# Stimulus Conditioned Autonomic Response Suppression: a Behavioural Therapy (l968)

Douglas A. Quirk

Clarke Institute of Psychiatry, University of Toronto

In his several discussions of the application of his desensitization procedure, Wolpe (1958) has claimed therapeutic benefit in many neurotic patients, but has disclaimed the effectiveness of this procedure with 'psychotic' patients. Indeed, in spite of some remarkable instances of improvement among our psychiatric patients subjected to Wolpe's procedure for reciprocal inhibition therapy (RIT), in our preliminary attempts to treat psychotic patients with this procedure, we were occasionally unable to alter these patients symptoms at all. However, these preliminary failures did suggest ways to account for the ineffectiveness of RIT in these instances, and, in turn, to suggest possible technical solutions to the treatment problems posed by such patients. Moreover, concurrent with the development of procedures, a model emerged which seemed to have some utility in accounting both for the presence of many patient symptoms, and for some of the effects which were being produced in the therapeutic program. Subsequently, from a rather direct application of the model, a treatment procedure emerged which appeared to have considerable therapeutic efficacy regardless of whether the patients treated were neurotic or psychotic.

In the present paper an attempt will be made to outline in broad terms the conceptual models which we have developed both for the neuroses and for the psychoses. Next we will outline the treatment procedure which emerged from those models. And, finally, some research problems we are investigating, and some data we have collected will be presented. Although the present paper is intended as a technical and theoretical paper rather than as a research report, some preliminary data is presented concerning the effectiveness of the procedure in question, arising from some clinical trials with the technique.

## Model for the Neuroses

The model for the 'neuroses' which was adopted differs in no important way from models frequently adopted by psychologists attempting to translate psychiatric models into behavioural terms. Neurotic behaviour is merely transient behaviour which is of little consequence when it emerges temporarily and comes readily under conscious control or under the control of normal environmental contingencies. However, when conscious effort on the part of the subject and normal environmental contingencies fail over a significantly long period of time to control or extinguish disruptive forms of behaviour, so that they become troublesome to the subject or to those around him, at that point in time it may become meaningful to speak of a neurotic disorder. That is to say, it is the <u>persistence</u> of the 'neurotic' behaviour which makes neurotic behaviour problematic. We have called the persistent, and apparently self-perpetuating, character of the neuroses the 'neurotic chain'.

Ignoring, for present purposes, the causal sequences which may have led up to the establishment of particular forms of neurotic behaviours in particular subjects, it may be possible instead to

restrict our examination of neurotic disorders to the moment-to-moment behaviours of the neurotic chain, in order to determine what makes the neurotic chain persistent or self-perpetuating. There are many accounts in the psychiatric literature which seem to give essentially similar descriptions of the moment-to~moment behaviours of neurotic subjects associated with occurrences of their symptoms. Freud (1926) has made a general statement of the sequence of events involved in the neurotic chain, which, stripped of some of its aetiological and inferential elements, differs little from frequently appearing accounts of such behaviour. Freud's account may be paraphrased roughly as follows: a formerly punished, and now threatening, stimulus appears in the subject's field; it is perceived as threatening or dangerous; anxiety is aroused in the subject; the anxiety aroused serves as the signal or motive for the activation of a defensive act; and, if the defensive act is successful in reducing awareness of the threatening stimulus, the organism returns to a state of homeostasis. In this view, the defensive act is the symptom of the neurosis and the evidence for the existence of a neurosis, and the repetition or persistence of the symptomatic defence defines the severity of the neurosis.

To fit the above sequence of moment-to-moment events into a behavioural model which might account for its persistence, it is necessary only to add a few elements which are unlikely to be accessible to introspection, and hence would not be expected to appear in a description derived from introspective methods. It is noteworthy that the above description terminates the occurrence of each symptom elicitation with a return to homeostasis (i.e., with a drive reduction). That is, the defined sequence provides for a reinforced practice trial each time the symptomatic defensive behaviour is elicited. Consequently, in order to account for the persistence of the symptomatic defensive behaviour in the chain, it is necessary only in addition to account for the maintenance of the anxiety response unextinguished in the system (to provide the drive to be reduced as the reinforcement) in spite of changes in the subject's circumstances and in his environmental contingencies. In other words, it would seem necessary at least to include an emotional or classical conditioning element in the model in order to maintain an anxiety response unextinguished in the chain to be relieved following 'successful'? defence or avoidance.

On the basis of the foregoing, a model for the moment-to~moment behaviours of the neurotic chain might be hypothesized as follows (see Illustration I):

1. A situational stimulus (eg., a sexual object) appears at a supra-threshold level of stimulation in the subject's sensory field (CS).

2. The stimulus is identified as having a meaning (e.g., its contextual framework implies a threat, impending danger, hurt, etc.4) of anxiety evocation (higher order conditioned CS).

3. Technically, one must postulate that there originally was a painful or emergency situation to serve temporarily as an unconditioned stimulus, although this stage may be missing in the mature form of the neurotic chain (UCS).

4. Anticipation of anxiety as a set occurs following the assignment of the meaning of anxiety evocation, to serve as a higher order conditioned, or secondary, 'unconditioned' stimulus (secondary drive stimulus or higher order UCS).

5. An autonomic (sympathetic) anxiety response is the automatic unconditioned response to pain or its equivalents (eg., sudden stimulation, loud sound, etc.). The importance of this step in the chain, although it may not occur once the pain stimulus ceases to occur, is that it likely sensitizes the autonomic system to render it more labile, even in response to representational symbols (UCR).

6. A series of anxiety sensations or 'feelings' occurs. These were initially activated as the afferent representations of sympathetic visceral responses, but they are readily reactivated by the anticipation of anxiety, mediated by the anxiety meaning of the stimulus. These anxiety sensations serve as the other component of the unconditioned stimulus, in addition to the secondary drive stimulus of anxiety anticipation (secondary drive stimulus or higher order UCS).

7. The arousal of further autonomic activity, and the (resulting) increase in general activity level of the organism serve as the unconditioned response to anxiety and its sensations. It does not matter for this model whether the increase of activity output associated with increased anxiety drive is random (in the Hullian sense) or directed (e.g., as a result of some sort of imprinting). The drive incremented response output will rapidly be shaped (UCR and higher order UCR).

8. Avoidant or defensive behaviour, which serves to restrict or direct the variability of the response output aroused, is shaped rapidly, operantly or through instrumental escape, from the activity output, thus incremented in response to the increased anxiety drive (CR and operant R).
9. Anxiety-relief follows if the avoidant behaviour generated is successful in reducing exposure to the CS or UCS. This serves as a reinforcing drive reduction to reward the maintenance of that avoidant response in the presence of any or all of the stimuli in the preceding sequence (drive reduction or reinforcement).

This model provides that each occasion on which the symptomatic(defensive) behaviour is elicited will serve as a practice trial, strengthening the habit for that response to be elicited in the future under any or all of those stimulus conditions. The tendency for each symptom elicitation to add to its own habit strength, and thus to the self-perpetuation of the symptomatic behaviour, is made likely in this model by the provision that each elicitation of a symptom will be reinforced operantly by subsequent drive reduction, and the provision of a complete classical conditioning sequence to add to the habit strength of the anxiety response in the given stimulus situation. Of course, it is necessary to provide a means by which the anxiety response to the stimulus situation can be maintained to insure that anxiety is present in the situation in order to be relieved as reinforcement for the avoidant behaviour.

