He then attempted a re-definition of emotional behaviour and came to regard anxiety as "an expression of a disruption of a homeostatic equilibrium, which could be threatened; broken down, or reestablished at a new set point." (Canon, 1927, p. 120)

Despite this auspicious start, research into the physiologi-

cal parameters of anxiety did not become noteworthy until the 1950's. The resurgence of this line of research can be traced to the enunciation of the concept of arousal or activation theory, which was initiated by Lindsley (1952, 1957) and made popular by Duffy (1957), Malmo (1959), and Schacter and Singer (1961). According to the arousal concept, physiological reactions are simply a reflection of general arousal or activation. The subjective experience of a specific emotion on the other hand exists solely on the cognitive or psychological level.

In order to demonstrate this, Duffy (1957) in a series of experiments appeared to show that difference in arousal, as measured by the E.E.G., and muscle tension were correlated with other forms of responsiveness. Similarly, Malmo (1957) conceived of anxiety as "a disease of over-arousal" and saw physiological measures such as palmar skin conductance, E.E.G.'s, skeletal muscle tension, heart rate, blood pressure and respiration as reliable indicators of arousal and hence of anxiety.

The arousal concept, as proposed, suggested that clinicians and researchers could avoid subjective reports and measure anxiety directly through physiological indices. This was quite an attractive proposition, and was the stimulus for a great deal of research. For example Wenger (1957), measured anxiety in terms of blood pressure change and sweat gland activity. Malmo and Shagass (1952) looked at levels of sympathetic nervous system activity and adrenocortical arousal. Lader and Wing (1964) used palmar skin conductance levels,

while Kelly (1966), and Kelly and Walter (1968), used forearm blood flow as their anxiety measures. However as will be noted later the promise of arousal theory did not hold up.

Learning Theory Approaches. Almost concurrently with the work of the physiological researchers, learning theorists were attempting to explain the anxiety phenomenon. The beginning of this line of research can be traced to a publication by Mowrer (1939) entitled "A Stimulus Response Analysis of Anxiety as a Reinforcing Agent". This paper put forward a view of anxiety as a conditioned 'pain' reaction in which anxiety was elicited by signals that had previously been experienced in conjunction with 'pain' or injury. Later Mowrer (1961) reviewed this notion and proposed that anxiety could be viewed as that which motivated or-arouses the organism to action. A second and equally important aspect is that the anxiety also acts as a reinforcing agent in that responses which reduced the anxiety state were strengthened and maintained. In summary, Mowrer (1961) proposed a two-factor theory. Anxiety once acquired, could be used as a drive to motivate instrumental learning. The second stage was that a sudden reduction in the strength of anxiety could be used as a reward to reinforce such learning.

In yet another learning theory approach, Skinner (1953), defined anxiety as the behaviour pattern observed during the interval between a warning signal and an unavoidable and strong aversive stimulus. To Skinner (1953) almost every strong aversive stimulus is preceded by a characteristic or neutral stimulus which may come to

generate anxiety. Since conditioning may take place as the result of one pairing of these stimuli, a single aversive event may result in a previously neutral stimulus (e.g. a flash of light) acquiring properties which can thereafter generate an anxiety state (Skinner, 1953 p. 179). Thus, for Skinner, anxiety is more a consequence of some behavioural event than a cause of the behaviour, and any therapeutic attempt to reduce the effects of anxiety must operate upon the behaviour, not upon any intervening state.

The learning theorist whose ideas probably stimulated the most clinical research in the area of anxiety is Wolpe. When one looks at Wolpe's contribution, it is noted that he believes that anxiety is central to most neuroses (Wolpe, 1966). He defined anxiety as an emotional habit, i.e. the autonomic response patterns that are characteristically part of the organism's response to noxious stimulation.

These examples, while not exhaustive, demonstrate the variability in theoretical approaches to the concept of anxiety. While each area contributed to the overall understanding of the concept, individually they have serious limitations, as will be shown at a later point.

Problems in Definition. The psychoanalytic formulations of anxiety appear limited for two reasons. Firstly, they tend to be qualitative e.g., Rank's formulation of separation anxiety (Rank, 1929) or Freud's conceptualization of oral anxiety, anxiety of the Id, and castration anxiety (Freud, 1936). The nature of such conceptualizations make them

difficult to utilize in diagnosis or treatment. Furthermore, there is no question of being able to measure them and thus they are impossible to test empirically. Secondly, in the psychoanalytic tradition, even though Freud spoke of "true anxiety" vs. "neurotic anxiety" (Freud, 1936), empirically, he did not differentiate between them. This lack of differentiation contributed to the confounding of the idea of anxiety as process or neurotic anxiety, and anxiety as a reaction, or 'true' anxiety.

In looking at the physiological approaches to anxiety, one also finds several problems. Firstly, the expected precision in the measurement of anxiety did not materialize (Lacey, Bateman and Van Helm, 1953; Engel, 1960). Furthermore, while a centrally mediated concept of arousal would predict high intercorrelations among autonomic measures, this has not been the case; most studies report low intercorrelations of the various physiological indices across subjects (Ax, 1953; Lacey, 1967; Lange, 1968; Terry, 1953). Secondly, most of the studies use different measures. For example forearm blood flow (Kelly, 1968; Kelly & Walter, 1966), galvanic skin responses (Beam, 1955; Bitterman & Holtzman, 1952; Lader & Wing, 1964), blood pressure, and heart rate (Lesse, 1970; Malmö & Shagass, 1952), and skeletal muscle activity (Malmö & Smith, 1955; Sainsbury, 1964), are representative of measures that have been employed. With such a diversity of measures cross comparison of studies becomes difficult if not impossible. Another problem encountered involves the subject population used. Often used were patients who either had previously

been or currently were diagnosed as having an anxiety reaction, the diagnosis being made on criteria that did not include physiological responses (e.g., Malmö & Shagass, 1952; Kelly, 1966; Lader & Wing, 1964). In a similar manner other researchers used patients who had been designated as neurotics (e.g., Spence & Taylor, 1952; Finesinger, Sutherland & McQuire, 1942). The extent to which the measures obtained reflect chronic physiological responses of the specific population, and the extent to which they may represent acute anxiety responses of a reactive nature, is thus not clear.

When we look at the learning theorists, we find that initially they tended to focus on the behavioural parameters of anxiety, and ignored the physiological components (O'Brien & Borkovec, 1977). This position was facilitated by the fact that for a number of years the major problem investigated was phobic behaviour, and the bulk of this research was performed on predominantly analogue populations (Bernstein & Paul, 1971; Borkovec, 1973; Mathews, 1978; Rosen, 1975). However, even when learning theorists began utilizing physiological measures in their research (Lader & Mathews, 1968), these tended generally to be single measures (Gauthier & Marshall, 1977; Reinking & Kohl, 1975; Thomas & Rapp, 1977). The problem here is that these measures, an example of which is the galvanic skin response, reflect general arousal, which makes them highly questionable as adequate measures of anxiety (Hodgson & Rachman, 1974; Leitenberg, Agras, Butz & Wincze, 1971; Van Egeren, 1971).

It can be ~~noted~~ therefore, that within anxiety research

there are widely differing major theoretical orientations. In addition these orientations tend to direct anxiety research in widely differing directions.

Contemporary Approaches. A look at the current literature on anxiety indicates that fundamentally the situation has not changed very much and widely divergent definitions of anxiety still exist. These definitions range from being broad generalizations, which reflect the position of major theorists, to narrower definitions which reflect a particular way of conceptualizing the phenomenon. A sample of these various conceptualizations will be subsequently presented.

An example of a definition of anxiety by a major theorist is that of Spielberger's (1972, p. 10). He sees anxiety as "a transitory emotional state" (A-state) which consists of feelings of apprehension and tension, and heightened activity of the autonomic nervous system. In this approach, it is assumed that A states vary in intensity depending upon the individual. This definition can be contrasted with one by Lavalee (1977, p. 65) who conceptualizes anxiety as "a persistent or recurrent state of apprehension or non-specific fear accompanied by physiological signs of excitation such as palpitations, tachycardia, tremor or dizziness." In addition subjects were required to have a sten rating on the I.P.A.T., (Cattell, 1957) of 7 or more. These two definitions are obviously not identical, though they refer to the same phenomenon.

Recent advances in the biochemistry of anxiety, have led some researchers to attempt to formulate definitions of anxiety from

what can be broadly called, biological perspectives. In one of these approaches, Pitts (1969) concludes that anxiety symptoms result from a high concentration of lactate ion and that anxiety attacks can be induced in neurotic patients by lactate infusion. Similarly Barratt (1972) attempting a basically neuropsychological model of anxiety sees it related to the hypothalamic-hypophyseal control of endocrine functions, which result in A.N.S. changes and other somatic changes felt by the anxious person; and (2) the cognitive awareness of tenseness resulting from non-specific reticular control of cortical activity.

In yet other approaches, Epstein (1972, p. 311) in what is basically a revival of the arousal concept defines anxiety as "an acutely unpleasant state of diffuse arousal following the perception of threat." Grinker (1966) conceptualises anxiety in terms of stress. Thus he states that there is very little "free anxiety" since anxiety usually is tied to whatever stress the individual is susceptible to.

These formulations by no means exhaust the current available definitions of anxiety, but only serve to indicate the diversity of approaches. At this point in time and at the current state of knowledge the simplest, the most parsimonious, and at the same time the most comprehensive view of anxiety which has been developed defines it as a response occurring in three systems, verbal-cognitive, overt-motor, and the physiological-somatic (Borkovec, 1976; Lang, 1971). According to this definition of anxiety, while these systems often show synchrony, they are sometimes independent (Borkovec, 1972; Lang, 1971; Leitenberg, Agras, Butz & Wincze, 1971; Mathews, 1971;

Rachman & Hodgson, 1974)). Such a definition, based as it is on what are fundamentally gross categories, attests to the general lack of information as to precisely what anxiety is.

Measurement of Anxiety. Historically, the prime criterion in the assessment of anxiety was the patient's self report, and even though it is still widely used (Lick & Katkin, 1976), over the years attempts have been made to establish more precise measures of anxiety. One approach was through the development of anxiety inventories. Of these, one of the first developed was the Hildreth Feeling and Attitude Battery (Hildreth, 1946). This was a set of scales that measured various moods and affect states. Hamilton (1959) then developed the Hamilton Inventory. This was designed for the rating of anxiety neurosis as a syndrome, and therefore covers a variety of responses. These included fears, insomnia, autonomic response symptoms, mood level, etc. While these scales were widely used, they are basically clinical scales and contributed little to the area of research.

However, a further stimulus to anxiety research came from the development of Taylor's Manifest Anxiety Scale (Taylor, 1951, 1953) and Mandler and Sarason's Test Anxiety Questionnaire (Sarason & Mandler, 1952). These represented the first of a number of psychometric scales designed to assess anxiety both clinically and in research (e.g., Endler, Hunt & Rosenstein, 1962; Freeman, 1953; McReynolds, 1968).

Between 1961 and 1966, the trait-state categorization of

anxiety was formulated by Cattell (Cattell, 1966, Cattell & Scheier, 1961) and later affirmed by Spielberger (1966, 1971). Generally, trait anxiety (A-trait) refers to relatively stable individual differences in anxiety proneness, while state anxiety (A-state) refers to differences in the frequency with which an individual experiences anxiety reactions over time (Spielberger, 1976). This formulation led to the development of scales to measure either A-state anxiety, or A-trait anxiety. Examples of measurement scales of A state anxiety are the Nowlis-Green measures (Nowlis & Green, 1965), the I.P.A.T. 8-Parallel Form Anxiety Battery (Scheier & Cattell, 1960), and Zuckerman's Affect Adjective Check List (Zuckerman, 1960). Examples of A-trait anxiety measures are the Objective Analytic Anxiety Battery (Cattell & Scheier, 1960), the I.P.A.T. (Cattell, 1957), and the Taylor Manifest Anxiety Scale (Taylor, 1951; 1953). In yet further developments, inventories were developed to provide self report assessments of both state and trait anxiety. For example, the Multiple Affect Adjective Check List (Zuckerman & Biase, 1962; Zuckerman & Lubin, 1968), and the State-Trait Anxiety Inventory (Spielberger et al., 1970).

While these inventories have been proliferating and are extensively utilised, there has recently been a trend in research studies on anxiety to employ simple self-rating measures. These are used either alone or in conjunction with other more extensive inventories. This is a reflection of the fact that the question how do you feel, is still the best measure of anxiety. These measures use

scales ranging from 0-5 to 0-15 (Marks, 1972; Ost, 1978; Kelly, 1978; Trudel, 1979; Marzillies, Carroll & Newland, 1979; Mathews, 1976) or even from 0-100 (Hiebert & Fitzsimmons, 1978), when they are often called subjective units of discomfort (S.U.D.S.) (Solpe, 1973).

On the other hand, some researchers still attempt to measure anxiety by employing physiological indices. These include: heart rate, skin conductance, respiration, frontalis E.M.G. and other measures (Mathews et al., 1976; Marks, 1972; Odum, 1978; Ost, 1978; Gatchell, 1978; Kelly, 1966). Then again as a further addition to the above approaches, independent therapists' ratings are also employed in anxiety measurement (Chambless & Foa, 1979; Kelly et al., 1970; Gatchell et al., 1979; O'Brien & Borkovec, 1977; Zenmore, 1975).

In summary, measurement techniques utilized in assessing anxiety focus on the cognitive, physiological or the behavioural aspect of the phenomenon, and researchers employ these measures either singly or in combination. However, one is still left with the question of efficacy and accuracy in measurement of anxiety, and the current tendency to use multiple measures has not led to increased precision in measurement.

Problems in Measurement. In spite of the proliferation of assessment techniques, there are still many unanswered questions which are caused in large part by the measures themselves. For instance, in examining the state-trait dimension, while undoubtedly a strong case can be made for differentiating between anxiety as state (situational anxiety) versus anxiety as trait (chronic anxiety), the extent to which

the present measures are valid indicators of these dimensions is questionable. Thus, Zuckerman (1960) found that both general and specific trait measures were equally predictive of A-state response in an actual classroom situation, while other researchers such as Hodges and Spielberger (1969), Houston and Hodges (1970), and Katkin (1966), have found trait anxiety to be unrelated to autonomic response and performance measures in stressful situations. Furthermore, many of the measures which purport to measure the same dimension of anxiety (e.g., trait anxiety) show little intercorrelations between themselves (Spielberger, Gorsuch & Lushene, 1970).

Upon examining the use of self rating measures, the situation is found to be similarly confused. As already noted, these self-rating measures can range from the use of relatively small unit scales 0-5 to 0-15 (Odum, 1978; Ost, 1978) to larger 0-100 scales (Wolpe, 1973).

The first problem with this variability of range is the difficulty involved in cross comparison of studies using different measures. A second problem is the question of individual idiosyncratic responses. Thus on the same scale, what one individual might rate as mild, another might rate as severe.

When we look at physiological measures, despite the prediction of arousal theory, the situation is no clearer. The first problem here is the question of individual autonomic response to stress. For example, some individuals may respond with increased heart rate and blood pressure but show little change in palmar sweating, while others may show the reverse pattern (Lacey, Bateria & Van Helm, 1953; Engel, 1960).

Secondly, there are numerous factors other than anxiety that affect physiological response. These can range from factors such as sex differences (Biase & Zuckerman, 1967; Lieberman & Lieberman, 1975); age (Kelly, 1966; Kelly, Brown & Shaffer, 1970); differences in habituation rate (Watts, 1975); circadian, seasonal and other cyclic variations (Wenger & Cullen, 1972); to even culture and race (Appley & Trumbull, 1967). Because of this it is not surprising that most studies employing physiological indices report low intercorrelations between the measures across subjects, (e.g., Hokanson, 1976; Shevidy & Kleinman, 1977; Wilson & Wilson, 1970; Blook & Trault, 1977).

A further problem concerns the discrepancies between the physiological and psychological or subjective measures of anxiety. Investigators find little correspondence between concurrent autonomic, verbal, or overt measures of anxiety (Dykman, Ackerman, Galbrecht & Ruse, 1963; John, 1977; Reinking & Kohl, 1975; Schroeder & Rich, 1976). In a recent study specifically designed to investigate this relationship, Morrow and Labrum (1978) compared the physiological parameters of heart rate, systolic and diastolic blood pressures, epinephrine, norepinephrine and Vanillylmandelic acid (V.M.A) with measures on the Taylor Manifest Anxiety Scale (Taylor, 1953), Mood Adjective Check List, and State-Trait Anxiety Inventory (Spielberger et al., 1966; 1970) and Multiple Affect Adjective Check List Zuckerman et al., 1960; 1962; 1964; 1965). The intercorrelation matrix revealed a significant positive pattern of relationships among the four psychological tests, a non-significant positive pattern of relationship among the physio-

logical indices, and a non-significant negative pattern of relationship between the psychological and physiological indices. In summarizing their results, Morrow and Labrum (1978) commented negatively on the advisability of assuming that studies on anxiety which use diverse physiological and psychological measures yield results that may be validly compared.

In attempting to assess these findings, one possible conclusion is that there is not one anxiety state but many, each with differing characteristics. Indeed, researchers appear to be moving in that direction by conceptualizing different types of anxiety states subsumed under broad headings. For example, Noyes, Clancy, Crowe, Hoenk, and Shymen (1978) conceive of anxiety neurosis as a syndrome which is characterized by either (a) relatively persistent generalized anxiety, nervousness or apprehension, or (b) anxiety occurring at times other than during marked physical exertion of life-threatening circumstances. In a similar manner, Lofft and Demars (1974) use a two-dimensional approach to what they term "psychoneurosis." For an overall definition, patients had to present more than one of the following specific symptoms supposedly characteristic of psychoneurosis: anxiety, agitation, tension, apprehension, mixed anxiety-depression and sleep, disturbances. However, within this broad formulation they presented two diagnostic categories; one of anxiety and/or depressive neurosis, and the other depersonalization neurosis.

However, the fundamental question of what exactly is anxiety
or the anxiety state still remains, and rather than clarifying the
issues, much of the theorizing about anxiety would appear to have
served only to complicate the issue further.

TREATMENT OF ANXIETY/PHOBIAS

So far in the evaluation of the anxiety literature the phenomenon has been found to be ill-defined and poorly measured. In looking at treatment for anxiety the picture is very much the same. The three most widely used approaches to the treatment of anxiety are the psychoanalytic-psychotherapeutic, the behavioural, and the pharmacological. Though treatment may involve a combination of all or some of these approaches, they are nevertheless distinct enough to be considered separately.

The Psychoanalytic Psychotherapeutic Approach. This approach is by far the most important in terms of theorizing on anxiety both as a theoretical construct, and as a psychopathological state. This is particularly so since anxiety as a construct has been seen as the common ingredient in many clinical syndromes (Gilliland, 1979; Wolpe, 1979). Nevertheless, the area has produced comparatively little by way of empirical research, and this makes evaluation of the area difficult, particularly in terms of treatment. Given this it will be dealt with briefly in this paper.

Psychotherapy, literally treatment of the psyche, is essentially a talking treatment. Of the systems of psychotherapy practised the psychoanalytic method is probably the best known (Chessick, 1969, 1974; Katzenelbogen, 1958). In the psychoanalytic approach, the essence of therapy is to bring into consciousness unconscious disturbed feelings caused by repressed childhood conflicts. This is achieved

through the gradual resolution of resistance. The techniques for achieving this included free association, dream analysis, and working through transference neurosis (Wolberg, 1967; Katzenelbogen, 1958; Chessick, 1974).

Apart from psychoanalysis, there are numerous other psychotherapeutic approaches employing various techniques which are used in the reduction of anxiety. Harper (1959) documented some 36 systems of psychotherapy. His list was not exhaustive even then (1959) and since then many new therapies have emerged (Davanloo, 1978). This proliferation of therapy systems has prompted some researchers to attempt comparative evaluations of differing therapeutic approaches (Davidson, Davidson & Freedland, 1977; Di Loreto, 1971). However to date, the bulk of the research in psychotherapy involves investigations into patient and therapist variables, and outcome studies (Kiesler, 1973; Meltzoff, 1970; Stieper & Wiener, 1965; Strupp, 1973).

Behaviour Therapy. Unlike the psychoanalytic-psychotherapeutic approach behaviour therapy has been more extensively researched and can be examined in much greater detail from an experimental viewpoint. For example, phobic anxiety has been one area extensively investigated, because by definition if not in fact those suffering from this condition manifest their anxiety or fear in a manner which permits fairly precise delineation (McAllister & Olley, 1975).

The term behaviour therapy was popularized through the work of Wolpe (1958, 1973) and Eysenck (1960, 1964). Behaviour therapists assume that the patient's maladaptive behaviour which is itself termed

the "neurosis" represents efforts to reduce anxiety by escape, avoidance and other means (Sloane, 1975).

While behaviour therapy borrows heavily from the learning theories, there is no one single approach to treatment, instead a wide range of techniques are practised. This is a sufficiently large problem that it has recently led Wolpe (1976) to suggest that since there is no one "modern learning theory" it is meaningless to define behaviour therapy in terms of such a theory. Nevertheless, of the techniques available one of the most widely used is systemic desensitization. In this procedure which was developed by Wolpe (1958), there are two distinct stages. Firstly a physiological state which is assumed to be inhibitory of anxiety is induced in the patient through training in muscle relaxation (it must be noted here however that other states which may inhibit anxiety include assertive responses (Wolpe, 1958) or sexual responses (Wolpe, 1958; Brown, 1978; La Femina, 1979). The second stage involves exposing the patient to the anxiety arousing stimulus using a gradual approach, while simultaneously having them undergo relaxation. With repeated exposure, the stimulus progressively loses its ability to evoke anxiety (Wolpe, 1958; 1973).

Another approach to anxiety reduction involves the use of relaxation training by itself. Now relaxation within the systematic desensitization procedure has generally been induced through a modification of Jacobsonian progressive muscle relaxation (Wolpe, 1973; Amit & Sutherland, 1974) although drugs (Poole & Csillag, 1977; Wolpe, 1973) and hypnosis (Rubin, 1972; Scott, 1970), have also been used. However

relaxation training by itself has been found to be effective in reducing anxiety. This has been achieved either through progressive muscular relaxation as originally devised by Jacobson (1938), or with later modifications of this technique (e.g., Bernstein & Borkovec, 1973; Reinking & Kohl, 1975; Schandler & Grings, 1976), through autogenics training (Schultz & Luthe, 1959; Wallace & Benson, 1972) or through biofeedback training (e.g., Budzynski & Stoyva, 1969; Schwartz, 1974).

In marked contrast to the desensitization-relaxation approach reduction is the flooding-implosion approach. Flooding can be traced to the work of Malleon (1959) and Stampfl (1964) and is similar to the "paradoxical intention" therapy of existential psychiatrists like Frankl (1960). This technique involves the evocation of high levels of anxiety in the patient, either in imagination, when it is called implosive therapy (Stampfl & Levis, 1967; 1968) or in vivo, when it is known as flooding (Watson, Gind & Marks, 1972; Rachman, 1969). The efficacy of both systematic desensitization and flooding has been readily demonstrated (Levis, 1974). However, over a number of years, numerous studies have been conducted to test their comparative efficacy. In some instances, desensitization procedures have been found more effective (Melia & Nawas, 1971; Cornish & Dilley, 1973; Rachman, 1966; Rudestan & Bedrosan, 1977). In other studies, flooding techniques have proved superior (Boulougouris, Marks & Marset, 1971; Boudewyns & Wilson, 1972; Marshall et al., 1977). Still other studies have found no difference (De Moor, 1970; Gelder et al., 1973).

However, given the question surrounding the mode of action of these treatments, there is some doubt as to the validity of their conceptualized differences (for an extended treatment of this question see Marks, 1975). Future research will undoubtedly clarify many of these questions.

While these techniques are the most widely used, there are many other behavioural approaches for the treatment of anxiety. These include: modeling (Bandura, Blanchard & Ritter, 1969; Ritter, 1968); cognitive rehearsal, stress inoculation, cognitive modification, and anxiety management training (Goldfried & Goldfried, 1975; Meichenbaum, 1972; Meyer & Reich, 1978; Suinn & Richardson, 1971); induced anxiety (Ascough, 1972; Noonan, 1971; Sippelle, 1967); fading (Ost, 1978); thought stopping (Anthony & Adelstein, 1975; Rimm, 1973; Wolpe, 1973); participant modeling (Denny et al., 1977); heart rate biofeedback (Gatchel et al., 1979); and E.M.G. feedback (Raskin et al., 1973; Canter et al., 1975).

Pharmacological Treatments. Over the past decade there has been a marked increase in the use of pharmacological treatments for anxiety. Of the drugs employed, the most widely used are the benzodiazepine compounds. Of this group of drugs which includes diazepam, flurazepam, chlordiazepoxide, nitrazepam and oxazepam, diazepam is the one that is most often prescribed (Goth, 1974). Thus it was noted by Greenblatt and Shader (1974) that in a 3 month period approximately one in ten American adults would take diazepam for nervousness or tension.

While the benzodiazepines are the most widely used anti-anxiety agents utilised in treatment, other types of drugs are also employed. These include antidepressants e.g., chloimipramine (Casey et al., 1975); thioridazine (Lofft & Demars, 1974); phenelzine (Tyrel et al., 1973); and barbituates such as brevital (Marks et al., 1972; Whitehead et al., 1978). In instances of panic anxiety, anti-depressants such as imipramine, as well as the M.A.O. inhibitors, are the drugs of choice (Bassuk & Schoonover, 1977; Quitkin et al., 1972; Zitru, Klein & Woener, 1978; Klein, 1964, 1967). The barbituates and carbamates have also been used extensively for the treatment of anxiety. However, since they are more toxic and addicting and produce more severe CNS depression than the benzodiazepines, they are now used less frequently (Bassuk & Schoonover, 1977).

As can be seen from the above an examination of the strategies employed in the treatment of anxiety reinforces the concept of the whole issue of anxiety being surrounded by confusion. The treatments provided range from the talking cures of the psychotherapies to the nervous system agents of chemotherapy, and to a considerable extent what is used depends upon the training and ideological orientation of the practising clinician. There are three main classes of health professionals who treat patients for anxiety. These are psychiatrists, psychologists and similar mental health workers, and physicians other than psychiatrists especially general practitioners, gynecologists, internists and neurologists (G.A.P., 1975). These physicians with a background in medicine and pharmacology but a limited background in

psychotherapy tend to prescribe medication. Indeed, the majority of prescriptions for psychotropic drugs are written by non psychiatrists (Rickels, 1979). Psychologists and other health workers lack a medical degree and generally do not have a background in pharmacology. Furthermore, because of their training they tend generally to use either behaviour therapy techniques, or some other system of talking psychotherapy depending on their specific orientation. Psychiatrists through their medical degrees and training are in a position to use both drugs and psychotherapy. However, they lack a coherent body of literature integrating the diverse positions, and they tend to follow one course or the other (G.A.P., 1975; Lesse, 1970).

THE UTILITY OF THE PHOBIC RESPONSE AS A GENERAL
MODEL FOR THE ANXIETY RESPONSE

The third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM 111) (1977) lists under the category of anxiety disorders the following syndromes: panic disorder, generalized anxiety disorder, phobic and obsessive compulsive disorder. The one common factor in all of these disorders is the predominance of the anxiety state in the clinical picture. Given such a variety of presentation, however, a major issue confronting researchers in this field is the possibility of finding some parsimonious way of studying the anxiety reaction, some method that will aid in the clarification of the present definitional system.

One possible approach could be to use the phobic response as a model for the study of anxiety. A model can be defined as an object of imitation, something that accurately resembles something else, something that has all the known variables in the phenomenon that it models. In psychopathology, a good heuristic model must have symptoms, causal events, and even anatomical structures in common with the pathology (Maser & Seligman, 1977). The question, therefore, is whether the phobic response can fulfill such criteria, and thereby serve as a model for the study of anxiety. To even attempt to answer this question requires a detailed examination of phobias.

Definition of Phobias. While the phobic response has been documented in the literature as early as the 17th century (Burton, 1621 cited in

Marks, 1969), the first usage of the term phobia dates back to 1801. Over the next century it gradually evolved into common usage (Marks, 1969).

While many theorists have contributed to the literature on phobias, amongst the most influential have been those from the psychoanalytic tradition, which had its major impact in the area of theory; and those from the learning theory branch, which had its major contribution in the area of treatment.

Psychoanalytic Approach. In his early approach to phobias, Freud (1894) believed that phobias had their roots, as did hysteria, in the psychological mechanisms that were also operating in the 'apparently' formal cases of Anxiety Neurosis. As he wrote in 1894:

"in the phobias of anxiety neurosis, the affect is always the same, always that of anxiety; it does not originate in a repressed idea, proves not susceptible to further psychological analysis, and is not amenable to psychotherapy."

and later on, (1885):

"Nothing is ever found (in the investigation of a phobia) but the anxiety state, which by a sort of selection, brings up all the ideas adopted to become the source of the phobia."

Thus, in his early formulations, Freud stressed the role of anxiety in his conceptualization of phobias, adopting what was basically an associationist stance (Snaith, 1968). At this time he divided the phobias into two groups according to the nature of the object feared: (1) 'common phobias' or an exaggerated fear of all those things everyone fears to some extent e.g., night, death, illness and (2) 'specific

phobias' or the fear of special circumstances, which he considered not normally present e.g., agoraphobia (Freud, 1895).

Much later on, however, following his analyses of two infant zoophobias, he refashioned his conceptualization of phobias, (Freud, 1936). Phobias, now came to be interpreted as a defence against anxiety, and in fact all, or at least the majority of phobias were seen as traceable to a fear on the ego's part of the demands of the libido (Freud, 1936). Thus, for example, fear in zoophobia was seen as castration anxiety and fear in agoraphobia was seen as fear of temptation. This later approach, which suggests that phobic symptoms are representations of an underlying disorder, came to represent the basic tenet of the psychoanalytic theorists (e.g., Brill, 1960; Horney, 1950; Hendrick, 1966; Fenichel, 1945).

Learning Theory Approach. The development of learning theory approaches to the amelioration of phobic disorders can be traced to the work of researchers such as Pavlov (1927, 1928, 1941), Watson (1930), Masserman (1943), Eysenck (1960), and others. However, the theorist whose work had the greatest impact was Wolpe (1958). From his work involving the evocation of experimental neurosis in cats, Wolpe (1952, 1958) came to formulate the reciprocal inhibition principle: "If a response inhibiting anxiety can be made to occur in the presence of anxiety evoking stimuli, it will weaken the bond between these stimuli and therefore the anxiety." To Wolpe (1958, 1973), anxiety is a prominent constituent of neurotic reactions, and a phobic response is simply a neurotic reaction to a

specific stimulus. Wolpe's position differed from psychoanalytic theory in defining neurotic anxiety (hence phobias) as persistent maladaptive autonomic responses which are acquired through a classical conditioning process.

The Nature of Phobias. Thus, generally speaking, in their initial formulations the major theorists in the field conceive of phobias as being somehow related to anxiety. However, the name phobia itself (from the Greek word "phobos" meaning flight, panic, fear or terror) as well as the early literature (e.g., Jaspers, 1923; Ross, 1937; Terhune, 1949; Langhlin, 1956; Errera, 1962) depicted phobias as a strong, persisting, special form of fear which (1) is out of proportion to demands of the situation, (2) cannot be explained or reasoned away, (3) is beyond voluntary control, and (4) leads to avoidance of the feared situation. The implications of this approach are that all phobias are clinically similar, in that they reflect a similar disorder with a similar aetiology. However, a closer examination of the disorder reveals that this is not so and it appears that under the rubric of phobic states are a range of related disorders with overlapping features.

In general, most researchers now differentiate between two clear cut syndromes, the monosymptomatic phobias, and the multisymptomatic phobias. A good example of the former is the phobia of small animals. These tend to be discrete disturbances, most commonly occurring in women (Agras et al., 1969; Marks & Gelder, 1966). They are

characterised by early onset and good response to treatment (Marks & Gelder, 1966). An example of the multisymptomatic phobias is agoraphobia. This is characterised by the presence of panic attacks, higher chronic anxiety, more overall fearfulness and depression, later onset, and a less responsive reaction to treatment (Hallam & Hafner, 1978; Rohs & Noyes, 1978; Snaith, 1968; Shafar, 1976; Zitru, Klein & Woener, 1978; Mathews, 1978). In addition to the above two syndromes, researchers identify other phobias which appear to have their own characteristic dimensions, these include social phobias (Hallam & Hafner, 1978; Marks, 1969; Tyrer, Candy & Kelly, 1973), school phobias (Baker & Wills, 1978; Hersov, 1960; Leventhal & Sills, 1964), and miscellaneous other phobias (Snaith, 1968; Amit et al., 1974; Connolly, Hallam & Marks, 1975).

Despite the differences, there is one feature that is common to all the various phobic states, and that is an anxiety response. To quote Wolpe (1962) "The distinctive feature of a classical phobia is the presence of clearly ostensible sources of anxiety" (1962 p. 316). A similar response is adopted by other theorists who in attempting to delineate the syndrome equate it with anxiety (e.g., Borkovec, 1974; Hiebert & Fitzsimmons, 1978; Kelly & Walter, 1968; Kelly, Brown & Shaffer, 1970; Mathews, 1978; Shaw, 1979; Snaith, 1968; Watson & Marks, 1971). Further support for this position is evident when one considers that most treatments of phobias, e.g., systematic desensitization, flooding, paradoxical intention, relaxation techniques, etc., usually accord a key role to anxiety and its reduction during treatment.

A further development in the literature that supports the above argument is the tendency of some researchers to propose definitions of anxiety that are couched in terms of fear and phobic avoidance. For instance, Marks and Lader (1973) noted that phobic avoidance is common in clinical anxiety and that when it is marked, the clinical features become indistinguishable from severe agoraphobia. Similarly, Woodruff, Guze and Clayton (1972) reported the presence of phobic avoidance in patients diagnosed as anxiety neurotics. Beck, Laude and Bohnert (1974) claim to have observed that all the patients with neurotic anxiety in their series had cognitions of danger just prior to or during the onset of an exacerbation of anxiety. Thus, there is a blurring of the distinction between a phobia as a fear response, and a phobia as an anxiety response.

Yet another confounding issue is the question of the adequacy of the conceptual distinctions that have been made between the terms "fear" and "anxiety". Currently, based on available evidence, researchers (e.g., Lader & Marks, 1972; Lang, 1969; Lacey, 1967; Marks, 1975; Schroeder & Rich, 1976) define fear as an emotion that has many component reactions which can occur concurrently or sequentially, and are often poorly correlated with one another. It is an organised emotional response syndrome across three main dimensions, cognitive-subjective, motor-behavioural, and physiological. This definition cannot adequately be differentiated from prevailing definitions of anxiety (e.g., Lang, 1971; Borkovec, 1972). Indeed, many theorists in their work either do not differentiate between fear and anxiety (Eysenck, 1964; Gatchell

et al., 1978; Lick et al., 1977; Odum et al., 1978); or they conceptualize fear and anxiety as being equivalent states (Bandura, 1978; Borkovec, 1974; Hiebert & Fitzsimmons, 1978; Izard & Tomkins, 1966; Kelly, 1968; Levitt, 1967; Ost, 1978; Snaith, 1968; Spielberger, 1966; Wolpe, 1958; 1973). In summary, it would appear that labelling phobic states as fear responses rather than anxiety responses, tends to suggest that the phobic response differs from an anxiety response. However, as Borkovec (1974) noted, there is still no evidence on either physiological or self-report grounds that anxiety and fear can be differentiated, thus making the distinction at this point in time meaningless.

One result of the approach of viewing phobias as discrete fear responses has been the proliferation of possible phobic states. Melville (1977) listed a total of 241 familiar and rare phobias, from aerophobia (air) to parthenophobia (young girls) and her list was not even a complete one. A possible solution to the dilemma would be to classify phobias in broad categories, along the lines mentioned before. To the extent that these categories differ in their response to treatment and to the extent that they present different manifestations of the anxiety response, they can be utilised as models of the anxiety phenomenon, and through systematic study, greatly further our understanding of the state.

ISSUES SURROUNDING THE MODE OF ACTION OF VARIOUS
TREATMENT APPROACHES TO ANXIETY/PHOBIAS

As noted before, widely different approaches are used in the treatment of anxiety. These include techniques from the behaviour therapies, especially systematic desensitization and flooding, the anti-anxiety compounds, particularly the benzodiazepines, and the psychotherapies in general. To a greater or lesser degree these treatments are all effective in reducing anxiety. An understanding of the process by which they operate would therefore greatly clarify many of the issues regarding the syndrome.

In the following section, it will be shown however that on examining the mode of action of these treatments there are still many unanswered questions that need to be clarified.

Mode of Action of Systematic Desensitization/Flooding. The decision to examine the mode of action of systematic desensitization is an attestation of two factors. Firstly, of all the behaviour therapy techniques it can be considered to be the best known, and secondly along with flooding it has been the subject of extensive research and theoretical formulations (Marks, 1969, p. 182). The first theoretical explanation of systematic desensitization was the reciprocal inhibition hypothesis of Wolpe (1958). According to this hypothesis, relaxation training functions in systematic desensitization to produce a state of muscular relaxation that is incompatible with the state of anxiety

elicited by the phobic stimulus. This is purportedly achieved through the lowering of autonomic nervous system activity through decreased muscle tonus (Greenwood & Benson, 1977). That being the case, training in progressive relaxation should produce a state of reduced physiological functioning.

However, although progressive relaxation is widely recognized as an effective anti-anxiety agent, its action on physiological functions is still being questioned (Greenwood & Benson, 1977). While Paul (1969) and Jacobson (1938) obtained results that supported the notion of reduced physiological functioning through relaxation training, the majority of researchers who have investigated the phenomenon reported contrary findings. For example Mathews and Gelder (1969) found no significant decreases in forearm electromyogram (E.M.G.) within a population of subjects with phobias or generalized anxiety who practised progressive relaxation. Furthermore, there were no differences between this group and a control group. Similar results have been obtained by other researchers using frontalis E.M.G. (Edelman, 1971; Lader & Mathews, 1970), heart rate (Mathews & Golder, 1969), forearm blood flow (Benjamin, Marks & Huson, 1972), and respiratory rate (Lader & Mathews, 1970) as their dependent measures.

Apart from this general lack of support, the reciprocal inhibition model suffered from one further shortcoming. Some theorists suggested that systematic desensitization could be regarded as an inefficient form of flooding, and that exposure might be the basic ingredient of all phobic treatments (Amit & Sutherland, 1974; Marks,

1975). However, the reciprocal inhibition model was unable to accommodate within its framework the results obtained from flooding. This led to a search for hypotheses that could encompass these two approaches. Of these, the habituation/extinction hypothesis (Watts, 1979) has been the most frequently proposed. This hypothesis states that reduction of physiological arousal during exposure is essential to positive change in fear behaviour. However, in examining the studies that have attempted a test of this hypothesis, again it is found that the results are equivocal. Some support came from a study by Lader, Gelder and Marks (1967), who found a significant ($r = .49$) correlation across subjects between the rate of auditory habituation, and clinical response to desensitization. Similar findings were reported by Lang (1970), and Lang, Melamed and Hart (1970). On the other hand, Gillan and Rachman (1974), and Klorman (1974), in their experiments failed to support the hypothesis. As Watts (1979) in his extensive review of this question noted, while it is probably too soon to make a final judgment on the adequacy of the model, it still leaves many questions unanswered.

In conclusion, while it is noted that both systematic desensitization and flooding treatments are effective in reducing phobic avoidance behaviour (Barrett, 1969; Boudewyns & Wilson, 1972; DeMoor, 1970; Rachman, 1966), the mechanism by which this is achieved is still not well understood.

Mode of Action of Anti-Anxiety Compounds. The questions regarding

the mode of action of the anti-anxiety agents can be regarded as being more of an empirical nature than the theorizing surrounding systematic desensitization and flooding, in that the former can be more directly measured and is on the whole more quantifiable. Nevertheless, the situation is not very much clearer.

The anti-anxiety compounds now generally used are found in four different chemical classes, which are quite unrelated. These are the diphenyl methanes (the phenothiazines); the substituted amides (the barbituates); the propane diols (e.g., meprobamate); and the benzodiazepines (e.g., diazepam) (Heise, 1965). Their differences make it unlikely that they will have a similar chemical action. The understanding of the mode of action of these drugs as anti-anxiety agents was obtained from laboratory experiments on behaviour with animals such as cats, rats, and mice (Taylor & Laverty, 1969; Bookman & Randell, 1975; Dantzer, 1977). In these animals, the anti-anxiety agents produce sedation, decrease aggressive responses, and act as anti-convulsants and muscle relaxants (Bookman & Randell, 1975; Feldman, 1962; Heise, 1965; Hanson & Stone, 1964). Thus, they exhibit similar behavioural properties.

However, these behavioural properties notwithstanding, they apparently have different effects on the central nervous system. For example, in the case of the benzodiazepines, the most clinically important effects are mediated through the central nervous system (Greenblatt & Shader, 1974). As far as the actual site is concerned, some researchers implicate the limbic system (Greenblatt & Shader,

1974), some the thalamus and the spinal cord (Schallek et al., 1972; Chow & Wang, 1977) and others the cerebral and cerebella cortex (Möller & Okada, 1978). There is also some suggestion that their skeletal muscle relaxant properties may contribute to their anti-anxiety effects, but researchers are still unclear whether or not this property is centrally or peripherally mediated (Bassuk & Schoonover, 1977). The phenothiazines on the other hand, seemingly have their action as tranquilizers from a complex interaction of biochemical, biophysical, and morphological factors operating in subcortical regions of the brain (Richter, 1965).

Given the limitations regarding experimentation on humans, the attempts that have been made to obtain information from clinical drug trials on anxiety with humans have been largely unrewarding. Apart from the question of comparative efficacy (Jenner, Kerry & Korchin, 1961; Rickels et al., 1972; Rickles & Snow, 1964; Uhlenhuth et al., 1972), these studies have contributed little information regarding mode of action. Furthermore, particularly in the case of the minor tranquilizers, with the exception of the benzodiazepines (Läder, 1974; Daneman, 1964), these drugs are not consistently more effective than placebos (McNair et al., 1965; Reynolds et al., 1965).

Mode of Action of Psychotherapy. While psychotherapists generally address themselves to a range of problems other than anxiety, the following comments also pertain to anxiety. To the question of mode of action, each therapist provides explanations based both on the

particular techniques employed, and the theoretical frames of reference within which they operate. For the client-centered therapist for example, what is important is the focus on the patient's immediate experience, and the empathy and unconditioned positive regard of the therapist (Rogers, 1957). To the psychoanalyst, the analysis of transference and resistance that goes back to early childhood roots is essential (Chessick, 1969; 1974; Katzenelbogen, 1958). In holistic primal therapy, the therapist relieves anxiety by acting as a catalyst for regression, the dismantling of defenses, and the experiencing of a primal (Janov, 1970; Verny, 1978). Thus, for each system a unique explanation is offered. However, as Bromberg (1975) noted, on looking behind the technique used, one sees some general human reactions, the extra technical aspects of psychotherapy that play a vital role in successful therapy. Indeed, regardless of theoretical allegiance, the therapist is generally an expert conversationalist whose specialized equipment includes: sensitivity to the emotional nuances of the patient's communication, the ability to listen selectively, facility in encouraging the patient to initiate conversation, and deftness in leading the patient to emotionally charged topics (Schofield, 1968).

From the foregoing, one is left with a confused picture of the anxiety literature. This confusion at one level can be simply a reflection of the fact that anxiety has been approached and studied from differing points of view, each of which can be regarded as being a reflection of different facets of the phenomenon. The fact that it can be treated in different ways however poses a more challenging

dilemma. Is there one anxiety state? Are there several anxiety states, or do all the treatments have a common underlying mechanism, are only some of the unanswered questions. What is desperately needed is a comprehensive approach to anxiety, an approach which will attempt to integrate these disparate facets.

Such a comprehensive approach will also aid in answering three very fundamental questions. The first is the question regarding the role of arousal in anxiety. Essentially, to what extent are central changes basic to the mechanism of anxiety maintenance and reduction? This idea is central to the approach taken by theorists such as Lader, who proposed that central anxiolytics, acting specifically on the reticular activating system, should be the treatment of choice (Lader, 1974). Similarly, Borkovec sees the reduction of the physiological response as a necessary first step in anxiety reduction (Borkovec, 1974). Furthermore, implicit in the therapeutic strategies that utilize relaxation techniques and tranquilizers, is the conception of physiological reduction being related to anxiety reduction.

Secondly, there is the question of individual variation in autonomic response to anxiety. Many researchers (e.g., Barrell & Price, 1977; Lacey, 1950, 1957; Lacey & Lacey, 1958), have suggested that subjects tend to show idiosyncratic responses across stressors, suggesting the presence of response stereotypy. Other researchers have also suggested the existence of response patterning for homogeneous groups of individuals such as phobics and psychopaths (Ax et al., 1969; Graham, 1973; Hare & Blevings, 1975; Hinton & O'Neil,

1978; Hodapp, Weyer & Baker, 1975; Lovallo, Parsons & Holloway, 1973).

The third question concerns the likelihood of matching individual patients to specific treatment approaches based on the probability of response specificity. This issue while of great interest has not been systematically researched to any extent. One exception is a recent study performed by Davidson, Davidson and Freedland (1977). They selected female undergraduates on the basis of different modes of physiological responding. They were then tested under progressive relaxation analogue, and a rational emotive therapy analogue. Their overall findings, led them to suggest that anxiety can be differentiated into specific subcomponents, and furthermore that different therapeutic regimes differentially affect responding.

Similar observations on the differential action of various therapeutic regimes were made by John (1977). In that study, progressive relaxation and diazepam were compared for their efficacy in the reduction of anxiety as monitored by self-rating, frontalis E.M.G., pulse rate, systolic blood pressure and diastolic blood pressure, the anxiety being elicited under threat of electric shock. It was observed that progressive relaxation acted predominantly to reduce frontalis E.M.G. and was most effective in reducing self report of anxiety, while diazepam was most efficacious on heart rate and systolic blood pressure.

In summary, there is a great deal of ambiguity regarding the theoretical mechanisms of anxiety, both of the response itself, and of the treatment techniques utilised in its elaboration. Much

of this ambiguity is maintained by two factors. (1) The disparate number of theoretical formulations regarding the nature of the state, many of which are contradictory, and (2) The tendency of researchers to investigate the phenomenon from narrow perspectives, while making generalizations supposedly attributable to the state as a whole. What is needed is a research strategy that will attempt to integrate many of the seemingly contradictory findings now present in the literature.

The Present Research. In an attempt at a more comprehensive approach to the study of anxiety, a series of studies were designed. The first two studies compared progressive relaxation, diazepam, and placebo drug for their efficacy in reducing anxiety under two different levels of intensity. The first study looked at the reduction of low anxiety, this was defined as anxiety caused simply by taking part in an experiment that was investigating the state. The second study looked at the reduction of medium anxiety. This was anxiety induced through the threat of an electric shock. (The findings from these studies will be described in greater detail in the discussion).

The present study reported here, is the third in this series. It attempts to answer three questions. Questions one and two are related to anxiety reduction, while question three is related to the reduction of phobic avoidance. A further elaboration of these questions follows.

The first question looked at the comparative efficacy of

progressive relaxation, diazepam, and placebo drug in the reduction of high anxiety. Here high anxiety was induced through exposure to visually presented phobic stimuli. The second question asked was by what mechanisms do progressive relaxation and diazepam reduce anxiety i.e., monitoring anxiety by self report, frontalis E.M.G., pulse rate, systolic blood pressure and diastolic blood pressure, are there reliable differences between the actions of progressive relaxation and diazepam? The third question looked at the role of anxiety reduction in the flooding treatment of phobias. That is, does reducing anxiety during exposure enhance the treatment of phobias when exposure is utilised? In order to place this third question in proper context, a brief review of the literature will be given.

Flooding, as originally conceptualized, involves the exposure of the organism to high intensity anxiety-provoking stimulation (Boulougouris & Marks, 1969; Marks, 1972; Stern & Marks, 1973), and particularly when practised as implosion, the emphasis was on the patient experiencing anxiety as fully as possible (Stampfl, 1967; Stampfl & Levis, 1967; Hogan & Kirchner, 1967).