It will be seen that the above model provides for the self-perpetuating character of the neurotic chain, and that relatively little need be assumed about causal or aetiological factors in order to satisfy the requirements of the model. Moreover, as a theoretical model, the appropriate test of the model involves the question of its utility rather than of its inherent validity. Consequently, it may be well, before extending the model to include the 'psychoses,' to digress briefly to examine some of the testable possible consequences of this model for the 'neuroses.'

<u>Treatment considerations</u>

The neurotic chain elaborated above contains two overlapping conditioning sequences -- a classical conditioning sequence and an operant conditioning sequence -- each contributing to the maintenance of the habit strength of the whole chain.

Insert Illustration I about here

Interruption of the chain at any of several points might be expected to block further acquisition of habit strength for the chain, and thus contribute to extinction of the self-perpetuating character of the chain. Specifically, intervention at any of the following points in the chain might be feasible, and might be expected to reduce the habit strength of the chain:

a) Interference with the anxiety-relief contingent upon 'successful' avoidance should tend to reduce the habit strength of the avoidance response by robbing it of its reinforcement, although it might not interfere at once with the classically conditioned maintenance of the anxiety response or of the avoidance response as a CR. Consequently, symptom-contingent aversive stimulation (to counteract the effects of avoidance of the anxiety stimulus, and hence to neutralize anxiety-relief) as a treatment technique would be expected to have some effects upon symptom production (frequency) on a temporary basis, but would be expected to yield rather low power effects upon the neurosis (i.e., relatively low frequency of success in treatment, and relatively poor follow-up).

b) Interference with the avoidance response should provide for the eventual decay (extinction) of the operant element of the chain, and should permit somewhat active extinction of the classical elements of the chain by preventing the occurrence of one part of the CR. Consequently, therapies in which the subject is immobilized (eg., Stampfl's, 1967, Implosive method, where the subject may be restrained, and Wolpe's, 1958, Reciprocal Inhibition Therapy, where immobility is effected mediated by trained muscle relaxation) so that avoidant behaviour is at least difficult, would be expected to yield higher power effects upon the neuroses than aversive methods, since the immobility methods suppress part of the last element of the classical sequence – that is, the avoidant or defensive response – and thus also suppress the anxiety-relief reaction.

c) Interference with anxiety responses or sensations would be expected to yield fairly high power therapeutic effects on the neuroses if the resulting suppression of anxiety could be sufficiently complete to prevent its occurrence and to prevent the awareness of it. In these terms, anti-anxiety medications and anxiety-relief conditioning should have fairly powerful therapeutic effects, were it not for the fact that both of these methods (drug and conditioned) in most of their applications are sufficiently non-specific with respect to subjects' anxiety stimuli, and thus with respect to the conditioned responses they affect, that they may not generate particularly large effects on particular response (e.g., anxiety) systems. Moreover, at the present stage of development of medications and anxiety-relief conditioning techniques, only relatively incomplete suppression of anxiety responses can be achieved (or perhaps even tolerated by the subject). Consequently, in spite of the hypothesis that interference with anxiety responses should centrally affect the neurotic chain (by removing the anxiety available for anxiety-relief reinforcement, by reducing the need for the avoidant behaviour, by reducing the drive activated increase in response output, and by removing the other part of the CR and UCR from the

classical element of the chain), relatively low power effects from such interference should be expected at the present stage of development of techniques.

d) Interference with the anxiety meaning of the stimulus would be expected in this model to have relatively low power therapeutic effects on the neuroses, at least initially, since such interference would be directed mainly at the higher order conditioned elements of the chain. Consequently, one would expect that conventional psychotherapies (e.g., support, reassurance, interpretation, which act upon the symbolic systems) would have relatively low power effects on the neuroses, unless such techniques were effective in greatly reducing the anticipation of anxiety, or were effective in producing a strongly competing higher order conditioned expectation in the subject. However, it is also possible that intervention in the initial higher order conditioned elements of the chain might eventually result in decay of the anxiety response arising from reduction in the expectation of anxiety.[1]

e) Interference with the situational anxiety stimulus has also been used as a treatment technique. The subject may be removed from his usual environment into a new environment (e.g., a hospital) where anxiety stimuli may be more controllab1e, or the subject's social environment, as a source of anxiety stimulation, may be modified (e.g., by family therapy or rehabilitation) in order to reduce the frequency and intensity of anxiety stimuli in the subject's environment. While such interferences might serve to reduce the presence of anxiety responses temporarily, or might even permit eventual decay or extinction of the chain from disuse, it would seem likely that the habit strength of the chain could quickly be revived, so that the effects of such interventions would be expected to be rather temporary on the basis of this model.

The above hypotheses are stated solely to provide a context for the model under discussion. No satisfactory data is yet available in the literature on the basis of which to compare the relative power and efficiency of the several treatment methods referred to above, so that it is not yet possible to verify the above derivations from the model presented. However, such a task should certainly be possible in the future, and we are preparing to test some of the above hypotheses.

Of course, it is not necessary to restrict oneself to interfering with only one component of the neurotic chain at a time. Indeed, it would seem defensible to argue that the greater the number of components interfered with in a treatment application, the greater the probability of good and powerful treatment effects. For example, it seems justifiable to argue that the relatively powerful effects of Wolpe's (l958) RIT procedure, as reported in many studies appearing in the literature

---

[1] There are at least two existing techniques which may intervene with either the anxiety response or the higher order conditioned elements of the chain. The technique which we sometimes use of pairing a pre-conditioned anxiety-relief signal with the presentation of an anxiety stimulus may serve directly to affect either of these components of the chain. And the technique used by Wolpe (1958) as an auxiliary monitor of therapeutic progress, may perhaps account in large measure for his therapeutic results. Wolpe requires patients to rate the way the feel at the beginning and end of each session on a hundred point rating scale. Since relaxation intervenes between the two daily self-ratings, it is possible that the overt symbolization of subjective change is being operantly reinforced.

(Wolpe, l958; Lazarus, 1961; Lang & Lazovik,1963; Lang, Lazovik & Reynolds, 1965; Paul, 1966), may be partially attributable to the relatively large number elements of the neurotic chain affected in the method. The RIT method, by requiring deep muscle relaxation, at least induces immobility, and hence reduces avoidant behaviour and, presumably, prevention of anxiety-relief. The RIT method manipulates the anxiety stimuli and, by monitoring subjective levels of anxiety in response to the stimuli, insures that the anxiety levels stimulated are low enough to permit the system to handle the anxiety generated. Moreover, Wolpe (l958) claims that the relaxation induced modifies the sympathetic anxiety response by activating a competing parasympathetic response. Thus, although the last assumption may not be entirely justified (Rachman, 1968), it seems likely that Wolpe's procedure permits interference with at least three elements of the model proposed for the neurotic chain.

The possibility that interfering with several components of the neurotic chain might increase the power of a treatment method suggests the desirability of modifying as many components of the neurotic chain as possible in a treatment method. In the treatment model to be discussed later, an attempt will be made to control or modify all of the following components of the neurotic chain: (i) the anxiety-evoking stimuli, (ii) the anticipation of anxiety, (iii) the autonomic anxiety response, (iv) activity output and avoidance responses, (v) anxiety-relief attendant upon avoidance, and possibly (vi) anxiety sensations. Each element of the neurotic chain is thus represented in some fashion in the treatment model, and the extent to which each element is taken into account in the treatment method may be adjusted to some extent to suit the needs of the individual case.

However, before the treatment model is discussed it seems necessary to discuss briefly a model for the 'psychoses.' This is necessary largely in order to show why some procedural elements employed in RIT had to be abandoned in the treatment method under discussion.

## Model for the Psychoses

As is true of neurotic behaviour, 'psychotic' behaviour is relatively trivial and unimportant until it persists in the face of an individual's attempts to control it and in spite of normal environmental contingencies. Consequently, a useful model for the 'psychoses' need not concern itself with the aetiology of the condition, it if can account, relatively parsimoniously, for many of the moment-to-moment behavioural events observed in the 'psychoses', and can account for the persistence of the disorder. With some begging of the question, an interesting model for the persistence of 'psychotic' behaviour can be developed using little more than the assumption of a conditioned neurotic chain, and an assumption made by Mednick (l958) to the effect that, in 'psychotics,' anxiety increases stimulus generalization excessively. On the basis of these assumptions, the following sequence of events is presumed to occur in the moment-to-moment living of 'psychotic' subjects (see Illustration II):

1. A neurotic chain is conditioned and becomes self-perpetuating. The function or effect of this neurotic chain is to generate and maintain anxiety responses in certain stimulus situations.

2. Stimulus overgeneralization occurs as a result of the anxiety thus generated and perpetuated.

This stimulus overgeneralization results in two main sequences of events:-

3. (a) there is a rapid acquisition of a wide range of anxiety stimuli so that, eventually, few situations fail to evoke anxiety reactions, since most situations contain some stimuli generally like the anxiety-provoking stimuli;

(b) generalized or pervasive anxiety appears, maintaining fairly continuous, high intensity anxiety levels (which may, for example, prevent an experimenter from stimulating or modifying the level of anxiety manifestation – "flatness");

(c) there may be generalized avoidance of or withdrawal from the stimulus environment ("social withdrawal");

(d) there may develop a sense of isolation and social deprivation, removing the subject both from the effects of social reassurance and safety, and from complex distractions from his preoccupations with anxiety stimuli and his own reactions to them;

(e) these events may then lead to further increase in the subject's fear and anxiety, hence to further stimulus generalization, and hence to further repetition of the cycle of this sequence of events; and

4. (a) there may be a reduction in perceptual acuity, arising from a breakdown in effective perceptual discrimination, attendant upon reduction of differentiation of responses to stimuli, associated with the increasing equivalence of different stimuli in terms of (anxiety) response evocation;

(b) a diffusion of figure and ground may result from the reduction in perceptual acuity, so that figures may appear more 'groundal' and permeable – which, among other things, might lead to body image disturbances of the types described by Fisher and Cleveland (l958), and hence may lead to further fear and anxiety;

(c) there may develop a reduction in conceptual clarity, arising from the reduced perceptual discrimination and the consequent difficulty in classifying events accurately, which, among other things, might lead to confusion in thought or condensations of inappropriate perceptual elements in concepts, and hence may lead to further fear and anxiety;

(d) stimulus ambiguity and uncertainty may also arise as a produce of disturbances in perceptual acuity, figure-ground perception, and conceptual clarity, and this may result in brief disorientation in time and place which may, in turn, result in difficulty for the subject in predicting events in himself and his environment, and this may also lead to further fear and anxiety; and

(e) anxiety generated from all these sources may then further increase stimulus overgeneralization, and thus repeat the cycle to generate further anxiety.

5. In some 'psychotics,' however, the cycle of increasing anxiety may be blocked in part by a

‘corrective’ neurotic chain whose function is to avoid uncertainty and confusion in dealing with the stimulus environment by imposing “sense” or meaning on events. One form of this defence actively seeks out (all possible) solutions to the meaning of events in order to maintain an order in the stimulus environment (i.e., ‘obsessive' or ‘pseudoneurotic schizophrenia’); and another form of this defence anxiously adopts one solution out of the many recognized possibilities as the “correct” meaning to be assigned to an event, again in order to maintain an order in the stimulus environment (i.e., ‘paranoid').

Insert Illustration II about here

The above model has some advantages over many others which might have been posited equally legitimately. Its parsimoniousness is only partially cancelled by the reasoning by analogy that it employs. It accounts in part for many of the behavioural observations that can be made in the schizophrenias. It accounts for the perpetuation and growth of the symptoms, and it dovetails with the model for the neuroses. It is a behavioural model which makes anxiety of central importance in the disorder, both as a symptom and as a cause. And it suggests rather general stimulus dimensions which may be implicated in the process of anxiety evocation in the psychoses, and which may, therefore, be important to desensitize in treatment.

It is true that the treatment model to be discussed later deals only peripherally with the centrally implicated aetiological factor in this model for the ‘psychoses,’ namely, stimulus overgeneralization. However, the treatment model does deal with the hypothesized stimulus for stimulus overgeneralization in these subjects, namely anxiety, and other accessory treatment methods can be used to deal more directly with the problem of stimulus overgeneralization when this is necessary. Moreover, in addition to positing the relevance of anxiety (which the present treatment method does purport to treat) to stimulus overgeneralization, and hence to the symptomatology of the 'psychoses,’ the above model for the ‘psychoses’ serves three important functions in this presentation. First, it provides some implicit justification for the attempt to use an anxiety deconditioning procedure in the treatment of the ‘psychoses.’ Second, it suggests means for constructing anxiety stimulus dimensions which might be appropriate to use in desensitizing ‘psychotic’ subjects. And third, it draws implicit attention to some of the difficulties encountered in the attempt to treat ‘psychotic’ subjects by means of desensitizing methods.