Marks (1973) after reviewing the many fear techniques involved in fear reduction, among them being desensitization, flooding, modeling, prolonged exposure, aversion relief, paradoxical intention, operant shaping etc., suggested that one common mechanism shared by these techniques was exposure. This kind of theorizing questions the role of anxiety evocation in the flooding paradigm. Fundamentally, one is asking whether the heightened anxiety is not just an unfor-

tunate by-product of the technique, rather than being essential:

In an early test of this question, Hussain (1971) conducted a cross-over study comparing flooding with intravenous thiopental, to flooding with a placebo, and reported that the drug significantly enhanced the effectiveness of the exposure treatment. Similarly, Marks et al., (1972) treated 18 mixed phobics under one of three conditions (1) Two hours of continuous flooding in practice during "waning" diazepam effect (i.e., four hours after oral ingestion of .1mg of diazepam per kilogram of body weight); (2) Two hours continuous flooding during "peak" diazepam (i.e., one hour after diazepam ingestion) and (3) Flooding as for groups (1) and (2), but under placebo. Significant changes were obtained under all three conditions. However, these changes reached greatest level of significance in the "waning" diazepam condition, were next greatest in the "peak" diazepam group, and least in the placebo group. Similar results were obtained by Johnston and Gath (1973) though their sample consisted of only 6 agoraphobic patients.

In marked contrast, conflicting results have also been reported in both the animal (Kamano, 1968; Voss, Mejta & Reid, 1974; Cooper et al., 1974; Christy & Reid, 1975) and human literature (Hafner & Marks, 1976). In a recent study, Whitehead, Robinson, Blackwell and Stutz (1978) tested 12 small animal phobics in a design which compared flooding combined with either chronic diazepam (15 mg/day) or placebo. They reported that the diazepam administration did not enhance the effectiveness of flooding as a treatment for phobias

when compared to placebo. Similarly, Chambless and Foa (1979) tested twenty-seven agoraphobic patients under one of these conditions, eight sessions of flooding in fantasy with anxiety, flooding in fantasy with intravenous Brevital, or an attention-control procedure. The results indicated that while there were no significant differences between the treatment groups on behavioural outcome, on client's ratings of fear, the non-drug group showed a significantly greater reduction than the drug group.

In attempting to assess these conflicting results, several criticisms can be offered. Firstly, with some of these studies the sample sizes tended to be small, data being reported on as few as four subjects in one study (Johnston & Gath, 1973). Secondly, the population tested were either agoraphobics (Hafner & Marks, 1976), small animal phobics (Whitehead et al., 1978), or mixed phobics (Marks et al., 1972). The assumption being made when these studies are compared is that these different phobic states are equivalent disorders, but that proposition is questionable (Hallam, 1978; Torgensen, 1979). Thirdly, the studies tended to compare drug with placebo (Johnson & Gath, 1973; Hafner & Marks, 1976), or differing levels of drug with placebo (Marks et al., 1972). This kind of comparison ignores the fact that the placebo effect, as a factor in and of itself, has often proved to be quite effective (Raskova & Elis, 1978). Finally, studies differ in their outcome criteria, some utilising physiological and clinical measures (Marks, Viswanathan, Lipsedge & Gardner, 1972), some clinical scales (Hafner & Marks, 1976); while others used be-

havioural criteria (Johnston & Gath, 1973; Whitehead et al., 1978).

The present study attempted to address itself to some of these questions. Firstly, by utilizing anti-anxiety agents as well as a drug placebo, the placebo factor can be examined. Secondly, with six groups and nine subjects per group, the sample was fairly large. Thirdly, while the behavioural avoidance measure was the main dependent variable, self report scales and fear survey schedules were also utilised for comparison purposes. The attempt here was to try to resolve discrepancies of outcomes in previous studies which used some but not all of these anxiety measures. Indeed part of the purpose of the investigation was to document the possible inter-relations among behavioural, subjective and physiological measures of anxiety.

Method

Subjects

One hundred subjects were screened, over a period of three years, for phobic responses to rats, snakes or spiders. Thirty-one did not meet the criteria for inclusion (see below), a further eleven met the criteria but did not volunteer for the study, while six started but dropped out before completion. This left a total of fifty-four (54) subjects who completed the study.

Subjects ranged in age from 20 to 59, with a mean age of 29. There were six (6) males, (2 spider and 4 snake phobics) and forty-eight (48) females (5 rat, 7 snake, and 36 spider phobics). The subjects were recruited from the university population and the general public via newspaper, radio and television advertisement. The advertisement was worded as follows:

"Phobic? Afraid of rats, spiders or snakes?
The Psychology Department at Concordia University
is conducting a treatment study. For information call"

Design

There were six experimental groups with nine subjects per group. The groups were balanced for age and sex using a stratified randomized procedure.

The six groups were (1) A group receiving progressive relaxation exercises plus flooding (P.R.F.), (2) A group receiving 5 mg. of diazepam plus flooding (V5 mg. F), (3) A group receiving 7.5 mg. of diazepam plus flooding (V7.5 mg. F), (4) A group receiving a drug

placebo, plus flooding (Pl.F), (5) A group instructed to sit and relax, plus flooding (RC.F.) and (6) a no-treatment control group (N.T.C.).

Measures and Apparatus

A Sony cassette recorder type #T.C.-67 was used to present the taped instructions for the (P.R.F.) group. Electromyograph recordings (muscle action potential) were obtained from all subjects, across the frontalis muscle, using a B.F.T. 401 Feedback Myograph (Bio-Feedback Technology Inc.) with silver alloy electrodes coated with No. 228 B.F.T. electrode cream, and mounted on an adjustable headband. Slides of the animals used as phobic stimuli were projected onto a wall approximately ten feet from the subject, using a slide projector (Kodak Carousel 75QH). Systolic and diastolic blood pressure were recorded with an automatic blood pressure instrument (The Bonn S.P. Medeler International Corporation). Pulse rate was measured manually using a simple wrist count and a stopwatch.

Subjective Rating Scales

Three subjective rating scales were used; they required subjects to give ratings of phobic anxiety, general free floating anxiety, and present situational anxiety. These scales were adopted from Watson and Marks (1971), (see Appendix A for examples of these scales).

Battery of Anxiety and Phobic Tests

Subjects' general level of anxiety was also measured by the Institute for Personality and Ability Testing Self Analysis Form

(I.P.A.T.) (Cattell, 1957). Subjects' phobic responses were assessed by three measures; The Fear Survey Schedule (F.S.S.) (Wolpe & Lang, 1964), The What Are You Afraid of (W.A.Y.A.O.) (Sutherland & Amit, 1975), and The Behavioural Avoidance Test (B.A.T.) (John & Sutherland, 1977) adopted from Lick, Sushinsky and Malow (1977) (see Appendices B - E for examples of these measures).

Screening Procedure

After some brief introductory remarks, a questionnaire requesting demographic information was administered (see Appendix F). At this point, subjects who had previously received treatment for the phobia, who were currently on tranquilizers or similar medication, or who were presently in therapy, were excluded from participating in the study.

Following this the subject lay on a recliner, and base line measures of systolic and diastolic blood pressure, frontalis muscle tension, and pulse rate were recorded. In addition to these physiological measures, subjective ratings of phobic anxiety, generalised free floating anxiety, and present situational anxiety were recorded. Following these measures, a Fear Survey Schedule (F.S.S.) was administered. Upon completion of the F.S.S. the subject was handed written instructions regarding the behavioural avoidance test to be administered next (see Appendix G). The subject was reassured regarding the requirements of the behavioural test before being conducted to the room where the feared animal would be. These were either a common house spider approximately two inches in diameter,

a harmless eastern garter snake, approximately 18 inches long or a male hooded rat, approximately 300 grams in weight.

For those subjects who were unable to enter the room, the B.A.T. was terminated at this point, and they were introduced to the next step in the procedure. For those who entered the room, with the experimenter being present, they were allowed a maximum of seventeen minutes to interact with the feared animal. Five minutes were allowed for initial approach (Steps 1-4), six minutes for the second phase involving further approach and touching (Steps 5-9), and six minutes were allowed for handling (Steps 10-18). Those subjects who went beyond step four in this test, were excluded from the study.

It was explained to them that step four was the cut-off for the study, and that individuals who could go beyond this point did not meet the major criterion for inclusion.

Following the B.A.T., the subject was returned to the experimental room, and the physiological measures (E.M.G., P.R., S.B.P., and D.B.P.) along with self report of present anxiety were recorded. Finally the I.P.A.T., and the W.A.Y.A.O., were administered.

This marked the end of the screening procedure. Those subjects who met the criteria for inclusion, and this included a score of four or five on the S.R.P.A., and a score of four or less on the B.A.T., were then informed of the procedure to be followed. An informed consent form was then assigned, and the time for the first treatment session was arranged (see Appendix H).

Treatment Procedure

Each subject in the treatment groups met with the experimenter for eight treatment sessions. The first six sessions were held twice weekly for a period of three consecutive weeks. The seventh session was held in the fourth week, and the eighth in the fifth week. All sessions were held in a dimly lit room, with subjects lying on a soft leather reclining chair.

At the commencement of every session, for subjects in all treatment groups, pre-test measures of basal autonomic activity were recorded within two minutes of the subject reclining. These measures included, frontalis muscle action potential, systolic blood pressure, diastolic blood pressure, and pulse rate. In addition, subjective rating of present anxiety was also recorded. Following this the active treatment was begun.

Sessions 1-6

P.R.F. Group. The experimenter turned on the tape recorder, and instructed the subject to follow the relaxation instructions contained on the tape. The experimenter then left the room. The taped instructions consisted of a modified version of the Jacobsonian relaxation procedure (Jacobson, 1939) (see Appendix I), which lasted for a period of twenty-three minutes.

V5.7., V7.5F., and P1.F Groups. Subjects in these three treatment groups were given either 5mg., or 7.5mg. of diazepam (Valium, Hoffman La-Roche) or a placebo drug (Valium placebo). Both levels of drug and the placebo were given in identical capsules. Following the

administration of the capsule, the experimenter left the room, while subject remained relaxing in the recliner, for twenty-three minutes. This period of relaxation was equivalent to that of the taped relaxation exercises.

R.C.F. Group. In this group, the experimenter simply instructed the subject to relax, and then left the room for twenty-three minutes.

Following the initial relaxation phase, the experimenter returned and recorded post-test levels of autonomic functioning, and self rating of present anxiety. The subject was then exposed to slides of the phobic stimulus (rat, snake, or spider), for an initial presentation of nine minutes. This was followed by the recording of the autonomic measures, and subjects self report of anxiety, then a second nine minutes of slide presentation, and finally, the recording of the autonomic measures and self report.

During the first treatment session, six slides were presented, at the rate of three minutes of exposure per slide. Over the next five sessions, the number of slides was increased at the rate of two per session, with a total of sixteen slides being shown by the sixth session. Although the number of slides was increased over the first six sessions, the total time of the presentation remained constant, new slides always being shown last in any given slide presentation.

During the first two studies, subjects were tested one week following the six active treatments for carry over effect from treatment to non-treatment sessions. It was noted then that one week after

treatment the differential effects of both diazepam and progressive relaxation were still in evidence. It was therefore decided in this study to again test for "carry over" effects of treatment, and to extend the test period to two weeks. This meant that following the six active treatment sessions, there was a seventh session one week later, and an eighth and final session one week after the seventh.

Session No. 7

During the seventh session, the relaxation aspect of the treatment (drug; tape) was eliminated. Pretreatment measures were taken, followed by one nine minute period of exposure to the slides and finally post-test measures.

Session No. 8

This was a treatment and post test session, conducted as follows: the treatment phase was identical to that of session seven. Following the recording of the final measurements, subjects were then administered the F.S.S., the I.P.A.T., and the W.A.Y.A.O., in that order. Following the completion of these questionnaires, the behavioural post test was conducted. This was done in the same manner as the initial screening. Following the B.A.T. subjects were returned to the experimental room, where physiological measures were again recorded. Subjects were then questioned as to the experience, and whether or not they were satisfied with their progress.

No Treatment Control Group. Following the initial screening, subjects were informed that due to scheduling considerations, they could not be accommodated at that time, but that they would be contacted

at a later date. Two to three weeks later, subjects were telephoned, and an appointment was made for a date five weeks from the initial screening. Their function as a no-treatment control was then explained, and treatment was offered.

Results

Pre-Treatment Scores

An analysis of the pre-treatment dependent measures was performed using a one way analysis of variance. These measures included the Behavioural Avoidance Test, the Self Report of Phobic Avoidance, Pulse Rate, Frontalis E.M.G., Systolic Blood Pressure, Diastolic Blood Pressure, Fear Survey Schedule, and the I.P.A.T. As illustrated in Tables 1 and 2 the results indicated non significant differences across the groups for all the dependent measures. This demonstrates that even though the groups were selected primarily on their Behavioural Avoidance scores, they were generally well matched. There was a tendency for the Valium 7.5mg. F and the Placebo F groups to score highest on some of these measures, but in no case was the difference statistically significant. (For the means and standard deviation of the pretest scores see Tables 1 and 2 below).

Outcome Measures

The first set of analyses performed looked at the question concerning the efficacy of the treatment of Phobic Avoidance Behaviour, comparing flooding under five different conditions with a no-treatment control group. Because there were five dependent variables (Behavioural Avoidance Test, Self Rating of Phobic Avoidance, Fear Survey Schedule, What Are You Afraid Of?, and I.P.A.T.), multivariate analysis of variance seemed indicated. However, two way analyses of variance

Table 1

Means and Standard Deviations for Screening Data Across Six Groups

Variables	Groups					
	P. Rel	V5mg. F	V7.5mg. F	Pl. F.	R.C.F.	N.T.C.
F.S.S.						
M	185.4	186.6	191.1	175.3	162.3	176.0
S.D.	54.8	39.8	39.01	36.6	27.8	34.6
W.A.Y.A.O.						
M	151.5	146.3	152.2	148.0	143.7	145.2
S.D.	39.5	27.5	22.5	23.2	13.3	18.7
S.R.P.A.						
M	4.78	4.89	4.89	4.77	5.0	5.0
S.D.	.44	.33	.3	.4	0	0
I.P.A.T.						
M	6.44	6.33	7.44	8.0	5.89	6.66
S.D.	2.06	2.5	1.13	1.5	2.2	2.29
B.A.T.						
M	2.0	2.4	2.1	1.6	2.3	2.4
S.D.	1.9	2.3	1.9	2.1	2.1	1.7
Frontalis E.M.G.						
M	29.2	34.4	35.2	35.7	33.4	27.1
S.D.	10.2	23.2	15.2	16.9	22.5	16.4
Pulse Rate						
M	71.7	81.8	84.8	79.7	83.7	74.2
S.D.	6.3	13.1	11.4	8.2	12.1	12.3
Systolic B.P.						
M	109.2	116.7	118.2	115.3	108.9	113.3
S.D.	13.0	7.5	10.3	11.2	13.7	10.3
Diastolic B.P.						
M	64.4	66.7	68.0	70.2	63.3	65.5
S.D.	9.9	6.7	7.6	2.7	8.0	11.4

Note: F.S.S. = Fear Survey Schedule; W.A.Y.A.O. = What Are You Afraid of;
 S.R.P.A. = Self Rating of Phobic Avoidance; B.A.T. = Behavioural
 Avoidance Test.

Table 2

Summary Analysis of Variance Table for Dependent Measures
 Obtained on Six Groups During Initial Screening Session

Variable	Source	<u>df</u>	<u>SS</u>	<u>F</u>	<u>p</u>
F.S.S.	Between Groups	5	4912.37	.62	.68
S.R.P.A.	" "	5	.54	1.28	.28
W.A.Y.A.O.	" "	5	2913.20	.89	.49
B.A.T.	" "	5	3.70	.18	.96
I.P.A.T.	" "	5	27.42	1.34	.26
E.M.G.	" "	5	557.03	.34	.88
Pulse	" "	5	1275.20	2.14	.07
Systolic B.P.	" "	5	676.83	1.08	.38
Diastolic B.P.	" "	5	280.59	.71	.61

(Balanova, 5.01, 1973) were performed on the data because the cell sizes (nine subjects per group) were not sufficient to justify a multivariate analysis (Overall & Klett, 1972). The results of these analyses are summarized in the sections to follow.

Behavioural Avoidance Test. Upon examining the results from the B.A.T., (see Table 3) it was observed that there was a significant main effects of groups $F = 2.75$, $p < .05$, a significant main effect of sessions (pre-post) $F = 128.36$, $p < .01$ and a significant interaction $F = 5.65$, $p < .01$.

Table 3

Analysis of Variance (Balanova) on the B.A.T.

Pre Test to Post Test Data Obtained in the Experiment

(A = Groups B = Sessions)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A	5	191.08	38.21	2.75	.02
B	1	1089.34	1089.34	128.36	.001
A x B	5	239.82	47.96	5.65	.001

To further identify the source of the significant interaction, the data were subjected to a further analysis. This was done by plotting the pre-post data and computing confidence intervals as

a means of establishing significance levels. It must be noted here that the use of confidence intervals as post-hoc tests, provided a conservative post-test, as each interval is based on a smaller number (N) than the overall analysis. In addition, it allows (see Figure 2) for the visual representation of the degree of independence of the groups.

The post analysis (see Figure 1) indicated that of the six groups, the Valium 5mg. F and the Progressive Relaxation F groups showed pre-post increases in approach behaviour that were significant at the $p < .01$ level (99% confidence intervals). The Valium 7.5mg. F, the Placebo F and the Relaxation Control F groups showed pre-post increases that were significant at the $p < .05$ level (95% confidence intervals). By contrast, the no-treatment control group did not show any change in approach behaviour from pre to post test, and this was probably the source of the significant interaction.

As can be seen from Table 4, when the mean increases across the five treatment groups are compared, the largest difference occurred on the Valium 5mg. F group closely followed by the Progressive Relaxation F group. The Relaxation Control F group on the other hand showed the smallest increase in approach behaviour among the five treatment groups. The results suggest that while flooding alone can effectively reduce avoidance behaviour, the utilization of an anti-anxiety adjunct can facilitate the process.

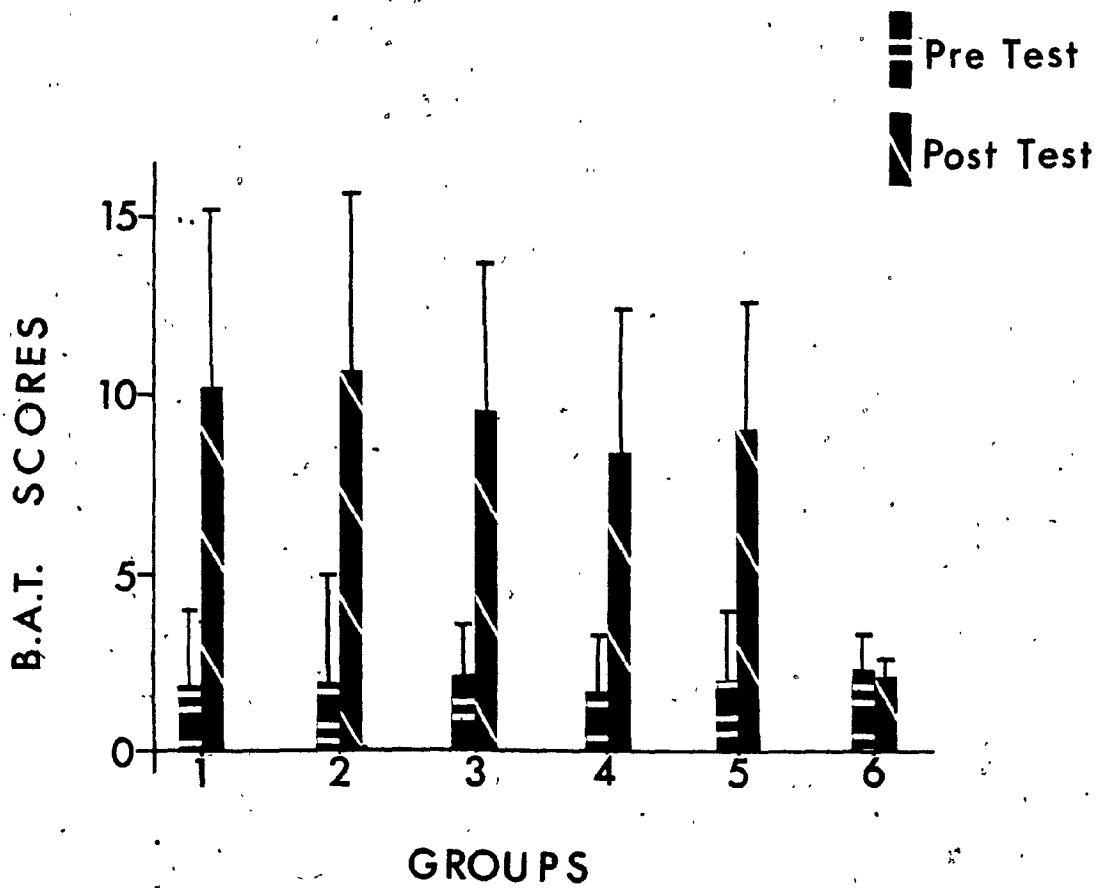


Figure 1. Pre-post measures on the Behavioural Avoidance Test across the six groups.

LEGEND
groups
1-PR
2-VS
3-V75
4-PI
5-RC
6-NYC

Table 4

Mean Difference Scores on the Behavioural Avoidance Test
 Obtained in the Experiment Across the Six Groups,
 For Pre and Post Tests

	Groups					
	P.R.F.	V5 F	V7.5 F	Pl. F	R.C.F.	N.T.C.
Mean						
Pre	2.10	1.88	2.11	1.66	2.31	2.44
Mean						
Post	10.00	10.88	9.55	8.66	9.11	2.31

The other four dependent variables, being either self rating, or paper and pencil measures, were expected to show pre-to post-test results that varied somewhat from that obtained on the behavioural avoidance test. The results obtained were consistent with expectations. The data from the Fear Survey Schedule, and the What Are You Afraid of?, closely resembled the results from the Behavioural Avoidance Test. The Self Rating of Phobic Avoidance was similar though less so, while the I.P.A.T. from pre to post test revealed changes that did not resemble any other measure. A detailed examination of these results

is presented in the following section and in the discussion their relationship to the Behavioural Avoidance Test data will be examined further.

Self Rating of Phobic Avoidance. On this scale subjects rated their degree of fear of the phobic object, on a 1-5 scale. Analysis of variance (Balanova, 1973) indicated a significant group difference $F = 2.73, p < .02$, a significant pre-post effect $F = 59.10, p < .001$, and a significant interaction, $F = 3.13, p < .01$ (see Table 5). To identify the source of the significant interaction, the data were plotted and confidence intervals were computed as was done for the Behavioural Avoidance Test data (see Figure 2). The results indicated that of the six groups, the Relaxation Control F ($p < .01$), the Valium 5mg. F, and the Placebo F groups ($p < .05$) showed significant pre-post reduction. The Progressive Relaxation F and the Valium 7.5mg. F groups showed non significant pre-post reduction in self reported fear. The no-treatment control group did not change from pre to post test, and this was probably the source of the significant interaction.

Fear Survey Schedule: Analysis of Variance (Balanova) indicated a significant sessions (pre-test to post-test) effect ($F = 47.64, p < .01$) but no significant interaction (see Table 6). The data were plotted and examined using confidence intervals (see Figure 3). This analysis failed however to reveal the source of the significant pre-post difference. Examination of the data as shown in Table 7,

across the groups thereby reducing the error. Examination of the data as shown in Table 7 indicates that all the treatment groups showed pre-post decrease in mean F.S.S. scores, ranging from 16.1 on the relaxation control F group to 32.7 on the Valium 7.5mg. F group. However, the no-treatment control group also showed a mean reduction of 9.0 on F.S.S. scores from pre-to post-test, and this may reflect the lability of paper and pencil measures such as the Fear Survey Schedule.

Table 5

Analysis of Variance (Balanova) on the
Self Rating of Phobic Avoidance, Pre-Test to Post-Test Data,
Obtained in the Experiment Across the Six Groups.

(A = Groups B = Sessions)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A	5	8.51	1.70	2.73	.02
B	1	23.03	23.03	59.10	.001
A x B	5	6.62	1.32	3.13	.01

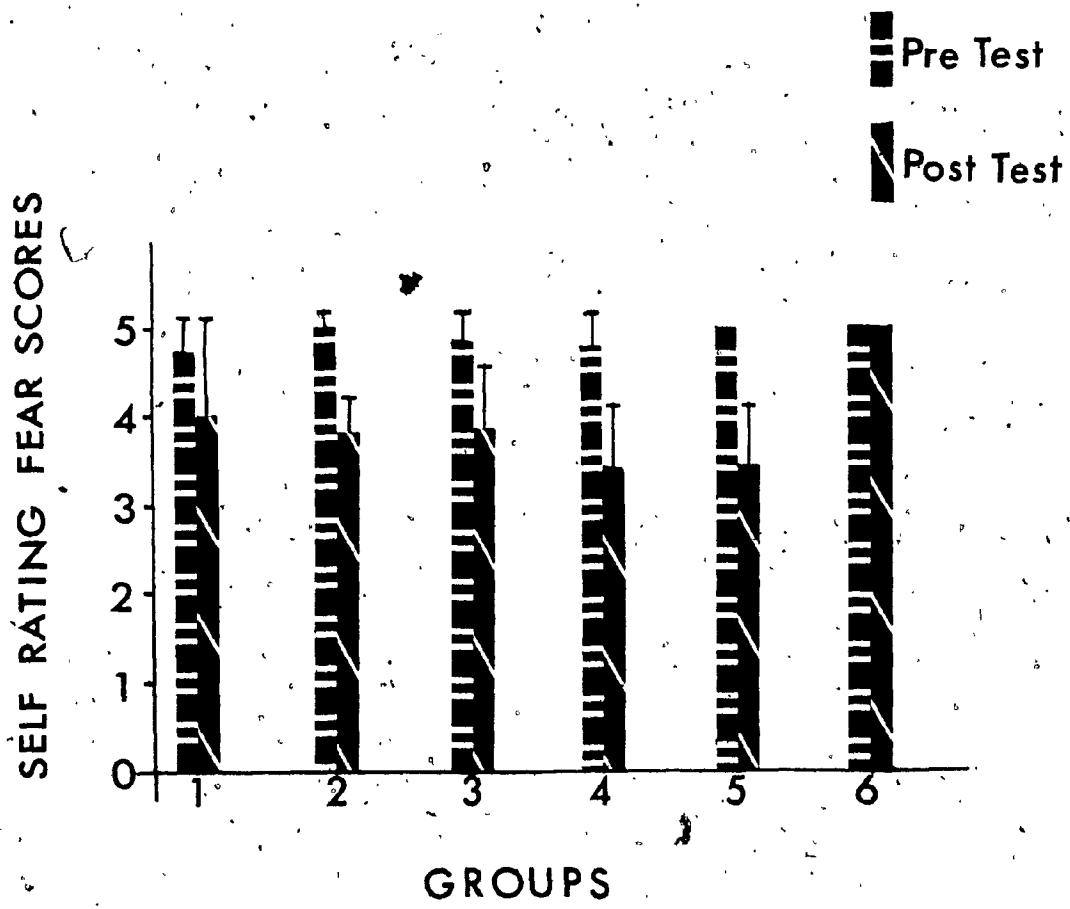


Figure 2. Pre-post measures on Self Rating of Phobic Avoidance across the six groups.

- LEGEND
groups
- 1-PR
 - 2-V5mg
 - 3-V75mg
 - 4-PI
 - 5-RC
 - 6-NTC

Table 6

Analysis of Variance (Balanova) on the Fear Survey Schedule

Pre-Test to Post-Test Data Obtained in the

Experiment Across the Six Groups

(A = Groups B = Sessions)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	5	6517.19	1303.44	.49	n.s.
B	1	14444.50	14444.51	47.36	.001
A x B	5	1801.05	360.20	1.18	n.s.

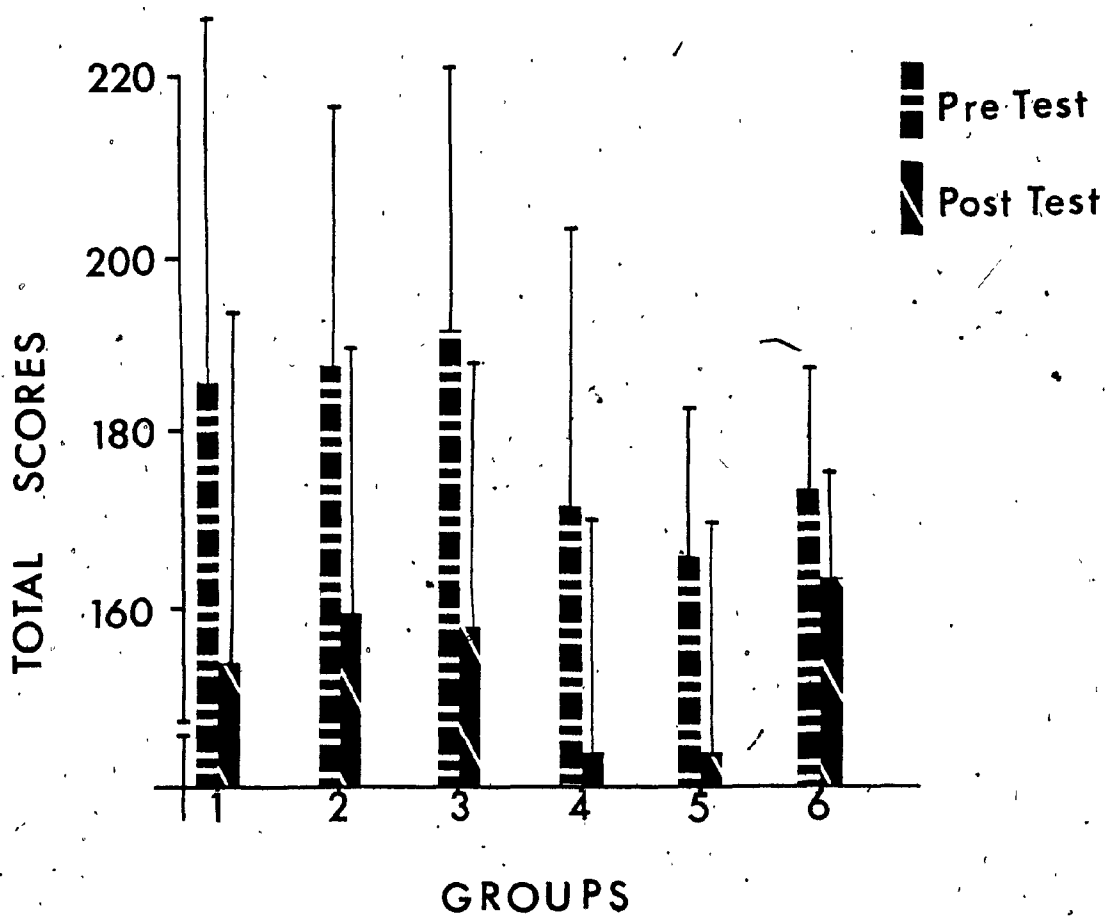


Figure 3. Pre-post measures on the Fear Survey Schedule across the six groups.

LEGEND
groups

1-PR

2-V5mg

3-V7.5mg

4-PI

5-RC

6-NTC

Table 7

Mean Difference Scores on the Fear Survey Schedule
Test Obtained in Experiment Across the Six
Groups, for Pre and Post Tests.

	Groups					
	P.R.F.	V5mg. F	V7.5mg. F	Pl. F	R.C.F.	N.T.C.
Mean						
Pre	185.4	186.6	191.1	175.3	162.3	176.0
Mean						
Post	158.4	162.4	158.4	145.5	146.2	167.0

What Are You Afraid Of? This scale, unlike the Fear Survey Schedule, was developed as a clinical instrument (Amit & Sutherland, 1975), and while there was some overlap of items with the Fear Survey Scale, there were many items not common to both scales. On this scale, the results indicated a significant sessions (pre-post) effect ($F = 34.79, p < .01$) and a significant group by sessions interaction ($F = 2.394, p < .05$) (see Table 8). When the data were plotted and subjected to post-test analysis (95% confidence intervals) (see Figure 4), the results indicated that while all the treatment groups showed non-significant pre-post reduction, the no treatment control group showed a slight increase on post-test. This increase most likely was responsible for the significant interaction observed. It must also be noted here that this increase was contrary to the decrease found on the Fear Survey Schedule, and suggests the possible unreliability of these self report tests as consistently accurate measures of avoidance behaviour.

I.P.A.T. The I.P.A.T. as an anxiety measure was used to evaluate whether or not a change in phobic behaviour is closely related to change in trait anxiety. The analysis of variance yielded no significant main effect of groups, but a significant pre-post difference ($F = 4.41, p < .05$) and a significant interaction ($F = 2.54, p < .05$), (see Table 9). The data were plotted and examined by 95% confidence intervals (see Figure 5). The examination revealed an inconsistent pattern of results that could not be readily explained, and may reflect random variation.

Table 8

Analysis of Variance (Balanova) on the W.A.Y.A.O

Pre Test to Post Test Data Obtained in the

Experiment Across the Six Groups

(A = Groups B = Sessions)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A	5	3122.60	624.52	.57	n.s
B	1	5194.45	5194.45	34.79	.001
A x B	5	1787.27	357.45	2.39	.05

Table 9

Analysis of Variance (Balanova) on the I.P.A.T. Scores

Obtained in the Experiment Across the Six Groups

(A = Groups B = Sessions)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A	5	34.75	6.95	.78	n.s
B	1	2.67	2.67	4.41	.05
A x B	5	7.71	1.54	2.54	.05

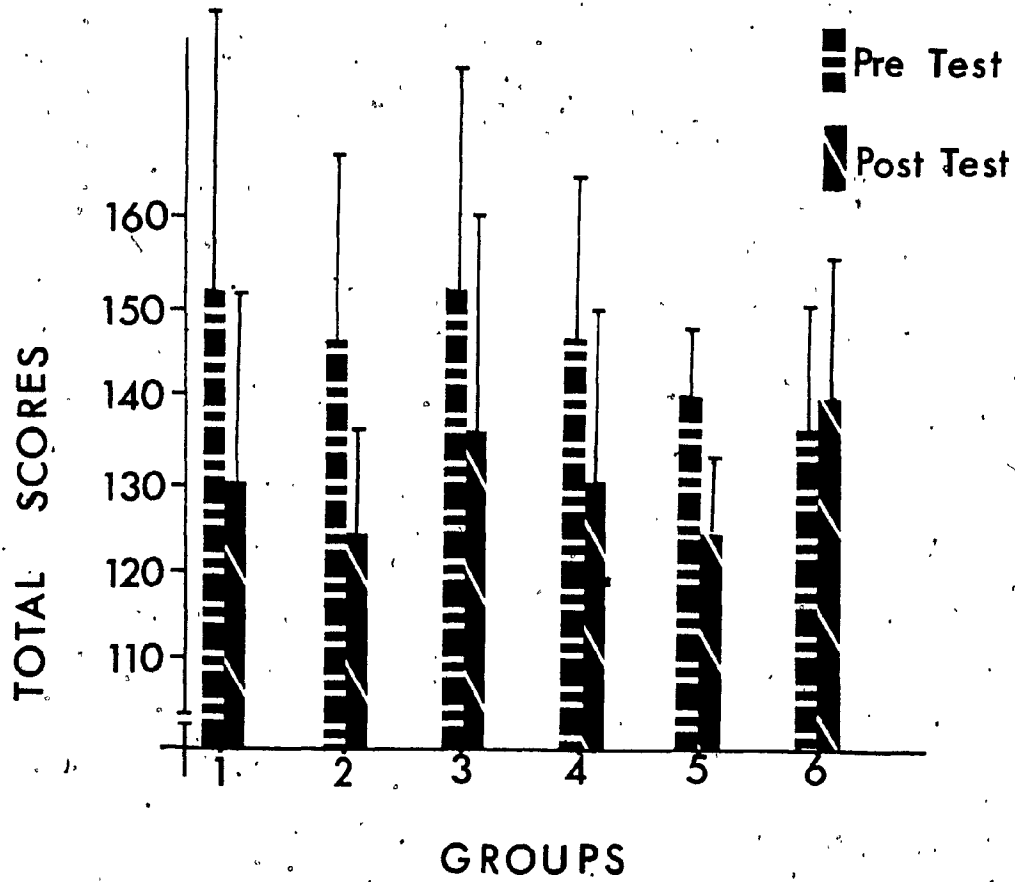


Figure 4. Pre-post measures on the What Are You Afraid Of, across the six groups.

LEGEND
Groups

- 1-PR.
- 2-V5mg
- 3-V7.5mg
- 4-PI
- 5-RC
- 6-NTC

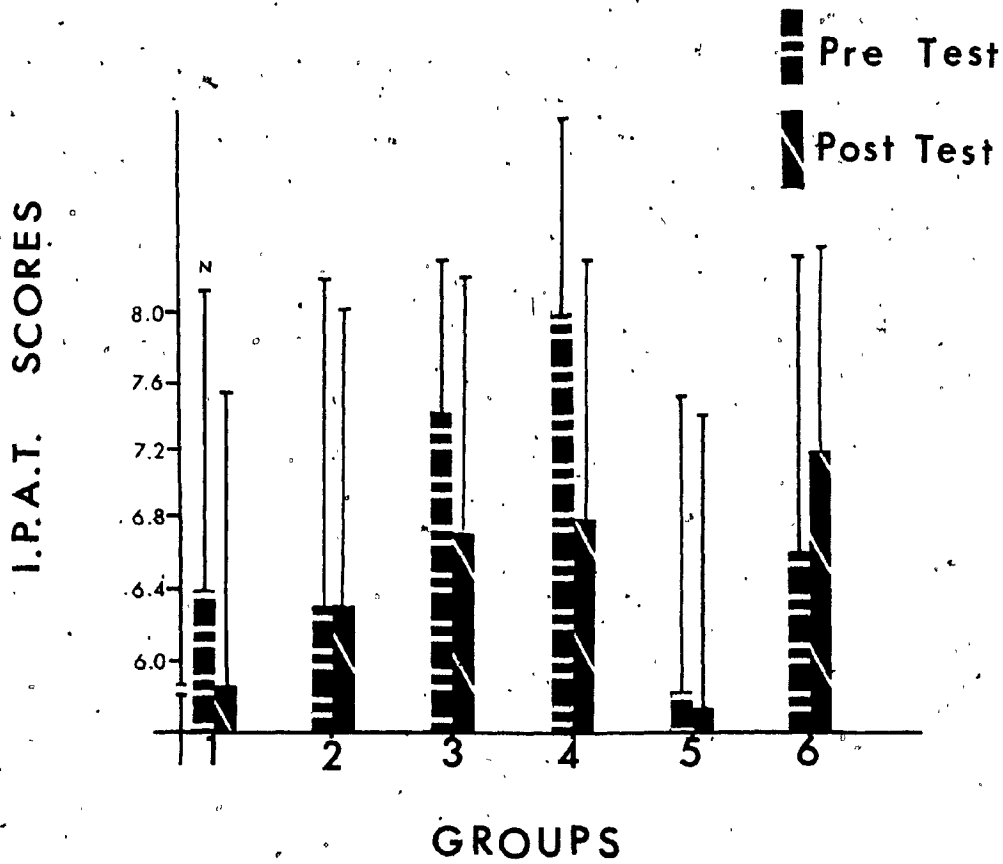


Figure 5. Pre-post measures on the I.P.A.T. across the six groups.

LEGEND
groups

1-PR

2-V5 mg

3-V75 mg

4-PI

5-RC

6-NTC

Analysis of Data From Active Treatment Sessions 1 - 6

These results are concerned with the comparative efficacy of Progressive Relaxation, Valium 5mg. and 7.5mg. and Placebo drug in reducing anxiety. The dependent variables investigated are Self Report, Frontalis E.M.G., Pulse Rate, Systolic Blood Pressure and Diastolic Blood Pressure. The data were analysed using a nested design with subjects as a replication factor (Balanova, 5.01, 1973). The analysis was performed on the difference scores obtained from the four sets of readings over the six active treatment sessions.

For this set of data, the self report of anxiety was the major dependent variable being investigated. However, an important issue being examined was the extent to which reduction in self report of anxiety paralleled reduction in one or more of the other dependent variables.

Self Report. Looking firstly at the data on self report, the results indicated a significant main effect for differences across groups, ($F = 2.56, p < .05$), for time of reading ($F = 19.840, p < .01$) and a significant group by time of reading by sessions interaction ($F = 1.388, p < .05$). (See Table 10). It might be necessary to recall that during each of the first six treatment sessions, physiological and self report data were recorded at four different times: #1, within two minutes after entering the session; #2, after the period of relaxation; #3, after the first ten minutes of exposure; and #4, at the end of the session. These are the periods referred to as the times of reading differences.

Table 10

Analysis of Variance (Balanova) on the Self Report Data (Composite Scores) from Sessions 1-6, Across the Five Treatment Groups

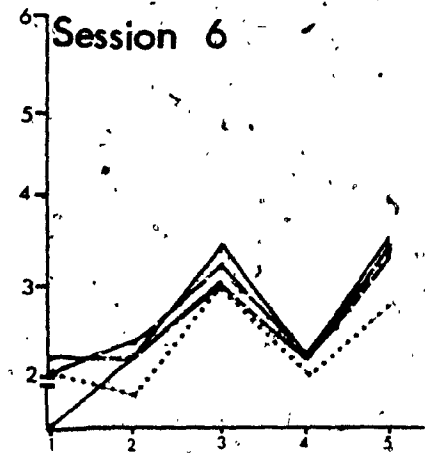
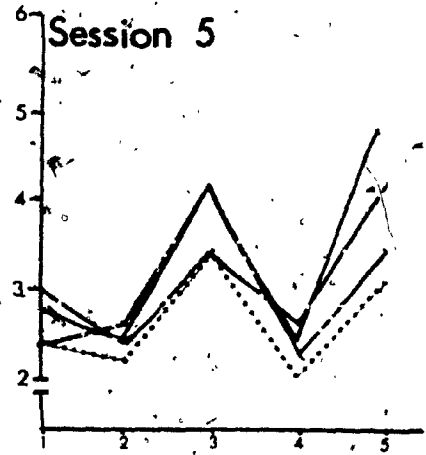
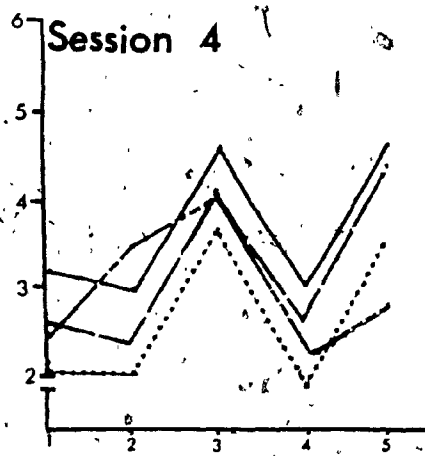
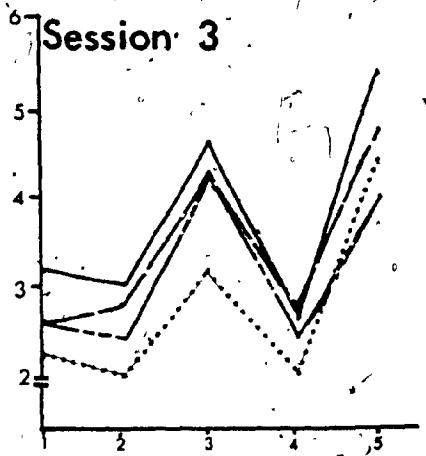
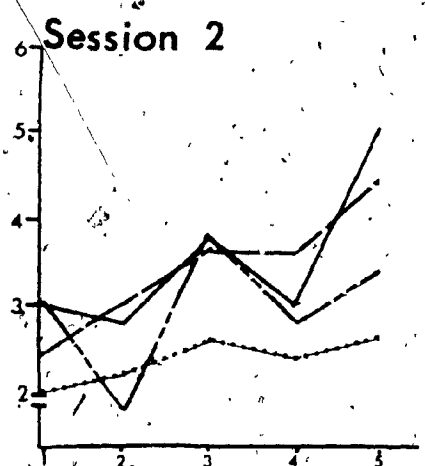
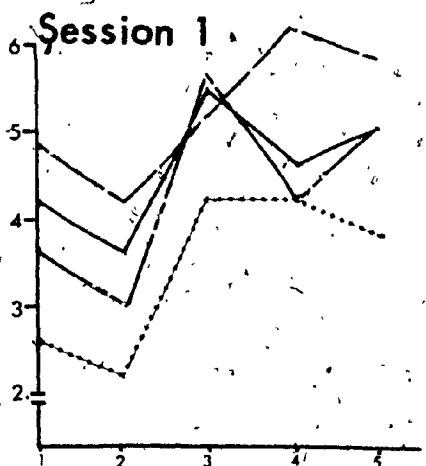
Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	413.87	103.46	2.56	.05
B	5	324.45	64.89	14.49	.001
A x B	20	91.68	4.58	1.02	n.s.
C	3	131.91	43.97	19.84	.001
A x C	12	30.34	2.52	1.14	n.s.
B x C	15	40.40	2.69	3.63	.001
A x B x C	60	61.70	1.02	1.38	.03

The data were plotted for the six sessions to visually try to identify the nature of the group by time of reading by sessions interaction. It was observed that the first two sessions showed a great amount of variability, while for the last four sessions, a more regular pattern of responses was evident. (It must be noted here, that in order, to facilitate presentation of the Self Report data, two sets of graphs have been utilised... See Figures 6 and 7). It was thought that the initial variability could be an artifact in that it might reflect the inability of the subjects to accurately identify the locus of their anxiety initially through a lack of experience in the situation. This is thought to be so because the other four sessions produced data that was similar to each other, but very different from the data collected in the first two sessions. To check for this latter possibility, the final four of these six active treatment sessions were subjected to a separate analysis of variance (Balanova). These results indicated a significant main effect for groups ($F = 3.11, p < .05$), a significant time of reading difference ($F = 13.028, p < .01$), and a significant session effect ($F = 4.732, p < .01$). (See Table 11). There was no significant interaction, and this appeared to confirm the initial speculations.

When the means were examined for the group difference, it was noted that the Placebo F, the Valium 5mg. F, and the Progressive Relaxation F groups tended to report the least anxiety over the treatment sessions, in contrast, the Valium 7.5mg. F, and the Relaxation Control F groups reported much higher anxiety. To identify

Figure 6. Readings for self report obtained from treatment sessions 1-6 across the five treatment groups.

SELF RATING OF ANXIETY



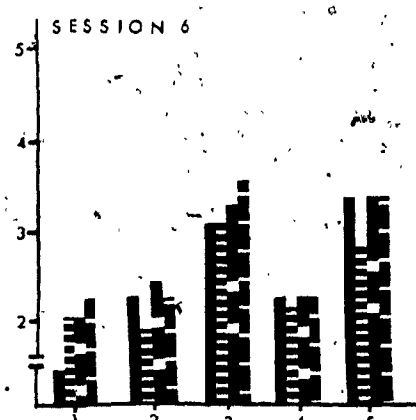
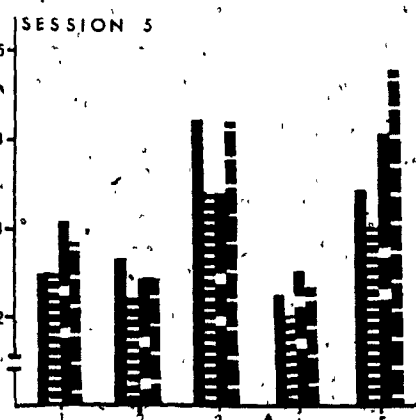
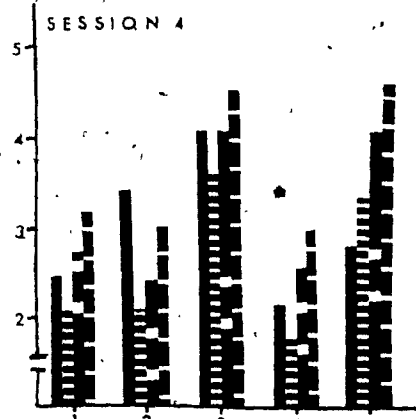
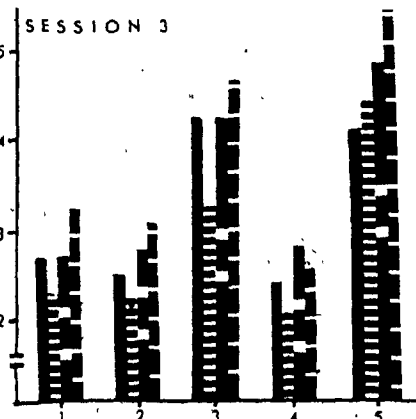
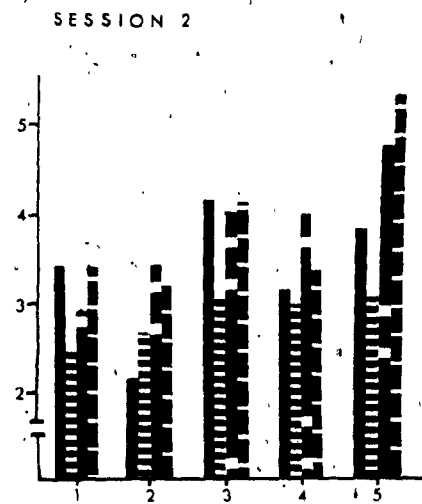
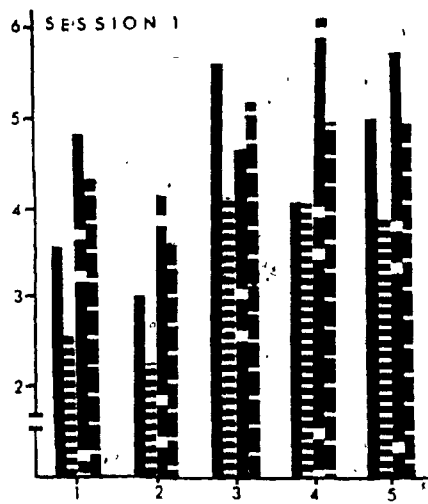
LEGEND GROUPS
 1-PR
 2-V3mg
 3-V7.5mg
 4-PI
 5-NC

GROUPS

----- First Reading
 Second Reading
 _____ Third Reading
 _____ Fourth Reading

Figure 7. Readings for self report obtained from treatment sessions 1-6 across the five treatment groups.