## Treatment considerations

Insofar as the above model for the ‘psychoses’ may be said to be valid, it will be clear that certain treatment considerations follow. In the first place, the postulate that anxiety is increased by the loss of close and clear contact with the stimulus environment (attendant upon reduced perceptual acuity, cognitive clarity and social contact) should suggest that any procedural requirement made upon the subject which increases his sense of isolation from his stimulus environment, or which implicitly robs him of control over the maintenance of proprioceptive or external environmental stimulation, will tend to increase his anxiety, and hence his symptomatology. Thus, structuring the treatment situation to reduce external stimulus input (e.g., requiring the subject to close his eyes), or to reduce proprioceptive stimulation (e.g., requiring the subject to relax his muscles and

to remain still), should have anti-therapeutic effects with ‘psychotic’ subjects, and should therefore be avoided as requirements of the treatment procedure, in order to avoid making the treatment situation noxious for the subject and hence to be avoided by him. In the second place, the postulated fluidity of the perceptual processes of ‘psychotic’ subjects suggests that they would find it extremely difficult to form or retain mental images volitionally, or to judge the relative degree of subjective reaction to mental images or stimuli in terms of such estimates as intensity of anxiety reaction. This, in turn, would suggest that a technique which required volitional formation of mental images by the subject or refined judgements concerning subjective states and reactions would prove difficult, if not ineffective, with many ‘psychotic’ subjects. Moreover, even the process of establishing a personal hierarchy of anxiety stimuli tailored to the particular needs of a particular subject, such as that used in Wolpe’ RIT procedure, may prove difficult or impossible for some ‘psychotic’ subjects in view of the rather fine discriminations required in setting up such hierarchies between degrees of anxiety experienced by the subject in response to successive items in the hierarchies. In view of these observations, in order effectively to treat ‘psychotic’ subjects, it may be necessary to avoid the use of personally constructed personal hierarchies, to avoid reduction of external environmental stimuli as much as possible, to avoid requiring the subject volitionally to form and to hold mental images, to avoid requiring the subject to monitor his own moment-to-moment levels of subjective disturbance, and to avoid requiring the subject to relax, or even to sit still.

In the third place, the hypothesized disturbances of body image and of subjective awareness in the ‘psychotic’ subject would suggest that any procedure which requires the subject to attend to, react to, or report his subjective states, of the state of his body, may serve to increase his anxiety in that situation. It would therefore seem desirable for a treatment procedure which is to be used with 'psychotic’ subjects to avoid requiring the subject to monitor or report his own subjective states, or to attend too closely to the state of his body functions.

Finally, the posited reciprocal interaction between anxiety and stimulus overgeneraliztion in ‘psychotic’ subjects suggests that some means needs to be found for treating psychotic subjects which either reduces stimulus overgeneralization and hence reduces the consequent anxiety-incrementing symptomatology, or which reduces the anxiety generated by the symptomatology and hence reduces the tendency to further stimulus overgeneralization. Although other methods are available which seem to reduce a subject's tendency to stimulus overgeneralization, since the treatment method under discussion is concerned primarily with the reduction of anxiety, it would seem necessary for it to contain some means for reducing the kinds of anxieties associated with stimulus overgeneralization-generated symptomatology. The model for the 'psychoses' presented postulates that the kinds of anxiety stimuli which are likely to increase a psychotic subject’s level of anxiety grossly, and hence his tendency to stimulus overgeneralization, should include such anxiety stimuli as form distortion or inaccuracy (to represent 4a, perceptual inacuity), figure permeability or penetration (to represent 4b, body image disturbances), condensations of unrelated images (to represent 4c,disturbances of conceptual clarity), and stimulus ambiguity (to represent 4d, ambiguity and uncertainty). It is not difficult to construct stimulus dimensions to represent these types of stimuli.

It will be seen that the model for the ‘psychoses’ supplied both a system of constraints to be

imposed upon a treatment method to be adopted for use with ‘psychotic’ subjects, and some indications of some of the types of accessory dimensions of anxiety stimuli which might be needed in order to deal with the kinds of problems posed by ‘psychotic’ subjects. Now with the treatment considerations relevant to the neuroses and the ‘psychoses’ in mind, it may be possible to proceed to a discussion of the treatment method which was derived.

## Treatment Method

The subject is seated comfortably in a high-backed (lounger) chair. The high back supports the head and neck in order to insure maximum comfort and some ease in sitting still. It is desirable for the subject to sit still in order to prevent defensive avoidance and anxiety-relief which perpetuate the neurotic chain. Consequently, if the subject finds it difficult to sit still, a high-backed chair makes it easier for him.

The subject is facing a projection screen and two speakers. In our application, the subject is in one room and the operator and equipment are in an adjoining room, to shield the subject from the equipment noise, and to minimize the relationship between the subject and the operator. The projection screen, in our application, is a milky plastic sheet mounted on an observation window. The subject will receive his visual stimulus presentations (pictures of cemeteries, from planes, etc.) on the screen, and his auditory stimulus presentations (recorded sounds of gunfire, creaking doors, etc.) from one of the speakers. Thus he can be subjected to anxiety stimuli which are under close control by the operator, without having to form mental images volitionally, without having to hold or retain mental images, and without having to reduce his contact with the stimulus environment too greatly.

The subject is instructed to sit absolutely still, and he is told that the reason for this is that if he does not sit still he will waste some time by upsetting the recordings we wish to make of his responses. It is true that it is not absolutely necessary for him to sit still. However, it is obviously desirable that he sit still (if only because in the physiological recordings which will be made motion produces artefacts which may slow down the procedure somewhat). To help him sit still, the second speaker will supply taped relaxation instructions if the subject wishes to receive help in relaxing. However, since the relaxation instructions are considered here to serve only to help the subject sit still and to give him a sense of having ‘company’ in the room with him, they are quite optional in the procedure. If the subject cannot sit still, whether or not he elects to receive the taped relaxation instructions, an 'immobility conditioner’ has been devised which emits a low intensity slightly aversive tone whenever the subject moves, and this seems to result in fairly rapid development of relative immobility.

Electrodes are attached to the subject to record arousal levels on a monitoring basis. We have elected galvanic skin resistance (GSR) as our main monitoring modality. There are several reasons for this choice. Technically, the GSR has an advantage over respiration and pulse recordings because it is less under conscious control, and it has an advantage of muscle potential recordings in that it is less influenced by movements of the subject(especially if both electrodes are placed on the same hand). Although the GSR has a relatively long latency of response, in practice using the technique under discussion, its latency (in terms of operator reaction to

changes in the recordings) is actually shorter than the same latency using respiration or pulse rates, although not perhaps if one were using a cardiotachometer. Most important for present purposes, the treatment model under discussion suggests an interesting way to view the relevance of GSR recordings to anxiety, which will be discussed in the next section, under the model for the treatment.

The subject is left in a dimly lighted room, in which he can make out familiar forms. He sits alone for a few minutes before the conditioning procedure begins. This period of time may vary from a minute or two while the operator sets up his equipment, to nine or ten minutes during which time GSR baseline has usually been established. Relaxation instructions may be played to the subject during this period of time, or he may just be left to sit and wait.

During the initial period or time, the operator loads the selected slide tray and/or auditory stimulus tapes. The present discussion will be restricted to visual (slide) stimuli, since these were the stimuli used exclusively with the subjects whose results are reported later. Slide trays have been prepared containing standard stimulus hierarchies, each tray depicting a different anxiety stimulus dimension. The trays were originally prepared from a list of common classes of phobias taken from Fenichel (1945), and some categories were added based on the model for the psychoses already discussed. The stimulus categories to be used with a particu1ar subject are selected on the basis of his responses to a revised form of the Fears Schedule used by Wolpe (l958), revised to include some of the kinds of anxiety foci reported by psychotic subjects. The trays are used in a kind of rotation (one tray per session) among the categories selected for the subject, unless the subject reports having experienced a particular kind of stressful situation (relevant to a particular one of the stimulus dimensions) when he comes for his treatment session. In the latter case, the stress-relevant tray is selected for use on that day.