SELF RATING OF ANXIETY



LEGEND groups

- 1-PR
- 2-V5mg
- 3-V75mg
- 4-Pl
- 5-RC

GROUPS

- First Reading
- Second Reading
- Third Reading
- Fourth Reading

Table 11

Analysis of Variance (Balanova) on the Self Report Data (Composite Scores) from Sessions 3-6 Across the Five Treatment Groups

(A = Groups B = Sessions C = Time of Reading)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	342.22	85.55	3.11	.02
B	3	50.78	16.92	4.73	.01
A x B	12	29.05	2.42	.67	n.s.
C	3	55.26	18.42	13.02	.001
A x C	12	18.07	1.50	1.06	n.s.
B x C	9	10.79	1.19	1.69	n.s.
A x B x C	36	25.09	.69	.98	n.s.

the nature of these differences, pair-wise comparisons were performed using Tukey's post-test analysis and collapsing the data across sessions. Only the comparison between the Placebo F, and Relaxation Control F groups, attained statistical significance ($p < .05$), indicating that the Placebo F group reported significantly less anxiety than the Relaxation Control F group which reported the highest level during the treatment. The data is summarized in Table 12.

When the time of reading differences for the Self Report data were analysed by post-test (Tukey), it was observed that there was a significant difference between the base line reading, and the first reading after the relaxation period ($p < .05$), across the Progressive Relaxation F, the Valium 5mg. F, the Valium 7.5mg. F, and the Placebo F groups ($p < .05$). The Relaxation Control F group on the other hand showed a slight non significant increase in self reported anxiety. When the data are examined for report of anxiety during the exposure periods, it is observed that between the second and third readings, which corresponds to the first exposure period, there were no significant differences in reported anxiety. It was noted however that the trend was towards an increase in reported anxiety across all the groups. By the fourth reading, taken at the end of the session, all the groups showed significant increases in self reported anxiety as compared to the second reading, taken after the relaxation period. Progressive Relaxation F, Valium 5mg. F, Valium 7.5mg. F, Placebo F, ($p < .05$) and the Relaxation Control F group ($p < .01$). The data are summarized in Table 13.

Table 12

Mean Self Report Scores Obtained in Experiment Across
The Five Treatment Groups Over the First Six Treatment Sessions

Treatment Sessions	Groups				
	PR	V5	V7.5	P1	R.C.F.
1	3.91	3.2	4.97	4.94	4.94
2	3.05	2.94	3.88	3.38	4.30
3	2.69	2.58	4.13	2.47	4.77
4	2.61	2.77	4.13	2.50	3.80
5	2.75	2.47	3.88	2.36	3.88
6	2.08	2.25	3.19	2.07	3.27

Table 13

Mean of Self Report Scores at Time of Reading Obtained in
Experiment Across Five Treatment Groups During Sessions 3-6

Time of Reading	Groups				
	P.R.F.	V5 F	V7.5 F	P1. F	R.C.F.
1	2.50	2.75	3.88	2.30	3.47
2	2.25	2.05	3.38	2.0	3.50
3	2.61	2.58	3.80	2.66	4.19
4	2.77	2.69	4.27	2.63	4.58

To summarize, the Self Report data suggest that the treatment groups were all somewhat effective in reducing anxiety both over the period of the six sessions, and within the individual sessions. However, there were important differences between these groups in their effectiveness in these areas. These differences will be elaborated more fully in the discussion.

Frontalis E.M.G. When the frontalis E.M.G. data were analysed, the results indicated only a significant time of reading effect ($F = 11.57, p < .01$, see Table 14). When the data were plotted (see Figure 8), it was observed that the tendency was for the muscle tension to increase from the first reading through the second, and third to the final reading. This suggests, that muscle tension increased during the period of relaxation, as well as during the exposure periods. This was an unexpected finding. Post-test analysis (Tukey) demonstrated that pair-wise comparisons between initial and final readings, across all of the groups were significant ($p < .05$) indicating significant increases in frontalis tension across the readings. An examination of the data failed to reveal any consistent pattern across the five groups, suggesting that neither progressive relaxation, nor diazepam were demonstrably more effective in reducing the frontalis tension.

Pulse Rate. The data for the Pulse Rate indicated a significant time of reading effects ($F = 62.27, p < .01$), and a significant group by time of reading interaction ($F = 3.20, p < .01$) (Table 15).

Table 14

Analysis of Variance (Balanova) on the Frontalis E.M.G. Data
 (Composite Scores) Obtained in the Experiment
 (A = Groups B = Sessions C = Time of Reading)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A	4	9381.34	2345.31	.34	n.s.
B	5	6794.96	1358.99	1.60	n.s.
A x B	20	82342.20	1117.11	1.32	n.s.
C	3	24056.60	8018.88	11.56	.001
A x C	12	9737.74	811.47	1.17	n.s.
B x C	15	3302.82	220.18	.49	n.s.
A x B x C	60	21175.80	352.93	.78	n.s.

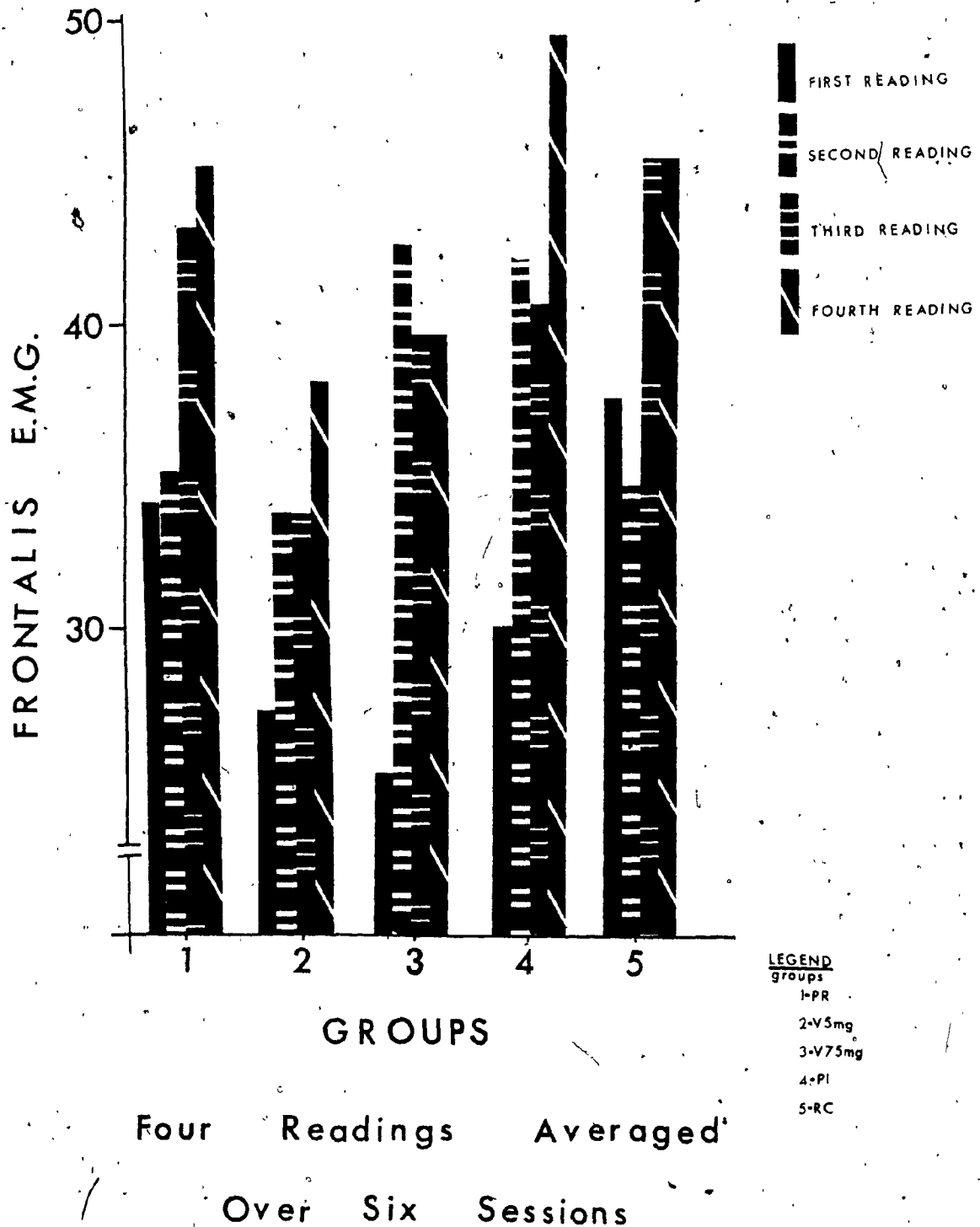


Figure 8. Frontalis E.M.G. data showing the mean scores at four times of reading over six treatment sessions across the five treatment groups.

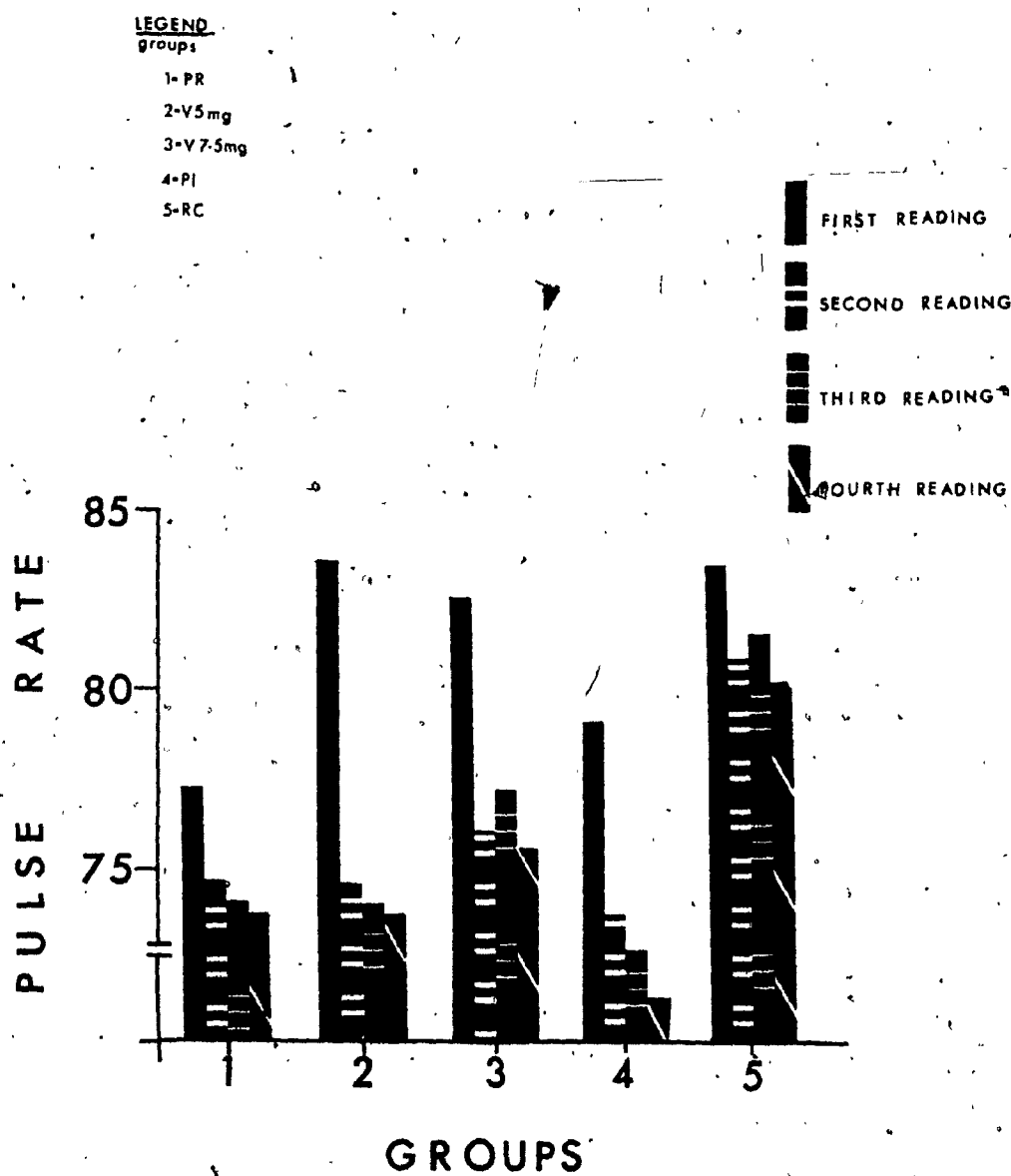
Table 15

Analysis of Variance (Balanova) on the Pulse Rate Data
 (Composite Scores) Obtained in the Experiment
 (A = Groups B = Sessions C = Time of Reading)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	7563.99	1891.01	1.25	n.s.
B	5	726.89	145.37	.92	n.s.
A x B	20	3081.21	154.06	.98	n.s.
C	3	6640.40	2213.47	62.26	.001
A x C	12	1366.35	113.86	3.20	.001
B x C	15	175.67	11.71	.59	n.s.
A x B x C	60	1090.41	18.17	.91	n.s.

Plotting the graph for the group by time of reading interaction, it seemed likely that the source of this interaction might be due to the difference observed between the base line reading, and the other three readings (see Figure 9). To test this possibility, it was first necessary to discover whether any of the last three readings differed significantly from any other. These three readings were therefore analysed by Scheffé post-test, and it was observed that they did not differ significantly from each other. For the next analysis, in order to identify the source of the significant interaction, it was decided to compare the base line readings with the average of these last three readings, using Scheffé post-test analysis. The results indicated that the Valium 5mg. F, the Placebo F and the Valium 7.5mg. F groups all showed significant reduction in pulse rate from the base line reading ($p < .01$). The Progressive Relaxation F and the Relaxation Control F groups exhibited non significant reduction from base line. Pair-wise comparisons were performed (Scheffé) to identify the order of magnitude of the reduction. It was observed that the Valium 5mg. F group attained a degree of reduction of pulse rate that was significantly greater than both the Valium 7.5mg. F, and the Placebo F groups ($p < .05$). The difference between the Valium 7.5mg. F and the Placebo F groups did not attain statistical significance.

Systolic Blood Pressure. From the analysis of the data on systolic blood pressure, the results showed a significant sessions effect ($F = 2.55, p < .05$), a significant sessions by group interac-



Four Readings Averaged
Over Six Sessions

Figure 9. Pulse Rate data showing the mean scores for four times of reading over six treatment sessions across the five treatment groups.

tion, ($F = 2.20$, $p < .01$), a significant time of reading effect ($F = 52.56$, $p < .01$) and a significant group by time of reading interaction ($F = 2.75$, $p < .01$). (See Table 16). After the data were plotted, a post-test analysis (Scheffé) was performed to reveal the source of the group by time of reading interaction. As with the pulse rate data, it was observed that the last three readings did not differ significantly from each other and that the significant interaction observed could be due to the difference between the base line reading and the last three readings (see Figure 10). Further analysis (Scheffé) comparing the base line reading with the average of the last three readings indicated that the Progressive Relaxation F group, the Valium 5mg. F group, the Valium 7.5mg. F group and the Placebo F group all showed a significant reduction in systolic blood pressure from base line ($p < .01$). By contrast, the Relaxation Control F group showed a non significant pre-post reduction. Pair-wise comparisons (Scheffé) revealed that while the largest reduction was on the Valium 5mg. F group, this group did not differ significantly from the Valium 7.5mg. F group. However, both these groups were significantly more efficient in systolic blood pressure reduction than the Progressive Relaxation F, the Placebo F and the Relaxation Control F groups ($p < .01$). These latter three groups did not differ significantly from each other. The data are summarized in Table 17.

For the sessions by group interaction, post-test analysis (Scheffé) revealed that on session one, the three 'drug' groups all recorded significantly higher incoming systolic blood pressure than

Table 16

Analysis of Variance (Balanova) on the Systolic Blood Pressure

Data (Composite Scores) Obtained in the Experiment.

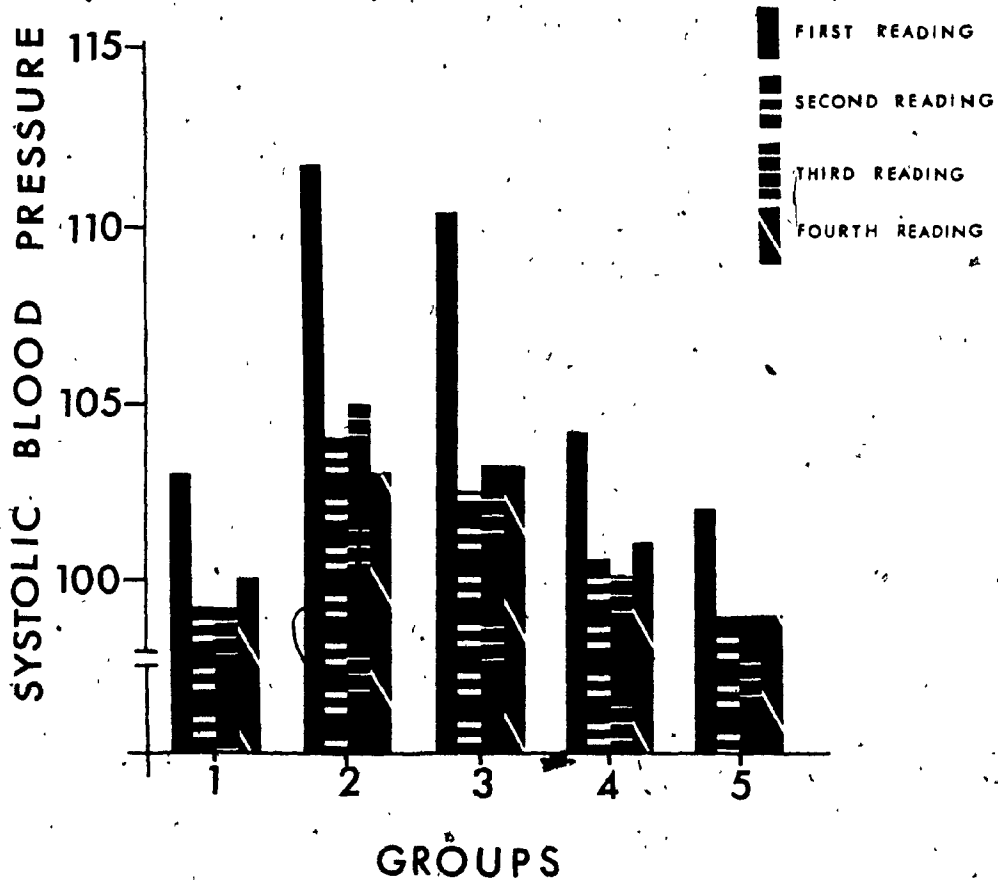
(A = Groups B = Sessions C = Time of Reading)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	6442.83	1610.71	1.05	n.s.
B	5	816.63	163.32	2.55	.02
A x B	20	2821.64	141.08	2.20	.01
C	3	5599.18	1866.39	52.55	.001
A x C	12	1173.76	97.81	2.75	.01
B x C	15	409.73	27.31	1.13	n.s.
A x B x C	60	1406.30	23.43	.97	n.s.

LEGEND

GROUPS

- 1-P R
- 2-V 5mg
- 3-V 7.5mg
- 4-Pl
- 5-RC



Four Readings Averaged
Over Six Sessions

Figure 10. Systolic Blood Pressure data showing the mean scores for times of reading over six treatment sessions across the five treatment groups.

Table 17

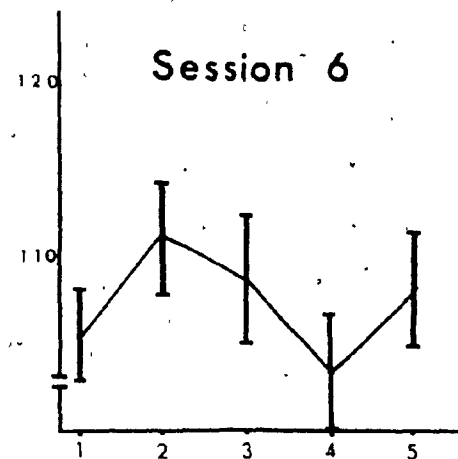
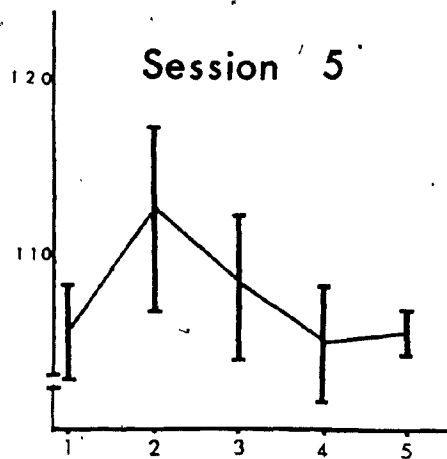
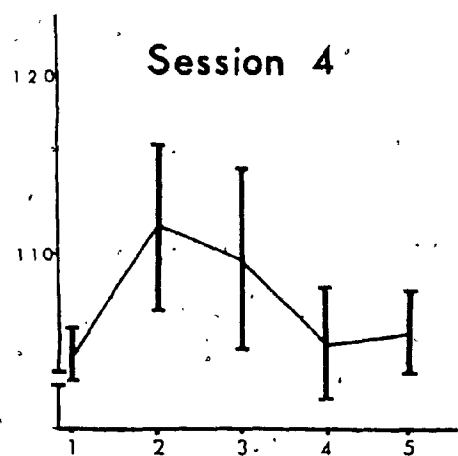
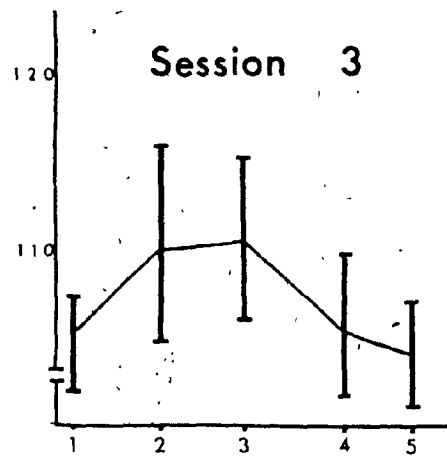
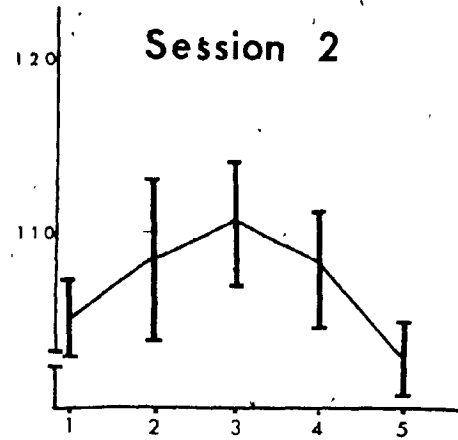
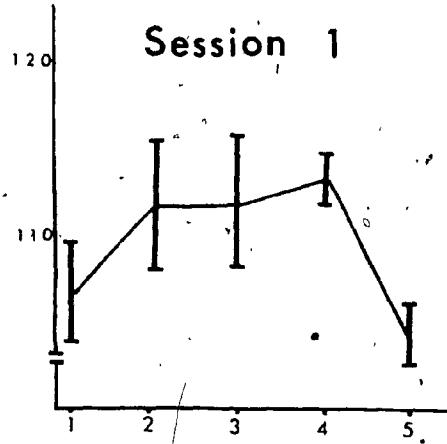
Mean Systolic Blood Pressure Scores Obtained Across Five Treatment Groups for Time of Reading Obtained in Sessions 1-6

Time of Reading	Groups				
	PR. F	V5. F	V7.5 F	Pl. F	RC. F
1	108.48	117.07	115.96	109.77	107.14
2	104.29	107.7	107.81	105.74	104.55
3	104.35	110.07	108.37	105.13	104.42
4	104.66	108.0	108.0	106.61	104.72

both the Progressive Relaxation F and the Relaxation Control F groups ($p < .01$). During sessions two and three, the Placebo F group began to reduce its incoming systolic blood pressure so that while the Valium 5mg. F, and the Valium 7.5mg. F groups still had a significantly higher incoming systolic blood pressure than both Progressive Relaxation F and Relaxation Control F groups, the Placebo group now did not. This trend continued through sessions four and five, and by session six, on incoming systolic blood pressure, both the Valium 5mg. F and the Valium 7.5mg. F groups were now significantly higher than the Placebo F group ($p < .05$). At this point however, they did not differ significantly from the Progressive Relaxation F and the Relaxation Control F groups (see Figure 11).

Figure 11. Systolic blood pressure showing the average of four readings over six sessions across the five treatment groups.

SYSTOLIC BLOOD PRESSURE



LEGEND
groups

1-PR

2-V5mg

3-V7.5

4-PI

5-RC

GROUPS

Diastolic Blood Pressure. The diastolic blood pressure analysis indicated no statistically significant reduction either across groups or over time (see Table 18). However, the pattern of reduction noted was similar to that observed across the systolic blood pressure. The Valium 7.5mg. F and the Valium 5mg. F groups showed the largest reductions across readings, while the Placebo F group showed the largest reduction for incoming blood pressure over sessions. In no case however, was any of these differences statistically significant.

Thus, on pulse rate, systolic and diastolic blood pressure, the most effective reduction within sessions occurs within the two valium groups. However the placebo group was also quite effective, particularly in reducing blood pressure over the entire six sessions. In general, both the Progressive Relaxation F and the Relaxation Control F groups had little effect in reducing pulse rate and blood pressure.

Analysis of Data From Sessions 7 and 8

As noted before, these sessions were designed to test whether the pattern of response observed during sessions one to six, when the anti-anxiety adjuncts were employed would carry over to sessions seven and eight where flooding only was employed. These latter sessions being given one and two weeks later respectively. In both of these sessions, the relaxation phase was discontinued, so that measures were recorded pre-exposure and post-exposure.

Table 18

Analysis of Variance (Balanova) on the Diastolic Blood Pressure

Data (Composite Scores) Obtained in the Experiment

(A = Groups B = Sessions C = Time of Reading)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	9446.82	2631.70	1.14	n.s.
B	5	5475.34	1095.07	1.20	n.s.
A x B	20	23770.20	1188.51	1.30	n.s.
C	3	1678.13	559.37	1.69	n.s.
A x C	12	4430.89	369.24	1.11	n.s.
B x C	15	4877.55	325.17	1.01	n.s.
A x B x C	60	18707.71	311.79	.96	n.s.

Session 7. There were no significant interactions observed, but the analysis of variance data yielded significant pre-post differences for the frontalis E.M.G. ($p < .01$), the pulse rate ($p < .01$) and the systolic blood pressure data ($p < .01$). (See Tables 19-23). Post-test analysis (Tukey) revealed the following. On the E.M.G. data, the Relaxation Control F and the Placebo F groups showed significant pre-post increases in frontalis E.M.G. ($p < .05$). The other three groups showed non-significant increases from pre to post-test. The pulse rate data indicated significant pre-post decrease on the Valium 7.5mg. F group. Of the other groups, the Relaxation Control F group did not change from pre to post-test, while the other three groups showed non-significant pre-post decreases in pulse rate. On systolic blood pressure, post-test (Tukey) revealed a significant pre-post reduction for the Valium 5mg. F group only. By contrast, the Placebo F group remained virtually unchanged, while the other groups showed non-significant pre-post reduction. For both the self report and diastolic blood pressure data, there were no significant pre-post differences across the groups. (The data are summarized in Table 24). Before commenting on these findings, it is necessary to look at the results from Session #8.

5

Table 19

Analysis of Variance (Balanova) on the Self Report

(Pre-Post) Data Obtained in Session 7

(A = Groups C = Pre-Post Reading)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	32.511	8.12	1.38	n.s.
C	1	.54	.54	.90	n.s.
A x C	4	.95	.23	.39	n.s.

Table 20

Analysis of Variance (Balanova) on the Frontalis

E.M.G. Data Obtained in Session 7

(A = Groups C = Pre-Post Reading)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	4439.21	1109.81	1.19	n.s.
C	1	3039.21	3039.21	11.57	.001
A x C	4	1378.96	319.73	1.21	n.s.

Table 21

Analysis of Variance (Balanova) on the Pulse Rate

(Pre-Post) Data Obtained in Session 7

(A = Groups C = Pre-Post Reading)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	235.73	58.93	.19	n.s.
C	1	230.40	230.40	12.40	.001
A x C	4	66.48	16.62	.89	n.s.

Table 22

Analysis of Variance (Balanova) on the Systolic Blood Pressure

(Pre-Post) Data Obtained in Session 7

(A = Groups C = Pre-Post Reading)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	1124.71	281.17	1.50	n.s.
C	1	291.60	291.60	15.41	.001
A x C	4	73.51	18.37	.97	n.s.

Table 23

Analysis of Variance (Balanova) on the Diastolic Blood

Pressure (Pre-Post) Data Obtained in Session 7

(A = Groups C = Pre-Post Readings)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A	4	109.33	27.33	.24	n.s.
C	1	12.84	12.84	.94	n.s.
A x C	4	29.15	7.28	.53	n.s.

Table 24

Mean Pre-Post Scores on Frontalis E.M.G., Pulse Rate and
Systolic Blood Pressure Obtained in Experiment During Session 7

Frontalis E.M.G. Data

	Groups				
	PR. F	V5 F	V7.5 F	P1. F	RC. F
Pre	32.2	24.3	21.5	31.2	30.7
Post	39.0	26.6	30.5	50.3	52.3

Pulse Rate Data

	Groups				
	PR. F	V5 F	V7.5 F	P1. F	RC. F
Pre	83.55	84.4	83.5	82.6	85.33
Post	79.11	80.8	78.6	79.5	85.33

Systolic Blood Pressure Data

	Groups				
	PR. F	V5 F	V7.5 F	P1. F	RC. F
Pre	109.77	115.77	114.88	105.55	106.66
Post	106.22	110.22	110.44	105.33	102.44

Session 8. Analysis of variance (Balanova) indicated a significant pre-post difference for E.M.G., ($F = 5.86, p < .02$) a significant pre-post difference ($F = 17.26, p < .001$), and a significant interaction ($F = 2.80, p < .03$) for pulse rate, and a significant pre-post difference for systolic blood pressure ($F = 15.63, p < .001$) (See Tables 25-29). Post-test analysis (Tukey) revealed the following. On systolic blood pressure, both the Progressive Relaxation F and the Placebo F groups showed significant ($p < .05$) pre-post reduction. The other three treatment groups showed non-significant reduction. The pulse rate data indicated significant ($p < .05$) pre-post reduction on both the Progressive Relaxation F and the Valium 5mg. F groups. Both the Relaxation Control F and the Valium 7.5mg. F groups remained virtually unchanged from pre-to post test, while the Placebo F group showed a non-significant reduction. The E.M.G. data showed increases in frontalis tension across four of the treatment groups, the Progressive Relaxation F group, the Valium 5mg. F group, the Valium 7.5mg. F group and the Placebo F group. Of these groups, the Valium 7.5mg. F group registered a significant pre-post increase in frontalis tension. By contrast, the Relaxation Control F group showed a slight non-significant decrease on this measure.

As was noted before from the Session 7 results, there were no significant pre-post effects for the self report and diastolic blood pressure data.

As can be seen when the pre-post means are examined (see Table 30), the results reflect the same general trends to reduction

Table 25

Analysis of Variance (Balanova) on the
Self Report Data Obtained in Session 8

(A = Groups C = Pre-Post Readings)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A	4	55.62	13.90	1.77	n.s.
C	1	1.11	1.11	2.05	n.s.
A x C	4	3.22	.80	1.48	n.s.

Table 26

Analysis of Variance (Balanova) on the Frontalis E.M.G.

(Pre-Post) Data Obtained in Session 8

(A = Groups C = Pre-Post Readings)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A	4	4823.51	1205.88	1.18	n.s.
C	1	2300.28	2300.28	5.86	.02
A x C	4	1333.11	333.27	.84	n.s.

Table 27

Analysis of Variance (Balanova) on the Pulse Rate

(Pre-Post) Data Obtained in Session 8

(A = Groups C = Pre-Post Readings)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	472.71	118.17	.56	n.s.
C	1	321.11	321.11	17.26	.001
A x C	4	208.88	52.22	2.80	.03

Table 28

Analysis of Variance (Balanova) on the Systolic Blood

Pressure (Pre-Post) Data Obtained in Session 8

(A = Groups C = Pre-Post Readings)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>P</u>
A	4	883.73	220.93	1.22	n.s.
C	1	336.40	336.40	15.63	.001
A x C	4	69.15	17.28	.80	n.s.

Table 29

Analysis of Variance (Balanova) on the Diastolic Blood

Pressure (Pre-Post) Data Obtained in Session 8

(A = Groups C = Pre-Post Readings)

Source	<u>df</u>	<u>SS</u>	<u>MS</u>	<u>F</u>	<u>p</u>
A	4	678.40	169.60	1.41	n.s.
C	1	2.17	2.17	.21	n.s.
A x C	4	50.48	12.62	1.23	n.s.

Table 30

Mean Difference Scores on Frontalis E.M.G., Pulse Rate and
Systolic Blood Pressure Data Obtained in Experiment During Session 8

Frontalis E.M.G. Data

Groups

	PR. F	V5mg F	V7.5mg F	Pl. F	RC. F
Pre	31.77	20.77	37.0	31.66	32.0
Post	42.88	31.22	58.33	42.2	29.1

Pulse Rate Data

Groups

	PR. F	V5mg F	V7.5mg F	Pl. F	RC. F
Pre	84.0	88.4	80.8	82.6	86.0
Post	77.3	80.8	80.4	76.6	85.7

Systolic Blood Pressure Data

Groups

	PR. F	V5mg F	V7.5mg F	Pl. F	RC. F
Pre	110.88	117.33	113.55	112.2	108.88
Post	105.11	114.66	111.11	106.0	106.66

as obtained during the active treatment phase. However, despite the significant pre-post findings, the results are too varied, and most of the differences too small, for a definitive statement to be made on the issue. Furthermore, none of these differences observed, were reflected in self report of anxiety.

Correlations. Two different sets of correlations were performed on the data. In the first, scores on the Fear Survey Schedule (F.S.S.) pre-test were correlated with pre-test scores on the I.P.A.T. The results yielded a correlation of $r = .42$ suggesting a degree of overlap in both measures. However as noted before, post-test scores on the F.S.S. showed a marked decrease, while the I.P.A.T. did not show similar pre-post decrease. These findings raise important questions concerning the nature of these measures.

The second set of correlations looked at the relationship between self report of anxiety and physiological measures, using data from the screening session, and the final session. As the summary of the data from Tables 31 and 32 indicate, there were no consistent relationships between self report and physiological indices of anxiety, and this finding is quite consistent with results from previous studies.

Table 31

Correlations Between Subjective Ratings of Anxiety and Four
Autonomic Measures from Screening and Post-Test Data
Obtained Across Fifty Four Subjects in Experiment

Data Set I (Screening)

Autonomic Measures

Self Rating	E.M.G.	Pulse	S.B.P.	D.B.P.
1	.01	.06	-.12	.07
2	-.01	-.11	-.12	-.14

Data Set II (Post Test)

Autonomic Measures

Self Rating	E.M.G.	Pulse	S.B.P.	D.B.P.
1	.26	.12	.18	.18
2	.39*	.02	-.23	-.26

* $P < .05$

Table 32

Correlations Between Subjective Ratings of Anxiety and Autonomic Indices Across Six Groups for Post-Test Data

	PR. F Group			PL. F Group					
	E.M.G.	Pulse	S.B.P.	D.B.P.	E.M.G.	Pulse	S.B.P.	D.B.P.	
SR ₁	.42	.60	-.35	-.04	.38	-.36	-.30	.56	
SR ₂	.44	.79	-.07	-.09	.45	-.51	-.22	.20	
	V5mg. F Group			RCF Group					
	E.M.G.	Pulse	S.B.P.	D.B.P.	E.M.G.	Pulse	S.B.P.	D.B.P.	
SR ₁	-.09	-.55	-.45	-.48	.18	.45	.34	-.00	
SR ₂	-.43	-.04	.05	-.38	.20	-.26	.01	-.03	
	V7.5mg. F Group			MTC Group					
	E.M.G.	Pulse	S.B.P.	D.B.P.	E.M.G.	Pulse	S.B.P.	D.B.P.	
SR ₁	-.66*	-.08	-.25	-.38	-.44	.26	-.40	-.17	
SR ₂	-.34	-.42	-.09	.12	-.06	.16	-.43	-.54	

*p < .05

**p < .01

Discussion

The present research investigated two separate but related questions. One question investigated was the treatment of phobias by flooding. There were two issues, (a) whether treatment of phobias by flooding was effective, and (b) whether flooding was more effective when combined with an anti-anxiety adjunct. At a broader level the study also addressed itself to the possible clinical usefulness of anti-anxiety adjuncts in flooding.

The second question investigated was the reduction of anxiety. Again, two issues were examined. The first concerned the comparative efficacy of progressive relaxation, diazepam and placebo drug in reducing anxiety. The second sought to determine the mode of action of these treatments.

Given the fact that the above questions are touching on different areas, they will be discussed separately, and an attempt will be made to integrate them.

The Treatment of Phobias. In testing the first hypothesis, the results show clearly that flooding was an effective treatment for the reduction of phobic fears, when the five treatment groups were compared to the no treatment control group on the behavioural avoidance test. According to the definition of phobic response employed in this study, all the subjects in the active treatment groups improved to the extent that they could no longer be defined as being phobic. More specifically, the minimum number of steps they could now complete

on the B.A.T., would have initially excluded them from the study. Of the other dependent measures employed, similar results in the sense of overall reduction were obtained on the Self Rating of Phobic Avoidance Scale, the Fear Survey Schedule, and on the What Are You Afraid Of. It is interesting to note at this point that on the I.P.A.T. which is designed to measure trait anxiety (Cattell, 1957), there were no significant pre-post differences observed. This finding will be elaborated at a subsequent point. It must also be noted that there was a close relationship between behavioural change and self reported reduction of anxiety in this study.

The above results are quite consistent with the notion that flooding or exposure is an effective treatment for phobic responses. However, they also suggest that the experience of heightened anxiety might be, as noted by Marks (1975), an unfortunate by-product of exposure. In response to this question of whether flooding is more effective when combined with an anti-anxiety adjunct, the data for the five treatment groups were examined without reference to the no-treatment control group. It was decided to compare the groups on both the Behavioural Avoidance Test and the Self Rating of Phobic Avoidance as an examination of these variables would allow comparison with previous studies. In doing this, it was noted that there were differences between the treatment groups on these dependent measures, and that these differences raised important questions which may have possible clinical implications. These findings can be summarized as follows: (a) Of the five treatment groups, the Valium 5mg.F., and the Progressive Re-

on the B.A.T., would have initially excluded them from the study. Of the other dependent measures employed, similar results in the sense of overall reduction were obtained on the self rating of phobic avoidance scale, the fear survey schedule, and on the What Are You Afraid Of. It is interesting to note at this point that on the I.P.A.T. which is designed to measure trait anxiety (Cattell, 1957), there were no significant pre-post differences observed. This finding will be elaborated at a subsequent point. It must also be noted that there was a close relationship between behavioural change and self reported reduction of anxiety in this study.

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laxation F groups were the most effective in reducing avoidance behaviour, as determined by performance on the Behavioural Avoidance test. (b) When self rating of phobic avoidance rather than behavioural avoidance was the outcome criterion, significant pre-post differences were obtained from the Relaxation Control F., the Valium 5mg.F., and the Placebo F groups. Non significant pre-post differences were obtained from the Progressive Relaxation F and the Valium 7.5mg F groups.

These results while not clear cut, nevertheless incorporate many of the seemingly contradictory findings in the literature on the question of flooding combined with an anti-anxiety adjunct. The main problem with these previous studies, would appear to be related to dose levels, and the possible limitations of experimental design in general. For example, when the two active drug groups were compared, it can be seen that the lower dosage (5mg.) was more efficient than the higher dosage (7.5mg.) in reducing fear both on the behavioural avoidance test and the self report data. This finding is similar to that of Marks et al. (1972). They found that flooding under waning diazepam (valium) was more effective than flooding under peak diazepam. Thus in both studies, the higher dose level or the more active level of drug resulted in a less efficient reduction.

Another example can be seen by looking at the results obtained by Whitehead et al. (1978). They found that flooding with chronic administration of diazepam (15mg./day), was not more effective than flooding with placebo, when behavioural avoidance was used as the outcome criterion. The present study used acute rather than

chronic administration of diazepam; nevertheless an argument can still be made for making this comparison. It is possible that due to factors involved in the half-life of diazepam and its steady state properties, a chronic administration of 15mg. a day of diazepam has a steady state concentration that is roughly equivalent to that of a peak level of an acute administration of 7.5mg. diazepam (Greenblatt & Shader, 1979). If this is true, it is not surprising that in the present study, as in the Whitehead one, there were no differences between the Valium 7.5mg. F group and the Placebo F group, when they were compared on the Behavioural Avoidance test.

A further example of these seeming inconsistencies in the literature and this study, is the finding that the Valium 5mg. F group was more effective than the Placebo F group on behavioural avoidance. This finding can be regarded as being consistent with the findings of Johnson and Gath (1973) and Marks et al. (1972). Both of these studies reported that flooding under diazepam was more effective than flooding with a placebo, and in both studies outcome was in terms of behaviour.

As noted before, the central point to be made is that most of the discrepant findings can be explained in terms of dose levels, and the limitations of the experimental designs employed. In addition one major factor not taken into consideration in many of these studies, is that placebos (in anxiety research) are not non-treatment, but quite often are as effective as anti-anxiety agents in reducing anxiety (McNair et al., 1965; Reynolds et al., 1965). Looking at the present

study it is apparent that if one attempts to assess the results overall, a definitive statement is difficult. By contrast, if the data are examined in terms of behavioural avoidance and self rating as separate dimensions, the picture becomes less confused.

Looking first at the area of behavioural avoidance, the results of the present study suggest that when behaviour is the outcome criterion, a certain degree of anxiety reduction, however induced, leads to a more efficient reduction of avoidance behaviour. This is aptly demonstrated by the results of both the Valium 5mg. F group and the Progressive Relaxation F group. Furthermore, when one looks at the discomfort level or degree of anxiety experienced during the experiment, it is observed that these two groups were among the three that experienced the least discomfort over the duration of the experiment, the other being the placebo group. By contrast, the Relaxation Control F. and the Valium 7.5mg. F groups experienced higher levels of discomfort over the sessions.

These results at first glance would appear to confirm the position that the evoking of anxiety during flooding is unnecessary (Foa et al., 1977); and suggest that reducing anxiety during exposure should be the method of choice regarding treatment. This would be the logical conclusion because the subjects treated under these conditions do better on post test avoidance, and in addition, they experienced less discomfort during treatment. Before making that conclusion however one should examine the results from the Self Rating of Phobic Avoidance Scale.

When the self rating of phobic avoidance is used as the outcome criterion a slightly different picture emerges. Here it is observed that the Relaxation Control F group shows the most efficient reduction, though the Valium 5mg. F and the Placebo F groups also effectively reduced this self rating. Thus even though the Relaxation Control F group reported the highest level of discomfort during the treatment, on post-assessment the subjects in this group saw themselves as being less afraid than other treatment groups, even those that subsequently performed somewhat better on the behavioural post test. This finding is similar to that of Chambless and Foa (1979). In their study, they used brevitall in the treatment of agoraphobics and found that differences between the drug and non-drug groups were significant only on clients' ratings of fear, the non-drug group reporting significantly less fear on post test. The data would suggest that at least for self rating under some conditions, the experience or confrontation with anxiety during exposure can enhance treatment effects for phobics.

In attempting to explain these results, one is presented with a possible dichotomy between anxiety as it is expressed in behaviour, and anxiety as it is expressed in cognitions. One possible conclusion is that for fears which are producing behavioural avoidance, and where the clinical treatment target is reducing this avoidance, then a reduction of the anxiety experienced during exposure might facilitate outcome. For fears where cognitive avoidance or cognitive ruminations are the dominant clinical symptoms and therefore the prime

treatment target, anxiety experienced during exposure might facilitate outcome. Support for such a dichotomy can be implied from the literature on fear survey scales and anxiety scales. The fear survey scales were developed to assess change in phobic behaviour and generalized anxiety (Wolpe & Lang, 1964), while anxiety scales have been designed to measure state or trait anxiety. Nevertheless studies that have compared Fear Survey Schedules (I & II) with the Taylor Manifest Anxiety Scale (Taylor, 1951, 1953) report correlations ranging from .38 to .80 (Geer, 1965; Grossberg & Wilson, 1965; Hersen, 1971; Lang & Lazovik, 1963; Suinn, 1969). Despite the fact that these correlations were significant, such a wide range suggests that the domain covered by these scales, while overlapping, were not equivalent. In the present study a significant correlation ($r = .42$) was obtained between the scores on the Fear Survey Schedule (F.S.S.) (Wolpe & Lang, 1964) and the I.P.A.T. (Cattell, 1957). However, on pre to post-test the I.P.A.T. did not demonstrate any significant change while both the F.S.S. which is predominantly an experimental scale, and the W.A.Y.A.O. (Sutherland & Amit, 1975) which is a clinical instrument showed consistent though non-significant pre-post reduction.

The results from the fear scales suggest the possibility of a certain degree of generalization from the treated fear to other non-treated fears. This observation is consistent with the findings of Watson and Marks (1971); they demonstrated that treating irrelevant fear cues significantly reduced other non-treated phobias. Similarly the experiments by Meichenbaum (1971) suggest that individuals might be

immunized to stressors, including phobias by subjecting them to stress that is not connected with their particular fear. However, given the lack of generalization that occurred on the I.P.A.T., it is only possible to conclude that (in this study at least) the two different types of scales were measuring semi discrete domains. This leads to at least three possibilities. (i) That fears and anxieties are totally separate entities. (ii) That fears and anxieties exist along a continuum and shade one into the other, or (iii) That fears and anxieties are different representations or different aspects of the same phenomena. The data from this study again illustrates this as a fundamental theoretical problem, but unfortunately does little to clarify the issue.

The question of anxiety evocation and its role in the treatment of fears by flooding appears to be a complex one as can be seen from the above discussion, and this is cogently demonstrated by the differential response of the two groups that experienced the highest anxiety during the treatment. Looking at the data it is observed that the Valium 7.5mg. F group experienced anxiety over the sessions which was comparable to that experienced in the flooding only group. In the Relaxation Control F. group, the experience of high anxiety led to a self-evaluation of competence on post-test; in the case of the Valium 7.5mg F group, a similar enhancement did not occur. It seems likely that cognitions play a role in this phenomenon, however, this issue will be elaborated more fully when the question of anxiety reduction is discussed.