The slide projector is turned on when the conditioning session is to begin. The operator may turn the projector on only when the GSR baseline has been established, or he may elect to turn the projector on after only a minute or two, while the GSR is still climbing toward its baseline. In the latter case, the purpose is to use the natural initial GSR increase to its baseline as an opportunity to provide some reinforcement in order to speed the establishment of a conditioned GSR level increase. In either case, however, when the first slide has been turned on the screen (being viewed by the subject), the operator waits. He waits until the probable GSR (downward) reaction to the appearance of the slide stops and the GSR again starts to return (upwards) toward its baseline. When the GSR level, or the basal skin resistance (GSR), has started to recover upwards towards the subject's baseline, the operator advances to the next slide. Again he waits, this time until the GSR increases above the GSR reading at which the last slide was changed, at which time he advances the slide again. The treatment procedure now involves nothing more than waiting, and then advancing the slide again for each successive increase in GSR above the level at which the last slide was changed. The amount of GSR increase above the level at which the last slide was changed which the operator requires in order to change to the next slide will vary depending upon the GSR lability of the particular subject, but it may be set quite arbitrarily. We select the quantity of GSR change to be required between each slide change on a daily basis for each individual subject. The quantity of GSR increase we require to warrant slide change is set in such a way that the subject is shown a new slide every 15 to 30 seconds during the first two

minutes of conditioning. In the average subject this amounts to an increase of between 1000 and 5000 ohms of skin resistance between slides.

The session covers a standard time interval. In our application we use half hour sessions. When the treatment session time has elapsed, the slide projector and GSR are turned off, and the electrodes are disconnected from the subject. Finally, several items of information are recorded on the subject's treatment card, including the category of slide stimuli shown to the subject during that session, and the number of slides from that tray shown during that session.

## Model for the Treatment Method

The above routine procedure defines the operations involved in the treatment method under discussion. The treatment procedure, however, involves several theoretical considerations. These more theoretical issues require brief comment.

The treatment procedure seems to involve two overlapping conditioning processes. In the first place, the GSR is operantly shaped steadily upward, towards increasing ‘comfort' (see below). In the second place, the ‘comfort' thus achieved is paired repeatedly with the slide stimuli, in order classically to condition the 'comfort’ response to the content of the (formerly) anxiety arousing slides. This is accomplished in the procedure by the simple tactic of using the act of slide change as the reinforcer to shape the GSR upwards operantly, and then using the elevated GSR as the ‘evidence' of 'comfort’ in the subject to condition (classically) the subject to a ‘comfort’ response to the content of the slide stimuli. While the latter (classical) component of the treatment procedure (conditioning the obtained reactions of the subject to the content of the slide stimuli) will pose little difficulty for its understanding, it may seem quite difficult to understand on what basis it may be stated that slide change might serve as a reinforcer for GSR shaping, and it may seem difficult to accept the simple proposition that GSR increase may be used as a satisfactory evidence of ‘comfort’ in the subject. These latter two issues require some discussion.

In the first place, it must be stated that, aside from the rather presumptive evidence implied in the data to be presented later, there is no satisfactory evidence available on the basis of which to state that GSR can be shaped operantly by means of slide change. This is merely an empirical statement based upon daily experience with patient subjects, based initially on a series of chance observations while working with patients. However, there is some evidence in the literature which shows that the GSR may function as an operant response, and can be shaped (e.g., Kimmel and Hill, 1960; Van Twyver and Kimmel, 1966; Gavalas, 1967). And there is some evidence that some operant responses can be shaped using slide change as a reinforcement (Benton and Mefferd, 1967). However, it was our chance observations, leading to our daily operations with patient subjects involving the use of slide change as a reinforcer for GSR, which led to one of our current series of experimental investigations using normal subjects in which we are testing several different types of reinforcers derived from hypotheses concerning the possible parameters involved in the reinforcement value of slide change. The current hypotheses under investigation in these studies (to be reported elsewhere) are that the apparent reinforcement value of slide change upon GSR derives from (a) the reduction in boredom arising from termination of one slide, or the increase in interest arising from the appearance of a new slide, (b) the change of

stimulation or the motion resulting from rapid, as opposed to slow, slide change, and (c) the reduction in anxiety stimulated by a slide resulting from the termination of that slide (time for which anxiety reduction is given by the apparent fact that it takes a brief interval of time before the anxiety evoking meaning of the next slide in the series is apperceived by the subject). Meanwhile, we are only able to hypothesize or assume that it is the reinforcing effect of slide change which permits us to generate anxiety suppression or 'comfort' in our patient subjects.

In the second place, it must be stated that, aside from some rather peripherally related data which appears throughout the literature, there is no satisfactory evidence available to permit one to conclude that shaping the GSR upwards might result in a state of 'comfort' for the subject. There is considerable evidence and assumption that GSR responses (downwards deflections) may reflect momentary increases in anxiety or, more properly, increases in arousal which may occur when an anxiety response is activated. For this reason, GSR (downwards) reactions have sometimes been used as evidence of anxiety responses, and the intensity of the anxiety responses have sometimes been inferred from the latency and/or amplitude of the GSR response. It is our impression, however, which seems to be supported by much of the discussion of the GSR in the literature, that no single GSR measure (latency, amplitude, etc. of response) is a satisfactory measure of anxiety. This applies also to the use of the subject's GSR baseline (or BSR) as a measure of anxiety. However, we have hypothesized, for study in another current research investigation, that a satisfactory measure of anxiety from the GSR might be taken to be the absolute overall amount of variability (i.e.,total range or variance in ohms) of the skin resistance, taken across many different sessions, across many days, and subject to a wide range of stimulus conditions. Such a measure of anxiety from the GSR would take into account all of the conventional anxiety measures from GSR. Thus, the GSR and its variability would enter such a measure, as would the amplitude of GSR responses to stimulus events. Such a measure would also tend to take latency of GSR response into account, since the shorter the latency of response, the greater would be the opportunity for GSR variations to occur. Moreover, in general agreement with some preliminary findings reported by Mednick (1966), we seem to have observed some rather characteristic relationships between GSR variabilities and patients' diagnostic categories. For example, it is our clinically derived impression, which we intend to verify, that 'psychotic' patient subjects exhibit very wide ranges of GSR levels, ranging all the way from about 15K ohms to 1000K ohms and more; neurotic patient subjects seem to exhibit a somewhat narrower range of GSR levels, ranging from about 20K ohms to about 350K ohms; and relatively normal subjects seem to exhibit an even narrower range of GSR levels, ranging from about 30K ohms to about 200K ohms. While these are very rough estimates, drawn from memory, they are stated here in order to illustrate a point of view regarding the possible relevance of GSR levels to anxiety, especially in the light of the models which have been posited for the 'neuroses' and for the 'psychoses.' Still more important, however, from the point of view of the present discussion is the consequences of this hypothesis concerning the relevance of GSR levels to anxiety for the treatment model under discussion.

It may be noticed that, as long as GSR is conceived as being generally related to anxiety reactions, the treatment method can be perceived as potentially effective in modifying anxiety regardless of whether the relevance of GSR recordings to anxiety is taken to be represented best as the downwards GSR deflections which are associated with arousal, the absolute GSR

(baseline)level, or the absolute amount of GSR variability on many different days and under many different stimulus conditions. If the best GSR measure of anxiety turns out to be the downwards GSR deflections associated with arousal, then the treatment method may be conceptualized as shaping toward a reduction in the frequency and intensity of such downwards deflections by shaping the CSR steadily upwards away from such reactions. If the best GSR measure of anxiety turns out to be the absolute GSR level, as a measure of amount of autonomic activation, then the treatment method may be conceptualized as shaping the GSR upwards toward its 'comfort' direction. And, if the best GSR measure of anxiety turns out, as we hypothesize, to be the absolute amount of GSR variability (under many conditions), then the treatment method may be conceptualized as shaping a general reduction in GSR variability by shaping GSR upwards against its own natural ceiling (which, interestingly enough, then starts to shift downwards) to shape less allowance for variability. Thus, insofar as GSR is in any respect a satisfactory measure of something akin to anxiety, it seems possible that the treatment method can be conceptualized as shaping a reduction in some aspect of anxiety, while pairing that reduction with the content of anxiety stimuli (slides).

It is argued, then, that this treatment procedure, which we have called Stimulus Conditioned Autonomic Response Suppression (SCARS), can permit modification or manipulation of all of the following variables: (i) anxiety stimuli, (ii) the autonomic anxiety response, (iii) the avoidant or defensive behaviour, and (iv) the anxiety-relief contingent upon avoidance. In addition, in some applications, we have pre-conditioned an anxiety-relief response in the usual fashion to a light stimulus or a low tone, and the conditioned stimulus is then used as a conditioned means to generate anxiety-relief during the SCARS treatments, either to stop a strong CSR (downwards) response in reaction to a slide stimulus, or to assist GSR elevation when a low ceiling is obtained. In these terms, it might be argued that the procedure also permits accessory modification of (v) the conditioned anticipatory anxiety expectation discussed in the model for the 'neuroses.' This same pre-conditioned anxiety-relict method has also been used occasionally, along with a slight alteration of procedure for SCARS (repeating the same slide tray until it is completed, and then moving to the next slide tray, rather than rotating through different slide trays or stimulus categories on successive sessions), in order to keep the subjective level of anxiety low throughout the treatment process. In this application, the pre-conditioned anxiety-relief stimulus is turned on to be paired with any stimulus which is expected or is found to arouse a particularly strong anxiety response. We have no idea why going through one category of anxiety stimuli at a time, rather than rotating through the categories (which seems to result in an initial increase in anxiety level, which peaks on the average at the thirteenth session), has its apparent effect. However, using this tactic, especially when it is coupled with accessory use of a pre-conditioned anxiety-relief signal, seems sometimes to have the effect of (vi) minimizing the subjective anxiety sensations of subjects.

The foregoing paragraph will have raised at least three technical and theoretical questions in the mind of the reader, namely, why the name SCARS for this treatment method, what expectations may one have about the course of such a treatment program, and when may one say that treatment or conditioning with respect to a particular (slide tray) category of anxiety stimuli is 'completed.' These questions will be discussed briefly in reverse order.

We have adopted a kind of rule of thumb by which to decide when a treatment program is 'completed.' It is our contention that if a subject is still anxious concerning a particular stimulus dimension, there should be some evidence of a kind of resistance to upwards shaping of the GSR. Such evidences might be observed in several downwards GSR reactions to stimuli in the dimension, or in a rather sluggish reaction to slide change resulting in slow GSR elevation. In either case, the result should be that the subject is shown relatively few slides in a time-limited session. Consequently, we employ retrospectively the subject's treatment record, which shows the number of slides shown in each session (and the category used at each session), in order to determine when a subject has been conditioned satisfactorily on a given stimulus category or dimension. Using our procedure, we accept exposure of over 80 slides in a single half hour session as satisfactory evidence that treatment is completed with reference to that stimulus category, unless (a) the session is the first session during which that stimulus category, claimed by the subject to be anxiety provoking, was shown,(b) the subject is not too 'dysthymic' and has had less than seven treatment sessions, and (c) the subject is quite 'dysthymic' and has had less than eleven treatment sessions. The last two exceptions are made due to the apparent fact that it takes a while initially for GSR conditioning to 'take,' during which time a large number of slides may possibly be shown at a session. The treatment program is completed when all of the main categories of anxiety stimuli reported by the subject have been exposed to the above criterion.

It is clear that the present treatment method does not treat subjects specifically for those stimuli which are anxiety provoking for them in their own lives. That is to say, this method uses standard stimulus dimensions or hierarchies which, at best, may represent for the subject similar types of anxiety stimuli in his life. Moreover, the conditioning involved is directed at the relatively non-conscious sweat aspect of the autonomic response, rather than at the subjective anxiety response which brings the subject to seek help. At best, then, the treatment procedure might be expected to work rather slowly due to the stimulus generalization and the transfer of training required. And, indeed, there is little evidence available that altering one modality (such as GSR) can be expected to produce any major effects upon another response modality (such as subjective anxiety), especially when the two response modalities are probably associated with different levels of behavioural analysis. Of course, both these questions are empirical issues which can only be resolved with data. However, some plausible expectations might be formulated before such data is made available.

When a subject is asked to report (eg., on an anxiety questionnaire) what is disturbing or anxiety provoking for him, he must surely search through his memories of past experiences to determine his reactions to similar stimulus events. From his reactions to a series of stimulus situations, we may be able to determine, by the process of abstraction, the categories of events to which he tends to react with anxiety. There is no guarantee, however, either that he will again be subjected to stimulus situations identical with the stimulus situations in which he felt anxious in the past, or that we will manage to ask him about just the right kinds of stimulus situations for him. If we were able to obtain just the right information about the anxiety situations he had formerly experienced, desensitizing him for just those very situations, as they are represented in his memory, would probably permit us to clear him of his anxieties about those past situations. But, our treatment activities are surely directed towards the subject's future rather than toward his past, even though the best way to predict events which may in the future be stressful for him may

perhaps be to determine the sorts of events which were stressful for him in the past. Still, since he may never experience in the future situations exactly like past stressful events, it might perhaps be well to direct treatment activities toward general classes of events such as those he was anxious about in the past, rather than toward specific anxiety situations of his past. We would then be counting on stimulus generalization to permit him to respond to entirely new situations in the future with comfort or non-anxiety. Insofar as such a treatment goal is held to be sound or appropriate, it seems reasonable to us to assume that such desired stimulus generalization to other related, but new, stimulus situations would be facilitated by conditioning directed toward a category of stimuli (with which, perhaps, the subject has not had much or any direct experience), which might serve as it were to force stimulus generalization, rather than by conditioning him with respect to stimuli from his own past life (which might be inclined to restrict the variability of his associations), which would probably be 'asking' for a restriction of stimulus generalization during training. In other words, it is argued that just as efficient, and perhaps more lasting, results might be expected using general and standard stimulus hierarchies compared with the results which might be expected using anxiety stimuli selected specifically for the particular subject. In any event, using, as this method does, slide (picture) stimuli for the presentations of anxiety stimuli to subjects, it would be virtually impossible to arrange for individual slide sets for each new subject, based directly upon the situations of his past in which he experienced anxiety. Consequently, we use standard stimulus hierarchies organized under standard categories, with each slide selected initially as hypothetically related to the anxiety category in question, and then rejected or retained, and later hierarchically organized for increasing intensity of anxiety stimulation, based upon the presence and intensity of obtained GSR (downwards) deflections from groups of subjects claiming to be anxious in reference to the anxiety stimulus category in question.