In summary, for the subject, confronting and overcoming his fear is obviously reinforcing and it seems that under somewhat reduced anxiety, behavioural responses may be facilitated. It also seems likely, and clinical reports would corroborate this, that in some cases, the experienced anxiety might be so high, that some reduction would be necessary in order to allow the subject to enter the feared situation so that the necessary exposure could take place. In cases such as this some preliminary pretreatment with an anti-anxiety adjunct may be mandatory. On the other hand, the data also suggests that too great a reduction in the experienced anxiety may possibly interfere with the individual's evaluation of his progress, to the extent that he might be receiving feedback that is at variance with his cognitive appraisal of his fear. Particularly in drug induced relaxation, the individual may attribute his improved performance to the drug rather than to himself. Some support for this latter assumption can be observed when one compares the results from the Valium 7.5mg. F group and the Valium 5mg. F group. On all the outcome measures, the subjects in the higher drug group performed less adequately than subjects in the lower drug group. Furthermore, in the inquiry following the completion of the experiment, subjective reports of the subjects tended to support the notion that response to the higher drug dose might have interfered with the subjects' perceptions.

The Reduction of Anxiety

In this section, the question of the comparative efficacy of progressive relaxation, diazepam and placebo drug in the reduction of anxiety will be considered. An attempt will be made to integrate these results along with those of the two previous studies in this series into a comprehensive framework.

In the present study which was looking at the question of the comparative efficacy of the treatments in the reduction of anxiety, one cannot look at any one global measure. There seems to be a number of different aspects to be considered, which can be divided into four areas. (1) The reduction of anxiety felt on simply entering the experimental situation over the first six sessions (Incoming Anxiety). (2) The reduction of anxiety that occurred during the 20 minutes after the administration of the anti-anxiety adjunct, but before the exposure period (Anticipatory Anxiety). (3) The reduction of anxiety during the actual periods of exposure (Exposure Anxiety), and (4) The carry over effect of the first six sessions to sessions seven and eight when the anti-anxiety adjuncts were no longer employed (Carry Over Anxiety).

Reduction of Incoming Anxiety. When the reduction of anxiety over the period of the active treatment sessions is examined it is observed that overall the Placebo F group exhibited the least discomfort coming into the sessions, and significantly less so than the Relaxation Control F group which experienced the most discomfort. Both the Progressive Relaxation F and the Valium 5mg. F groups also

markedly reduced their incoming anxiety in contrast to the Valium 7.5mg. F and Relaxation Control F groups, both of which continued to have high incoming anxiety.

Looking at the physiological data the picture varies slightly from measure to measure. On E.M.G., there were no consistent differences on this measure across the groups, and incoming measures tended to remain consistent over the six sessions. On pulse rate the tendency was for the Progressive Relaxation F and Placebo F groups to decrease somewhat over time while the Relaxation Control F, the Valium 7.5mg. F and the Valium 5mg. F groups showed slight increases over the six sessions in incoming pulse rate. It must be emphasized however that these differences did not attain statistical significance. When the data for systolic blood pressure is examined over the six sessions, a unique picture emerges. Initially the three 'drug' groups came into the sessions with elevated systolic blood pressure, while the Relaxation Control F and Progressive Relaxation F groups had slightly lower levels of systolic blood pressure. Over the next six sessions, the Valium 5mg. F and the Valium 7.5mg. F groups tended to elevate their incoming blood pressure, the Progressive Relaxation F and Relaxation Control F groups remained fairly constant, while the Placebo F group significantly reduced its incoming systolic blood pressure. When the data is averaged over the six sessions, the placebo group has the lowest incoming systolic blood pressure, significantly lower than the two active drug groups and about equivalent to the Relaxation Control F group. In addition, the data for diastolic

blood pressure reflected a non-significant trend similar to that of the systolic blood pressure, a finding that is consistent with the notion of diastolic blood pressure being a resistant measure.

Reduction of Anticipatory Anxiety. Looking at the data on anticipatory anxiety, it is noted that the drug treatments overall tended to be superior, but the tendency was not uniform. When self report is considered, the Valium 5mg. F and the Progressive Relaxation F and the Valium 7.5mg. F groups reported pre-post reduction that was significantly greater than the Relaxation Control F group. The Placebo F group even though it showed some pre-post reduction, did not differ significantly from the Relaxation Control F group. On systolic blood pressure both the active drug groups reduced systolic blood pressure to a significantly greater degree than the Relaxation Control F group. Both the Placebo F and the Progressive Relaxation F groups, did not differ significantly from the Relaxation Control F group in terms of reduction of systolic blood pressure. On pulse rate results were similar with one exception. Here all three 'drug' groups showed significant pre-post reduction compared to the Relaxation Control F group. The Progressive Relaxation F group did not differ significantly from the Relaxation Control F group. The diastolic blood pressure data showed no significant reductions, either across groups or over time.

Reduction of Exposure Anxiety. During the exposure period a somewhat different picture emerges. Firstly on self report all the groups reported non significant increases in anxiety from the second

reading. Though the Relaxation Control group tended to show the largest increases, they did not approach statistical significance.

On the physiological measures, a varied pattern was observed. The E.M.G. and systolic blood pressure both were generally rising during exposure, but differences were not significant, either over time or across groups. On pulse rate, in contrast to the other measures, all the groups continued to decline from the relaxation period though here again the differences were not significant. Diastolic blood pressure remained virtually the same during this period.

Reduction of Carry Over Anxiety. Finally, the data from the two final sessions which were designed to investigate the carry over effect of the anti-anxiety agents can be summed as follows.

(a) One week following the anti-anxiety phase of the treatment there were significant pre-post increases in frontalis E.M.G. for the Relaxation Control F and the Placebo F groups, while the other groups showed non-significant pre-post increases. The pulse rate data reflected significant reduction on the Valium 7.5mg. F group, the Relaxation Control F group did not change, while the other three groups showed non-significant pre-post reduction. The systolic blood pressure data were similar except that here the Valium 5mg. F group showed significant pre-post reduction, the Placebo group remained virtually unchanged, while the other three groups showed non-significant pre-post reduction. On both self-report and diastolic blood pressure, nonsignificant pre-post differences were recorded. (b) By the second week, overall significant pre-post differences were ob-

tained on frontalis E.M.G., pulse rate, and systolic blood pressure. Looking at the frontalis data, the Valium 7.5mg. F group showed a significant pre-post increase, the Relaxation Control F group showed a slight decrease, while the other three groups recorded non-significant increases. On pulse rate significant pre-post reductions were obtained on the Progressive Relaxation F and Valium 5mg. F groups, the other three groups reported non-significant pre-post reduction. On systolic blood pressure the Progressive Relaxation F and Placebo F groups showed significant pre-post reduction, the other three groups remaining basically unchanged. Here again, both self report and diastolic blood pressure did not show any significant pre-post changes.

Interpretation of Findings

In assessing these results, once again, we have to look at each aspect independently and then attempt an overall summary. But instead of looking at the question of incoming anxiety first, for reasons that will become clearer later, the discussion will start with the anticipatory period.

From the anticipatory period, the results indicated that progressive relaxation was as efficient as diazepam in doses of 5mg. and 7.5mg. in reducing self reported anxiety, and that all three are more effective than either a placebo drug or simple instructions to relax. The results also indicated that the diazepam was most effective in reducing pulse rate and systolic blood pressure. One unexpected finding was the tendency for the E.M.G. data to register increases both on the progressive relaxation group and with the

diazepam groups.

These results can be compared to those obtained by John (1977) from the two earlier studies. The first study compared progressive Relaxation, Diazepam 5mg. and 7.5mg. and Placebo drug in the reduction of anxiety caused by exposure to an experimental situation. In that study, it was found that none of the treatments yielded significant reduction on any of the autonomic indices measured. The tendency however was for Progressive Relaxation to be most effective in reducing frontalis E.M.G., while the two drug groups tended to reduce pulse rate and systolic blood pressure more effectively. The second study then compared Progressive Relaxation, Diazepam 5mg. and Placebo drug in the reduction of anxiety caused by threat of electric shock. Here it was observed that the Diazepam 5mg. group reduced pulse rate and systolic blood pressure to a significantly greater degree than the Progressive Relaxation and Placebo groups. The Progressive Relaxation group was more effective in reducing frontalis E.M.G., and it was perceived subjectively to be significantly more effective than either Diazepam 5mg. or a Placebo drug in reducing anxiety.

Looking at the three studies overall, the results seem to indicate consistently that progressive relaxation is as effective as diazepam at low levels in reducing anticipatory anxiety. An additional factor is that they are both more effective in this regard than a Placebo drug. Another finding is that diazepam invariably was the most efficient in reducing autonomic indices of anxiety. Given that

diazepam acts predominantly on the central nervous system (Greenblatt & Shader, 1974), its action within these studies, is consistent with expectations. Progressive relaxation on the other hand is expected to have its primary action on the skeletal musculature (Jacobson, 1938) and particularly within the framework of E.M.G. feedback, reduced levels of frontalis tension have been used as an index of relaxation and hence reduced anxiety. In looking at the E.M.G. data in these studies however, the relationship does not appear to be that direct. From the two earlier studies, the suggestion that frontalis E.M.G. was positively related to anxiety level and that progressive relaxation by reducing this measure could reduce anxiety, seemed to hold up. Contrary to expectations, in the present study this relationship did not in fact hold up, and furthermore frontalis tension over the eight sessions, showed consistent increases across all the groups.

One possible explanation for the E.M.G. data could be that at low levels of anxiety the frontalis muscle is less active and responds more readily to relaxation, but at higher levels of arousal the activity level of the muscle is higher and consequently it is less responsive. This explanation is commensurate with many of the findings in the literature on frontalis muscle tension and anxiety reduction. Researchers like Jacobson (1938), or Canter, Kondo and Knott (1975), who have demonstrated reduction of frontalis muscle tension levels following training in progressive relaxation usually employ a minimum of ten training sessions, and as many as one hundred.

Researchers who use fewer sessions, have generally reported no significant treatment effect for measurement of frontalis E.M.G. within populations of anxious subjects (Lader & Mathews, 1970; Mathews & Gelder, 1969).

When self report of anxiety reduction during the exposure period is considered, it is observed that all the groups responded to the phobic stimulus by an increase in anxiety. It is noteworthy however that the Relaxation Control F group reported the largest increase, even though the differences among the groups did not attain statistical significance. Thus it can be concluded that while the anti-anxiety adjuncts tend to reduce 'panic' levels of anxiety somewhat when compared to simple instructions to relax, none of these adjuncts was demonstrably better than the others. The physiological data during this period admirably demonstrated the inconsistent pattern that is characteristic of such measures in anxiety research (Morrow & Labrum, 1978; the frontalis E.M.G. increased across all the groups, pulse rate declined, while systolic and diastolic blood pressure exhibited a variable response, somewhat increasing sometimes decreasing. One further interesting observation, was that while all the groups showed a significant ($p < .05$) increase in self reported anxiety from the low post relaxation reading, to the final reading, the physiological data did not show any significant change for the same period.

To a great extent the data reported on so far has tended overall to present a fairly consistent picture even when the initial

two studies are included. In this study, when the process of anxiety reduction over the entire six sessions is noted, a somewhat different picture emerges. The self report data particularly from the screening session, indicated that initially all the groups were at equivalent levels of anxiety. By the third treatment session however, the progressive Relaxation F, the Valium 5mg. F and the Placebo F groups came into the sessions with reduced levels of anxiety, while the Valium 7.5mg. F and the flooding only groups maintained their high level of incoming anxiety, a pattern that was maintained for the next three sessions. When we look at the Placebo F group, it is observed that initially it had one of the highest incoming levels of self reported anxiety. Over the sessions this gradually decreased until by the last three sessions, this group had the lowest incoming anxiety. Curiously enough the systolic blood pressure data for the Placebo F group reflected a course parallel to that of the self report data.

There are several issues emanating from these findings. First is the question of the Placebo F₁ group and its effectiveness in reducing this type of anxiety over the sessions. Apart from the issue of mode of action, a question that will be discussed later, given the amount of reduction that occurred over the sessions it is quite possible that there occurred a ceiling effect, thereby reducing the possibility of significant reduction during the anticipatory phase.

The second issue relates to arousal theory which would predict that arousal levels and subjective reports of anxiety are

closely related (Duffy, 1957). The finding of the close relationship between the self report and blood pressure data for the Placebo F group on the incoming measures, would lend credence to such an idea. However, the fact that this relationship between any of the autonomic measures and self report did not hold for any other group, mitigates against any such conclusion.

The third issue demanding of explanation is the high incoming anxiety response of the Valium 7.5mg. F group. In attempting to explain this apparent anomaly it is necessary to invoke attribution processes. It would appear to be unquestioned that after the first two sessions, the whole experience was anticipated with much less anxiety by the subjects in the Progressive Relaxation F, Valium 5mg. F and Placebo F groups. The same was not true for the subjects in the Valium 7.5mg. F and Relaxation Control F groups. For the Relaxation Control F group a high level of anxiety was expected, but for the Valium 7.5mg. F group it was not. Looking at the three 'drug' groups, it is observed that initially they all came in with comparable levels of physiological activity. By the end of the six sessions, on the average the Placebo F group came in with the lowest physiological measures and the lowest self report of anxiety of these three groups, the Valium 5mg. F group adopted an intermediate position, while the Valium 7.5mg. F group tended overall to have the highest incoming measures. It can be hypothesised that the subjects in these groups were responding in part to the 'drug' reaction. For the Placebo F subjects, the 'mild' anti-anxiety agent was not noticeable, and so

they were not alarmed. To the subjects in the Valium 5mg. F group, the effects were noticeable enough to cause a physiological reaction, but not enough to cause alarm. For the subjects in the Valium 7.5mg. F group, the effects of the drug were noticeable enough to cause a physiological reaction and also some anticipatory anxiety.

Such an explanation is consistent with the observation of the differential response of the Valium 7.5mg. F group and the Relaxation Control F group on post-test self report of avoidance behaviour. It might be inferred that for the Relaxation Control F group, the exposure to the slides was anticipated with anxiety, in that they came in and the relaxation period did not effect a reduction in this anxiety, but often led to an increase. For the Valium 7.5mg. F group, what was anticipated was predominantly the drug response, and when the subject was actually in the situation, the anxiety to a large degree, became lessened. The experience of the whole situation was such however that there was enough residual cognitive anxiety remaining, that the incoming anxiety was maintained at a fairly high level.

This seemingly paradoxical response of the diazepam at the higher dose level has been noted previously. John (1977) in an earlier study compared diazepam 5mg., diazepam 7.5mg., placebo drug and progressive relaxation for their efficacy in reducing anxiety caused by being in an experimental situation. It was found that contrary to expectations the 7.5mg. diazepam group produced a significant increase in frontalis muscle tension. The other treatments had produced decreases in muscle tension, with the largest decrease being

recorded by the Progressive Relaxation group. Since self report was not monitored in that study, the results were interpreted as indicating that the 7.5mg. diazepam group was experiencing a higher degree of anxiety, and that this anxiety was reflected in the increased muscle tension. Further evidence for the existence of paradoxical responses to drug effects comes from the work of Barrett and Di Mascio (1965; 1966). They observed that while oxazepam, valium and librium reduced anxiety in groups of high anxious subjects, in groups of low anxious subjects they caused a statistically significant increase in self reported anxiety. In conclusion, it seems likely that even for the phobic subjects, 7.5mg. of diazepam was a high dose, and consequently produced a certain degree of 'anxiety' perhaps because the drug reaction experienced was out of line with expectations.

Finally, the anti-anxiety agents were compared for their carry over effect. From the two earlier studies, it had been noted that the pattern of reduction of physiological measures observed during the treatment, was maintained one week later. That is the Progressive Relaxation group still tended to reduce frontalis tension more than the other groups, while the drug groups tended to be more effective on pulse rate and systolic blood pressure, though the differences did not attain statistical significance. In the third study on the other hand there was one and two weeks later, significant pre-post reduction of pulse rate and systolic blood pressure overall, the tendency being for the drug groups to be particularly efficient in

reducing these measures. However, the often similar effectiveness of the Progressive Relaxation F group and the Placebo F group, on these measures, and the absence of demonstrated superiority of any treatment in reducing any of these measures, makes a definitive statement difficult. Furthermore there was a tendency for self report of anxiety to increase though this was small and non significant.

This thesis addressed itself in part to the question of the comparative efficacy of the above treatments for anxiety. The answer is clear, under differing conditions these treatments are all effective. Unfortunately what is still not clear is under what conditions do these treatments work best. For instance, in the situation of anticipatory anxiety, progressive relaxation and diazepam (5 and 7.5mg.) both seemed comparably effective; furthermore they were more effective than either a placebo drug or simple instructions to relax. When anxiety over a longer period was looked at, placebo drug was as effective as either progressive relaxation or the lower dosage of diazepam (5mg.) in reducing this anxiety, and all three did this more effectively than either 7.5mg. of diazepam or simple instructions to relax.

Many researchers (e.g., Meyer & Reich, 1978) suggest that in reducing anxiety the predominant mode of response, overt motor, autonomic somatic, or verbal cognitive should be considered when using a particular clinical intervention for a patient. The idea would be to use an intervention strategy that would act maximally on the target response. Given the efficacy of treatments as diverse as

progressive relaxation, diazepam, and placebo drug, it would be expected that their dominant mode of action should be readily identifiable. In this study unfortunately there were no data observed, which can substantiate such a claim, at least in the sense of giving definite direction for clinical use. They do however raise a number of clinical questions, and suggest ideas for future research.

General Issues

The results obtained in this thesis are quite in accordance with many of the findings in anxiety research. For example as has been observed with studies that have employed multiple measures of anxiety (e.g., Hohmann, 1966; Lader, 1967; Lee & Tyrer, 1980; Mathews, 1971; Morrow & Labrum, 1978; Schachter & Singer, 1962; Tyrer & Lader, 1976; VanEgeren, 1970), the results demonstrated the lack of a consistent relationship between self report and physiological indices of anxiety. This constant finding raises the question as to which measure is the most accurate representation of the anxiety state. It is unquestioned that anxiety as an emotional state can affect physiological responding (Fink, 1979), and arousal theory would predict that anxiety levels can be inferred from the level of physiological functioning. However as Lader (1967) noted, trying to ascertain what the physiological measures are in fact reflecting is not a particularly easy task, and as remarked upon earlier, they do not correlate very highly with other measures of anxiety. Given this, self report remains as the most salient measure of anxiety, a situation that some researchers and clinicians find unsatisfactory (Ayllon &

Azrin, 1968; Beigelman & Hersen, 1971; Gordon, 1975; Simkins, 1970).

Somewhat paradoxically, an explicit assumption in the reduction of anxiety by both progressive relaxation and diazepam, is that anxiety is highly correlated with autonomic processes, and that arousal reduction is necessary to effect reduction in the state (Borkovec, 1976). Some researchers even suggest that an efficient strategy in the treatment of phobias and anxiety may be twofold; first to assess the response system(s) that initiates the fear or anxiety response, and second to employ treatment techniques that produce improvement in the problematic system. This latter assumption, that treatment techniques act predominantly on some aspect of arousal, has not to date been borne out with any degree of consistency (Lader, 1967). This was also the finding in this thesis. Thus as indicated before, from the two earlier studies the pattern of the results seemed to suggest that progressive relaxation had its most significant effect on muscle tension, while diazepam had its major effect on pulse rate and systolic blood pressure. In the third study however when a higher level of anxiety arousal was employed this dichotomous relationship did not hold up. So in the case of the frontalis E.M.G., progressive relaxation did not differentially affect reduction, and as a matter of fact all the groups reported increases in their frontalis tension. On the other hand, while the diazepam groups did not reduce pulse rate and systolic blood pressure significantly more than the other treatment groups, the fact that subjects in the diazepam groups came into the sessions with

elevated pulse rate and blood pressure, is a confounding factor.

Essentially the question can be raised, was the elevation a response to the perception of drug action, and if so without this elevation, would significant pre-post differences have been obtained.

A further interesting finding was the effectiveness of the Placebo F group in reducing both arousal levels and self reported anxiety. Given that placebos demonstrate consistent effectiveness, particularly as anti anxiety agents (McNair et al., 1968; Reynolds et al., 1965), a major role for cognitions seems to be indicated in hypothesizing a mode of action. Undoubtedly as a multifaceted and multidetermined response (Morrow & Labrum, 1978), several factors contribute to the elicitation and maintenance of anxiety, and cognitions happen to be one such factor. Such a proposition is in conformity with a model of anxiety put forward by Lader (1974). He sees both external and internal stimuli as being evaluated cognitively. If a danger is perceived, emotional feelings arise as a derivative of the individual's cognitive appraisal of the threat. Central nervous system arousal then produces both an emotional state perceived in consciousness and labelled anxiety, and in addition, peripheral physiological changes such as tachycardia and sweating which in turn may then be perceived secondarily and reinforce the anxiety. Therefore emotional feelings which are cognitively induced, both modify and are modified by peripheral physiological changes.

A similar model of anxiety has been proposed by Borkovec (1976). In Borkovec's model, anxiety is initiated by one or more of

the following, external fear cues, internal fear cues, autonomic arousal, verbal and non verbal images, or proprioception from overt behaviour. These lead to the immediate anxiety reaction which again can be one or all of physiological arousal, cognition or overt behaviour. These latter mechanisms then serve subsequently either to maintain or reduce the anxiety.

It is apparent that in both models cognitions are seen as playing an important role in initiating, maintaining and subsequently reducing experienced anxiety. That being the case, the effectiveness of diverse treatment methods in general and placebos in particular becomes more readily explained. It can be hypothesized that cognitive factors are intrinsic to all treatment situations, and these cognitions could either operate by themselves, or in conjunction with other treatment modalities to affect anxiety. However the bidirectional nature of such factors, would make their action difficult to isolate and probably accounts for the so called non specific factors in anxiety reduction (Rickels, 1976; Uhlenhuth et al., 1969).

Finally, the study has some tentative implications for the treatment of anxiety. Firstly, it can be suggested that particularly for subjects who experience marked anticipatory responses to an anxiety evoking situation, pre-treatment with a mild anxiety reducer would be the method of choice as a preliminary measure. Given the comparable effectiveness of progressive relaxation on anxiety reduction at this level however, it would be preferred as a long term management strategy for several reasons. When taught as a learned skill that can be ac-

tively utilized in anxiety producing situations, it has been found to be quite effective (Goldfried & Trier, 1974; Zeisset, 1968). Unlike medication, there are no unwanted side effects, no habituation risks, no possibility of abuse, and no possible long term effects. Furthermore, the high non compliance rate with medication is not a factor (Porter, 1969; Schweitzer & Adam, 1979). The chief disadvantage of progressive relaxation is that many individuals simply do not respond (Wolpe, 1973).

The effectiveness of the placebo drug, while interesting and consistent, presents a particular challenge. It can be inferred that suggestion factors play a powerful role in anxiety reduction but nevertheless, continuing and serious study of the factors involved in the placebo effect is needed before we can understand how to utilize this factor.

So far, the question of treatment of anxiety has been viewed in terms of reduction through the aid of an external agent. The efficacy of flooding alone though raises the notion of the experience of anxiety as an adaptive mechanism. This would suggest that under some conditions, the open confrontation with the anxiety situation can itself be therapeutic, and apparently lead to a reduction in the experienced anxiety. There is much that is not known about the process of anxiety reduction; hopefully future research addressing itself to questions such as individual response, and combination of treatments, will elucidate many of the factors that are presently unclear.

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Appendix A

Phobic Anxiety

Low Fear

High Fear

1 2 3 4 5

Present Anxiety

Low Anxiety

High Anxiety

1 2 3 4 5 6 7 8 9

General Anxiety

Low Anxiety

High Anxiety

1 2 3 4 5 6 7 8 9

3. Remember, use the middle box only if you cannot possibly decide on a or c.
- 4.. Don't spend time thinking over the statement. Just mark your answer quickly, according to how you feel about it now.

It will take only ten minutes or so to finish. Hand in the booklet when you're through, unless told to do otherwise. As soon as you're told to, turn the page and begin.