The problem of transfer of training from one modality to another poses a more serious difficulty. That it does occur during learning practice across psychophysiological modalities (respiration, pulse and GSR) has been shown for one stress situation by Fenz and Epstein (1965, 1967). That such transfer of training from one modality to another, even across levels of analysis, should occur seems reasonable, and seems to be observed in every day experience. However, whether or not it can be shown to occur in studies planned for the future, it at least seems reasonable to expect that such transfer of training should require a considerable period of time to take place. And it is on this basis that we have hypothetically accounted for one rather peculiar aspect of the course of subject progress associated with SCARS treatments. In the first place, we have noted that it usually takes some time (3 to 6 sessions) for the slide stimuli to begin to produce appreciable GSR (downwards) reactions. In the second place, we have noted that during the middle period of the treatment program (peak averages about the 13th session) the subject often seems to experience stronger subjective anxiety than he experienced when he presented himself for treatment. And, finally, in contrast to subjects treated with other methods, we have noted that it is often only after a period of from four to six months following the termination of the treatment (no treatment in the interim) that the subject experiences freedom from subjective anxiety. We are aware that this whole problem requires much more study.

Finally, the question of the title given the treatment method requires some explanation. We contend that the method places the autonomic response (measured in our initial applications on

the GSR) under stimulus control – initiated by slide change reinforcement, but eventually conditioned to the content of the slides. Its effect should be to suppress the autonomic lability, or to modulate the autonomic response, in the face of formerly anxiety arousing stimuli, and perhaps also the subjective anxiousness associated with the anxiety stimuli. Consequently, we have titled the method, stimulus control over autonomic responses, conditionally suppressing these, or Stimulus Conditioned Autonomic response Suppression (SCARS).

Initial Clinical Trials: Contrast Study

Subjects

Clinical trials have been run using the SCARS method. The subjects were 97 patients treated by the author between 1962 and l968 in his hospital and in private practices. During the first three years of this period the author was using Wolpe's (l958) method for reciprocal inhibition therapy (RIT), during which time the first group of 40 patients reported here was treated and assessed. During the following year, SCARS was being developed, and some patients, 17 in number, who were still being carried, not yet having responded to RIT, were tried on SCARS. The remaining 40 patients reported here were treated during the next two year period, during which SCARS was being tried, and these patients were completed and assessed during that period of time. Thus the 97 patient subjects reported here include 40 patient subjects treated exclusively with RIT, 40 patient subjects treated exclusively with SCARS, and 17 patient subjects treated first with RIT and then with SCARS.

Of these 97 patients, thirty were from a chronic patients' ward of a provincial hospital, of whom 13 received RIT, 7 received SCARS, and 10 received both RIT and SCARS. Ten of the 97 patients were hospital out-patients, of whom 2 received RIT, 7 received SCARS, and 1 received both. Fifty-seven of the 97patients were from the author's private practice, of whom 25 received RIT, 26 received SCARS, and 6 received both. No explicit restrictions were imposed upon subject selection for either treatment procedure, although some selective effects were imposed by circumstances in determining which subjects would be treated with which procedure. It will be seen from the above that a larger number of out-patients were referred for treatment during the period in which SCARS was being used, and that a larger number of chronic in-patients were referred for treatment during the period over which RIT was being used, due to the author's circumstances during those times. However, all patients who were referred, and for whom treatment time was available, were accepted, and the time at which they were referred was the main determinant of which treatment method they received. At the same time, some patients (not reported here) were placed on combined treatment programs, sometimes using RIT or SCARS along with other behavioural tactics, and sometimes using other behavioural tactics without RIT or SCARS. The principles for such selection of treatment tactics, however, were rational rather than empirical ones, and were basely solely upon our understanding of the patients' needs at that time, rather than being based upon the needs of a research design. The point being made here is that those patients who were referred, but who are not included here because they received other treatments, were selected out on the basis of some quasi-rational considerations, which would have changed from time to time, so that they should pose relatively little by way of a systematic effect upon subject selection for present purposes.

Of some interest, in terms of estimating severity of behavioural disruption and difficulty of treatment of the patient groups subjected to the treatment procedures, might be the diagnoses given the several patient subjects studied, and their length of illness prior to the present treatments. The number of each patient subjects in each diagnostic category is presented in Table 1. The diagnostic labels were usually assigned to each patient by his psychiatrist, although some of the patient subjects from the private practice group were referred by general practitioners rather than psychiatrists, and in these cases the diagnosis was assigned by the author (mostly phobias and anxiety neuroses). Length of illness was calculated on the basis of the patient's report and the records of the referring physician. This category refers to the number of years during which the patient had the main presenting symptoms, or the number of years during which the subject was hospitalized more or less continuously (maximum six months out of hospital at a time) for his illness. Table 4 presents the average number of years of illness per subject for each outcome category of each type of treatment. Careful examination of these two tables, using them as a basis for assessing the severity of illness of the subject groups treated by the two main methods studied, suggests that on the whole the subjects treated with RIT may have been slightly more seriously ill than the subjects treated with SCARS, although this is due entirely to the kinds of patients referred at the times when the two treatment methods were being used.

Method

The patient subjects were treated with the treatment being used at the time when they were referred. Immediately following treatment the patients were rated for outcome of treatment, and informal and variable follow-ups were carried out to identify patients where there was a return of symptoms.

The SCARS treatment method employed with these patients has already been described in detail. Both SCARS and RIT sessions were held as frequently as three times per week, and as infrequently as once per week, depending upon the operator's and patient subjects' timetables. The RIT method of treatment followed closely the method described by Wolpe (l958), although, in accord with Wolpe's views expressed in a workshop in Toronto which was attended by the author, less emphasis was placed upon the training of deep muscle relaxation than is implied in his book. At the same time several additional techniques were used in order to aid initial muscle relaxation (beyond the standard initial muscle relaxation pre-training) in those patient subjects who found muscle relaxation particularly difficult. With some of the subjects daily relaxation classes were held by a physiotherapist, and some of the subjects were given several sessions in a whirlpool bath to aid muscle relaxation. Some of the stimulus hierarchies used in the RIT treatments were standard hierarchies adopted from the literature or from work with other patients. These standard verbal hierarchies were used mainly when the patient subjects seemed unable effectively to introspect in the task of hierarchy construction. Aside from these two minor departures (somewhat different emphasis in relaxation training and some use of standard hierarchies) the attempt was made to remain faithful both to the dictates and to the spirit of Wolpe's (l958) text.