A

1. My interests, in people and ways to have fun, seem to change quite fast.
(a) true, (b) in between, (c) false..... a b c
2. Even if people think poorly of me I still go on feeling O.K. about myself.
(a) true, (b) in between, (c) false..... a b c
3. I like to be sure that what I am saying is right before I join in on an argument.
(a) true, (b) in between, (c) no..... a b c
4. I am inclined to let my feelings of jealousy influence my actions.
(a) sometimes, (b) seldom, (c) never..... a b c
5. If I had my life to live over again I'd:
(a) plan very differently, (b) in between, (c) want it the same..... a b c
6. I admire my parents in all important matters.
(a) yes, (b) in between, (c) no..... a b c
7. It's hard for me to take "no" for an answer, even when I know what I'm asking is impossible.
(a) true, (b) in between, (c) false..... a b c
8. I wonder about the honesty of people who are more friendly than I'd expect them to be.
(a) true, (b) in between, (c) false..... a b c
9. In getting the children to obey them, my parents (or guardian) were:
(a) usually very reasonable, (b) in between, (c) often unreasonable..... a b c
10. I need my friends more than they seem to need me.
(a) rarely, (b) sometimes, (c) often..... a b c

A

11. I feel sure I could "pull myself together" to deal with an emergency if I had to.
(a) true, (b) in between, (c) false.....
12. As a child I was afraid of the dark.
(a) often, (b) sometimes, (c) never.....
13. People sometimes tell me that when I get excited, it shows in my voice and manner too obviously.
(a) yes, (b) uncertain, (c) no.....
14. If people take advantage of my friendliness I:
(a) soon forget and forgive, (b) in between, (c) resent it and hold it against them.....
15. I get upset when people criticize me even if they really mean to help me.
(a) often, (b) sometimes, (c) never.....
16. Often I get angry with people too quickly.
(a) true, (b) in between, (c) false.....
17. I feel restless as if I want something but don't know what.
(a) hardly ever, (b) sometimes, (c) often.....
18. I sometimes doubt whether people I'm talking to are really interested in what I'm saying.
(a) true, (b) uncertain, (c) false.....
19. I'm hardly ever bothered by such things as tense muscles, upset stomach, or pains in my chest.
(a) true, (b) in between, (c) false.....
20. In discussions with some people, I get so annoyed I can hardly trust myself to speak.
(a) sometimes, (b) rarely, (c) never.....

A Score

CONTINUE ON NEXT PAGE

B

21. I use up more energy than most people in getting things done because I get tense and nervous.
(a) true, (b) uncertain, (c) false.....
22. I make a point of not being absent-minded or forgetful of details.
(a) true, (b) uncertain, (c) false.....
23. No matter how difficult and unpleasant the snags and stumbling blocks are, I always stick to my original plan or intentions.
(a) yes, (b) in between, (c) no.....
24. I get over-excited and "rattled" in upsetting situations.
(a) yes, (b) in between, (c) no.....
25. I sometimes have vivid, true-to-life dreams that disturb my sleep.
(a) yes, (b) in between, (c) no.....
26. I always have enough energy to deal with problems when I'm faced with them.
(a) yes, (b) in between, (c) no.....
27. I have a habit of counting things, such as steps, or bricks in a wall, for no particular purpose.
(a) true, (b) uncertain, (c) false.....
28. Most people are a little odd mentally, but they don't like to admit it.
(a) true, (b) uncertain, (c) false.....
29. If I make an embarrassing social mistake I can soon forget it.
(a) yes, (b) in between, (c) no.....
30. I feel grouchy and just don't want to see people.
(a) almost never, (b) sometimes, (c) very often.....

a b c

a b c

a b c

a b c

a b c

a b c

a b c

a b c

a b c

a b c

B

31. I can almost feel tears come to my eyes when things go wrong.
(a) never, (b) very rarely, (c) sometimes..... a b c
32. Even in the middle of social groups I sometimes feel lonely and worthless.
(a) true, (b) in between, (c) false..... a b c
33. I wake in the night and have trouble sleeping again because I'm worrying about things.
(a) often, (b) sometimes, (c) almost never..... a b c
34. My spirits usually stay high no matter how many troubles I seem to have.
(a) true, (b) in between, (c) false..... a b c
35. I sometimes get feelings of guilt or regret over unimportant, small matters.
(a) yes, (b) in between, (c) no..... a b c
36. My nerves get on edge so that certain sounds, such as a screechy hinge, are unbearable and give me the shivers.
(a) often, (b) sometimes, (c) never..... a b c
37. Even if something upsets me a lot, I usually calm down again quite quickly.
(a) true, (b) uncertain, (c) false..... a b c
38. I seem to tremble or perspire when I think of a difficult task ahead.
(a) yes, (b) in between, (c) no..... a b c
39. I usually fall asleep quickly, in just a few minutes, when I go to bed.
(a) yes, (b) in between, (c) no..... a b c
40. I sometimes get tense and confused as I think over things I'm concerned about.
(a) true, (b) uncertain, (c) false..... a b c

STOP HERE. BE SURE YOU HAVE ANSWERED EVERY QUESTION

B Score

Appendix C

Fear Inventory

The items in this questionnaire refer to things and experiences that may cause fear or other unpleasant feelings. Write the number of each item in the column that describes how much you are disturbed by it nowadays.

	Not at All	A Little	A Fair Amount	Much	Very Much
1. Noise of vacuum cleaners					
2. Open wounds					
3. Being alone					
4. Being in a strange place					
5. Loud voices					
6. Dead people					
7. Speaking in public					
8. Crossing streets					
9. People who seem insane					
10. Falling					
11. Automobiles					
12. Being teased					
13. Dentists					
14. Thunder					
15. Sirens					
16. Failure					

	Not At All	A Little	A Fair Amount	Much	Very Much
17. Entering a room where other people are already seated					
18. High places on land					
19. Looking down from high buildings					
20. Worms					
21. Imaginary creatures					
22. Strangers					
23. Receiving injection					
24. Bats					
25. Journeys by train					
26. Journeys by bus					
27. Journeys by car					
28. Feeling angry					
29. People in authority					
30. Flying insects					
31. Seeing other people injected					
32. Sudden noises					
33. Dull weather					
34. Crowds					
35. Large open spaces					
36. Cats					

	Not At	A Little	A Fair Amount	Much	Very Much
37. One person bullying another					
38. Tough looking people					
39. Birds					
40. Sight of deep water					
41. Being watched working					
42. Dead animals					
43. Weapons					
44. Dirt					
45. Crawling insects					
46. Sight of fighting					
47. Ugly people					
48. Fire					
49. Sick people					
50. Dogs					
51. Being criticized					
52. Strange shapes					
53. Being in an elevator					
54. Witnessing surgical operations					
55. Angry people					
56. Mice					
57. Blood					
a - Human					
b - Animal					

	Not At All	A Little	A Fair Amount	Much	Very Much
58. Parting from friends					
59. Enclosed places					
60. Prospect of a surgical operation					
61. Feeling rejected by others					
62. Airplanes					
63. Medical odors					
64. Feeling disapproved of					
65. Harmless snakes					
66. Cemeteries					
67. Being ignored					
68. Darkness					
69. Premature heart beats (Missing a beat)					
70. Nude Men (a)					
Nude Women (b)					
71. Lightning					
72. Doctors					
73. People with deformities					
74. Making mistakes					
75. Looking foolish					
76. Losing control					

	Not At All	A Little	A Fair Amount	Much	Very Much
77. Fainting					
78. Becoming nauseous					
79. Spiders					
80. Being in charge or responsible for decisions					
81. Sight of knives or sharp objects					
82. Becoming mentally ill					
83. Being with a member of the opposite sex					
84. Taking written tests					
85. Being touched by others					
86. Feeling different from others					
87. A lull in conversation					

Appendix D

What Are You Afraid Of?

- | | | |
|---------------------------------------|---|--|
| <input type="checkbox"/> Cats | <input type="checkbox"/> Airplanes | <input type="checkbox"/> Dead people |
| <input type="checkbox"/> Dogs | <input type="checkbox"/> Driving a car | <input type="checkbox"/> Cemeteries |
| <input type="checkbox"/> Rats | <input type="checkbox"/> Riding in a car | <input type="checkbox"/> Suffocating |
| <input type="checkbox"/> Mice | <input type="checkbox"/> Trains | <input type="checkbox"/> Failing a test |
| <input type="checkbox"/> Hamsters | <input type="checkbox"/> Subways | <input type="checkbox"/> Roller coasters |
| <input type="checkbox"/> Gerbils | <input type="checkbox"/> Buses | <input type="checkbox"/> Crowded places |
| <input type="checkbox"/> Skunks | <input type="checkbox"/> Boats | <input type="checkbox"/> Blood |
| <input type="checkbox"/> Horses | <input type="checkbox"/> Heavy traffic | <input type="checkbox"/> Heights |
| <input type="checkbox"/> Cows | <input type="checkbox"/> Highways | <input type="checkbox"/> Illness |
| <input type="checkbox"/> Sheep | <input type="checkbox"/> Bridges | <input type="checkbox"/> Stores |
| <input type="checkbox"/> Snakes | <input type="checkbox"/> Tunnels | <input type="checkbox"/> Babies |
| <input type="checkbox"/> Lizards | <input type="checkbox"/> Sharp objects | <input type="checkbox"/> Enclosed spaces |
| <input type="checkbox"/> Bats | <input type="checkbox"/> Injections | <input type="checkbox"/> Choking |
| <input type="checkbox"/> Dead animals | <input type="checkbox"/> Doctors | <input type="checkbox"/> Writing |
| <input type="checkbox"/> Birds | <input type="checkbox"/> Dentists | <input type="checkbox"/> Stairs |
| <input type="checkbox"/> Flies | <input type="checkbox"/> Hospitals | <input type="checkbox"/> Doorknobs |
| <input type="checkbox"/> Bees | <input type="checkbox"/> Nurses | <input type="checkbox"/> Thunderstorms |
| <input type="checkbox"/> Wasps | <input type="checkbox"/> Pills | <input type="checkbox"/> Dark places |
| <input type="checkbox"/> Ants | <input type="checkbox"/> Medical TV Shows | <input type="checkbox"/> Strange places |
| <input type="checkbox"/> Spiders | <input type="checkbox"/> Dirt | <input type="checkbox"/> Strange noises |
| <input type="checkbox"/> Worms | <input type="checkbox"/> Infection | <input type="checkbox"/> Loud voices |

- | | | |
|---|--|--|
| <input type="checkbox"/> Snails | <input type="checkbox"/> Disease | <input checked="" type="checkbox"/> Crossing streets |
| <input type="checkbox"/> Shellfish | <input type="checkbox"/> Contamination | <input type="checkbox"/> Fire |
| <input type="checkbox"/> Fish | <input type="checkbox"/> Large open spaces | <input type="checkbox"/> Moving to a new house |
| <input type="checkbox"/> Being alone | <input type="checkbox"/> Speaking in public | <input type="checkbox"/> Falling |
| <input type="checkbox"/> People who look insane | <input type="checkbox"/> Sirens | <input type="checkbox"/> Strangers |
| <input type="checkbox"/> Medical odors | <input type="checkbox"/> Medical charts | <input type="checkbox"/> Escalators |
| <input type="checkbox"/> Water | <input type="checkbox"/> Sick people | <input type="checkbox"/> Balconies |
| <input type="checkbox"/> Losing control | <input type="checkbox"/> "Doing "something stupid" in public | <input type="checkbox"/> Travelling |
| <input type="checkbox"/> Involuntary excretion | <input type="checkbox"/> Weapons | <input type="checkbox"/> Lightning |
| <input type="checkbox"/> Missed heartbeat | <input type="checkbox"/> Travelling home | <input type="checkbox"/> Vegetables |

Other* (please specify) _____

Appendix E

Checklist Item: Behavioural Avoidance Test

1. Walks up to cage.
2. Touches cloth of cage.
3. Removes cloth.
4. Touches cage lid.
5. Removes lid.
6. Reaches into cage with bare hand a few inches.
7. Reaches into cage within an inch of animal.
8. Touches animal.
9. Touches animal for several seconds.
10. Grasp's animal with hand.
11. Picks animal up off the floor of the cage.
12. Holds animal off the floor of the cage for three seconds.
13. Holds animal off the floor of the cage for seven seconds.
14. Lifts animal out of cage.
15. Holds animal out of cage for seven seconds.
16. Brings animal within 18 inches of body.
17. Brings animal within 6 inches of body.
18. Brings animal within 1 inch of body.

Appendix F

Demographic Information

NAME:

PHONE NO.:

DATE OF BIRTH:

PRESENT OCCUPATION:

SEX:

MAIN PHOBIA (FEAR):

LENGTH OF DURATION OF PHOBIA:

ARE YOU CURRENTLY

(a) ON MEDICATION YES ___ NO ___

(b) IN THERAPY YES ___ NO ___

HAVE YOU PREVIOUSLY SOUGHT HELP FOR THIS PHOBIA? YES ___ NO ___

DO YOU WISH TO OVERCOME THIS FEAR? YES ___ NO ___

HAVE YOU WITHIN THE PAST 12 MONTHS, TAKEN OR PRACTICED

(a) MINOR TRANQUILIZERS (e.g.) DIAZEPAM YES ___ NO ___

(b) OTHER MAJOR MEDICATION YES ___ NO ___

(c) RELAXATION TRAINING YES ___ NO ___

DO YOU DRINK ALCOHOL IN WHAT CAN BE CONSIDERED LARGE AMOUNTS? YES ___

NO ___

Appendix G

Subjects Instructions

The object in front of you is a harmless (snake, spider, rat) enclosed in a cage which is covered over with a cloth. We would like you to try to do several things with this animal. It is very important that you do only those things that you feel comfortable doing. If on any task you feel any real fear or anxiety, do not go any further with it. In such a case leave the room and the test will be terminated. If you complete one task go right on to the next. Do as many tasks as you can. Remember, do the tasks in order and do only those tasks you feel comfortable doing. Okay, if you can comfortably do so.

Appendix H

Subjects Instructions²

We are comparing two well known treatments which have been used in the reduction of general anxiety, to test their comparative efficacy as adjuncts in the treatment of phobic states when a flooding or exposure technique is utilised. There will be eight treatment sessions in all. The first six will be held twice weekly for a period of three weeks, with the seventh in the fourth week, and the eighth in the fifth week.

The first six sessions will each be approximately one hour long, and will be conducted in the following manner. Two minutes after entering the experimental room, frontalis muscle action potential, pulse rate, systolic blood pressure, diastolic blood pressure, and self-rating of anxiety will be taken. This will be followed by a period of relaxation lasting approximately twenty three minutes. Depending on the group to which you are assigned, this relaxation will be either by a relaxation tape, a minor tranquilizing drug, or you will be just asked to sit and relax. During this period, you will be left alone in the room.

After this period of relaxation, the experimenter will return and repeat the measures. This will be followed by two nine minute periods of exposure to slides of the phobic stimulus. Measures are recorded, both in the middle, and at the end of the presentation. The seventh session lasts for approximately 20 minutes, and differs from the preceding six in that there is no relaxation period, and

only one period of exposure. The eighth session is a treatment and re-test session. The treatment phase is similar to that of the seventh session, this is then followed by a testing phase, similar to the original screening. If you are not satisfied with your progress at the end of the eighth session, you will be worked with until you are O.K?

Appendix

I the undersigned understand that I am participating in a study on the treatment of phobias of small animals, and that I could be in one of the following groups:

- (a) Progressive Relaxation Group
- (b) Placebo Drug Group
- (c) Drug Group (minor tranquilizer)

I have also read the procedure and agree to participate knowing that I may end my participation at any time I wish without any obligation to the experimenter.

5

Appendix J

Modified Jacobsonian Instructions
For Progressive Relaxation

Instructions for Progressive Relaxation

I want you to lie as comfortably as you can on the bed with your arms straight at your side and your legs straight. First, I want you to take a slow, deep breath... Take a slow deep breath and hold it... Then relax and let go.

In the first exercise, I want you to focus on the muscles across the forehead. I want you to tighten these muscles by raising your eyebrows as high as you can. Raise your eyebrows as high as you can... as if you were trying to force them right into your hairline. Now concentrate on the tension that builds up across your forehead. Focus on the discomfort you're feeling... Allow this discomfort to build up... and then relax, and let go. Smooth the muscles across your forehead. Try and let these muscles go more and more completely limp.

***** (10-second pause)

Now I want you to lower your eyebrows and force them together as if you are frowning. Lower your eyebrows and force them together and, at the same time, I want you to clench your eyes tightly shut. Focus on the buildup of tension around your eyes, across the bridge of your nose, and all along your eyebrows. Again, concentrate on the feeling of tension and feeling of discomfort. Hold this tension... and now, let go and relax. Feel the relaxation spreading around your eyes... around the corners of your eyes and across your eyelids. Feel the relaxation along your eyebrows and across the

bridge of your nose.

***** (10-second pause)

In the next exercise, I want you to do two different things. First, I want you to press your tongue against the roof of your mouth. Press your tongue against the roof of your mouth and, at the same time, clench your teeth. It's important to keep the counterpressure going between your tongue on the roof of your mouth and your clenched teeth. Concentrate now on the pressure building up along your jaw and along your tongue. Concentrate on this feeling. Focus on the tension. Focus on the discomfort... Now, let go and relax. Let your tongue go into your lower jaw without touching the roof of your mouth at all. And let your jaw go slack.

***** (10-second pause)

Breathe easily and deeply... and regularly. Each time that you exhale, I want you to concentrate on relaxing more and more completely.

***** (10-second pause)

Now focus on the muscles around your mouth. To tense these muscles, I want you to press your lips tightly together. Press your lips together as if you were trying to press your upper lip down into your lower lip. Tense these muscles and feel the tension all across your upper lip and around the corners of your mouth, and along your lower lip. Press... and now relax. As you relax, let your lips part and let your jaw go slack. Concentrate on the pleasurable sensation

of the muscles becoming more and more relaxed.

***** (10-second pause)

We're now going to shift our focus to the muscles around your neck. The first thing I want you to do is to turn your cheek so it's pressing against the pillow. Press your left cheek against the pillow and, with your shoulders flat, I want you to twist your head as if you were twisting it around on a pivot. Again, keep your shoulders flat and press your left cheek into the pillow as if you were twisting your head around on a pivot. Concentrate now on the buildup of tension along the right side of your neck and the right shoulder. Concentrate on this tension. Focus on the discomfort. And now, let go and relax.

Breathe easily and deeply. And now just let your head go back to its original position.

***** (10-second pause)

Now do the same thing, but on the other side. Turn your head so that your right cheek is resting on the pillow and again, keeping your shoulders flat, press your cheek into the pillow as if you're twisting your head on a pivot. Press your right cheek into the pillow and concentrate on the buildup of tension along the left side of your neck and left shoulder. Press and concentrate on the tension. And now relax... let go... enjoy the feeling of relaxation that spreads around your head and shoulder. Then let your head move back to its original position.

***** (10-second pause)

In the last exercise of the set around your neck, I want you to keep your shoulders flat on the bed but raise your head so that your chin is pressing into your breastbone. Lift your head off the bed and press your chin into your breastbone. Again, hold the tension... hold this tension, and concentrate on the discomfort building up around your neck.

When I tell you to let go, I want you to let your head just drop back onto the pillow. All right, relax... your head's dropped down onto the pillow and I want you now to just concentrate on the feeling of relaxation spreading around your neck.

Enjoy this feeling of relaxation spreading around your neck.

Enjoy this feeling of relaxation and continue to breathe evenly and regularly, concentrating on relaxing as you exhale.

***** (10-second pause)

Now shift the focus to the muscles along your arms and into your hands.

Keep your arms straight and make your hands into fists. Now slightly raise your arms off the bed, keeping your arms straight. Slightly raise your arms off the bed with your arms straight and your hands clenched into fists, and tighten the muscles all along your arms, your forearms, your upper arms, and right across your shoulders. Feel the tension building up in your arms. Concentrate on this feeling... and feel the vibration in the muscles as you keep these muscles tight. When I tell you to relax, I want you to just let your arms flop down onto the bed.

All right, relax.... Your arms have dropped down to the bed, and now I want you to focus on the wave of relaxation and the wave warmth that travels along your arms and across your shoulders. Just focus on this release... on this pleasant feeling of relaxation... and let your arms become more and more limp and more and more relaxed.
***** (10-second pause)

Now concentrate on the muscle of your stomach and your diaphragm. In the first exercise, I want you to press your stomach slightly out, and tighten it, as if you're preparing to receive a blow. Tighten this muscle as if you were preparing to receive a blow... Hold this tension.... Concentrate on the feeling that you're getting from these tensed muscles.... Hold it.... And now relax.

Relax, and let these muscles go limp. Feel the relaxation spreading across your stomach. Feel the pleasurable sensation of these muscles when they're relaxed. Just try to relax them more and more completely.

***** (10-second pause)

Now I want you to pull in your stomach. Suck in your stomach as if you're trying to touch the back of your spine. At the same time, I want you to tighten the muscles in your diaphragm.

Again, concentrate on these muscles.... Concentrate on the tension.... Concentrate on the discomfort. Hold this tension... and now relax. Just breathe easily and deeply, allowing these muscles to relax more and more completely... again focussing on the pleasurable

sensation of these muscles as they relax more and more completely.

***** (10-second pause)

Now shift your focus on the muscles between your waist and your knees. Again, I want you to do two exercises at once. First, I want you to press your knees together. Press your knees together and, at the same time, I want you to tighten the muscles along your thighs... the upper parts of your thighs, the lower parts of your thighs, and the muscles in your buttocks.

Tighten all these muscles between your waist and your knees and continue to keep the pressure going between your knees. Hold this tension... Feel the discomfort... And then relax. Let go and concentrate on the sensation of relaxation traveling along your legs.

Try and let these muscles relax more and more completely. Become more and more limp.

***** (10-second pause)

Now focus on the lower parts of your legs. In order to tense these muscles, I want you to point your toes back toward your knees. Point your toes back toward your knees and tense the muscles in your calves and in your shins.

You should feel the tension flowing from the tips of your toes, around the backs of your heels, and right up into your calves and shins. Concentrate on holding this tension, focussing on it.... And now, let go. Relax, and again concentrate on the feeling of warmth and relaxation that spreads along your legs.

***** (10-second pause)

Now, take a slow, deep breath.... Breathe easily and deeply and concentrate on letting your whole body relax more and more completely each time that you exhale.

***** (10-second pause)

I want you to imagine a wave of relaxation starting at the top of your head and traveling down your whole body to the tips of your toes. Concentrate first on the muscles across your forehead. Relax the muscles across your forehead and allow this relaxation to spread along your eyebrows and across the bridge of your nose. Relax the muscles across your eyebrows and across the bridge of your nose. Let this relaxation spread around your eyes, the corners of your eyes, and across your eyelids.... Let it continue to spread down along your nose to the corner of your nostrils... and spread out across your cheeks. Relax the muscles in your cheeks. Relax the muscles across your upper lip. Focus on relaxing just the muscles of the upper lip and around the corners of your mouth... then down around your lower lip.

Let your mouth go slightly open and let all these muscles go completely limp and heavy. Relax the muscles in your tongue... along your tongue and down underneath your tongue. Again, just let your tongue rest on your lower jaw without touching the roof of your mouth.

Continue to feel the relaxation spreading along your jaw. Let your jaw go slack, and let the muscles relax.

***** (10-second pause)

Relax the muscles along your neck and down into your shoulders.

Feel the muscles relaxing in your neck and down into your shoulders.

Allow this relaxation to spread slowly down your arms right to the tips of your fingers. Feel the relaxation spreading down your arms and right to the tips of your fingers.

***** (10-second pause)

Concentrate now on the contact between your back and the bed. Feel the contact between your back and the bed and try to maximize the contact. Just let your back sink down into the bed and feel the wave of relaxation starting across your shoulders and traveling down your back right to the tip of your spine. Concentrate on feeling the wave of relaxation going from across your shoulders down to the tip of your spine.

***** (10-second pause)

Relax the muscles in your legs. First the muscles in your upper legs... all around your thighs... down around your knees... into your calves and shins... right down into your feet. If any tension remain in your legs, I want you to imagine the tension draining down your legs and out through the tips of your toes. Just feel the tension draining down your legs and out through the tips of your toes.

***** (10-second pause)

Now concentrate on your whole body. Feel yourself being supported by the bed. You don't have to make any effort at all... you're being completely supported and you can just allow yourself to

sink down into the bed. Allow your muscles to become more and more completely limp and heavy.

Feel yourself sinking down into the bed.

***** (10-second pause)

In a moment, I'm going to tell you to open your eyes and to sit up slowly. I want you to maintain this relaxation, this feeling of being refreshed, even after you sit up. All right.... Open your eyes.... Slowly sit up... and continue to feel relaxed and refreshed.

(End of Instructions for Progressive Relaxation)