The major difficulty in the present data is certainly the outcome criteria employed. It is still standard practice to have difficulties with outcome criteria. However, due in part to limited

available facilities, due in part to the preliminary and tentative nature of the developing techniques, and due in part to the complexity of the data (patient subject material and its several sources), the outcome problem requires particular attention here.

The outcome ratings were defined initially in the following ways, and a solid attempt was made to adhere closely to these conceptual definitions throughout:-
Recovered refers to patients who, following treatment, were living and functioning as 'healthy' people in their environments. These subjects all report relief from their symptoms and anxieties, are fulfilling appropriate social roles as housewives or employees, and are not subject to any reported notable disturbances in interpersonal relationships or work. They appear to have rather good mental health.
Much Improved refers to patients who, following treatment, were living and functioning much like 'recovered' people, but who still reported occasional subjective disturbances from anxiety or symptoms, but not to the degree that they missed work or disturbed their interpersonal relationships appreciably.
Some Improved refers to patients who, following treatment, were functioning much like 'recovered' people, except that they periodically experienced bouts of anxieties or symptoms which did interfere with their work to the extent that they would some-times be absent for a day or two or to the extent that they would sometimes disrupt their interpersonal relationships by alienating friends or acquaintances by exposing some symptoms or anxieties. It was intended that down to this level the former patients would now be functioning essentially as well as many so-called 'normal' people in the community. However, it will be noted that these criteria were obviously constructed mainly for seriously ill, hospitalized patients, who comprised the main group of patient subjects in the author's practice at the outset of this data collection.
Slight Improvement refers to patients who, following treatment, have experienced some relief from their symptoms or anxieties, but who are still subject to a considerable amount of discomfort or symptomatology, sufficient for them to continue to seek further help.
No Change refers to patients who have shown or reported slight or no change in their original symptoms. This term refers to the residual when those subjects rated in other groups are taken out.

These outcome ratings are admittedly extremely rough, and they have very little of an objective or replicable character about them. They were not even very consistently obtained since in some cases the ratings were based largely upon conference assessments by a group of psychiatrists, in many cases the patient's subjective report and brief interview with the patient and/or relatives was the main basis for the outcome rating, and in some cases even the subjective reports were quite sketchy. At the same time, an attempt was made to test the internal consistency of the ratings. The ratings were all made by the author at the time of termination of each subject's treatment program. When he began to collate the material, before referring to the originally scored outcome rating and with reference only to the patient subject's name, he repeated the rating of outcome. Some of the ratings had been made four years previously, and in many cases he had forgotten when the patient was treated or which method had been used with the patient. In every instance, he assigned the same outcome rating to the patient at both occasions with the single exception of one patient who had since been re-admitted to hospital, and who he therefore rated lower on the second occasion than on the first. Another somewhat doubtful support for the

admittedly poor outcome ratings is available from another source. The subgroup of thirty provincial hospital patients had been receiving care in the same provincial hospital for many years (average 9 years, range 2 to 45 years), where they had always returned after brief (no longer than 6 months) periods out in the community. They mostly felt rather "at home" in that hospital, and they tended to report a good sense of having been helped at that hospital from the behavioural treatments they received. It seems reasonable to believe, then, that these patients would return for further care or treatment if it was needed at that hospital. Consequently, even although it might be virtually impossible to locate most of the patients treated, a kind of follow-up might be obtained by determining the readmission rate within this sub-group to that hospital. Moreover, condition on re-admission would tend to confirm original outcome ratings, and since about equal numbers of patient subjects in this subgroup of patients were treated with the three treatment procedures being contrasted, the inter-group outcome ratings might in this way be confirmed. Of the thirty patients from that hospital who were treated, 4 showed no change. Of the remaining 26 patients, only 2 have been readmitted to a local hospital since they were treated, and one of these patients was readmitted only for a brief period of time. One of these patient subjects was initially rated Some Improved (later rated Slight Improvement) and had been treated with RIT, and the other of these patient subjects was rated Much Improved and had been treated with SCARS. Nevertheless, it must be stated that the weakest aspect of this report is the outcome ratings used.

## Results

Table 1 shows the numbers of patient subjects in each diagnostic category treated with each method, and the outcome ratings obtained within each category. This material shows that a fairly wide range of patient subject material was studied, and that outcome seems to be relatively randomly related to diagnostic category.

Insert Table 1 about here

Table 2 summarizes the main findings with this material. It shows the out-come ratings for subjects treated with the three methods employed, showing the number of subjects in each outcome category and the cumulative percentage in each outcome category for each treatment subgroup receiving their own or a better outcome rating. For example, whereas 67% of the RIT subjects obtained ratings of Some Improved or better, 85% of the SCARS subjects, and 99% (really 100%) of the Both subjects, obtained ratings of Some Improved or better. No tests of

Insert Table 2 about here

statistical significance were run on these data both because of the roughness of the outcome ratings and because, due to probable initial differences, between the groups of subjects treated with the three methods (the RIT group would seem to have been slightly more behaviourally disturbed than the SCARS group), it would be inappropriate to imply statistical significance of differences due to the treatments. However, it seems justifiable to conclude that SCARS did at least as well as RIT on a group basis.

Table 3 is included to illustrate one reason for lower outcome ratings with some patient subjects as compared with others. It is noteworthy that, particularly with SCARS, patient subjects showing less improvement also had fewer sessions per subject than subjects showing more improvement. One reason for the low average number of sessions in the poorer outcome groups with SCARS is due to the tendency of patients to terminate treatment early in some instances, due, according to their report, to the feeling that SCARS is not a treatment, but more like an investigative method. That is, SCARS does not yield a sense of any change early in the treatment, and some subjects report some dissatisfaction with their lack of personal involvement in the method (they often feel as though they have nothing to do). In addition to the above, it is interesting to note from Table 3 that in the three best outcome categories, an average of 22 sessions was required per subject with SCARS, while an average of about 33 sessions was required per subject for RIT. While we would like to argue that SCARS seems to have been able

Insert Table 3 about here

to yield a higher percentage of successful outcomes in fewer sessions than RIT was able to achieve, we do not feel justified in running significance tests or in suggesting such differences since the data are not strictly speaking comparable in terms of the subject groups treated. Consequently, we feel justified only in concluding that the present material suggests that SCARS is at least as effective a treatment procedure as RIT (which is used here as a control condition for comparison), in a fairly wide range of patient disorders.

## Discussion

In the foregoing, an attempt was made to argue some models for 'illness' in order to provide a basis on which it might be possible to predict the probable effectiveness of different behavioural therapies. A therapeutic method and a model for its applicability were derived, and it was argued that, with reference to the models for 'illness' posited, SCARS should have some therapeutic advantages over an apparently similar procedure, RIT. Some data were presented contrasting the effectiveness of SCARS against RIT, using RIT as a control condition which has already been shown elsewhere to have therapeutic efficacy. Although SCARS seemed on gross analysis to perform somewhat better than RIT in the subject groups (a wide range of types of patients) studied, it was noted that the SCARS and RIT subject groups were not strictly speaking comparable initially. Moreover, many of the types of subjects treated were inappropriate as subjects for treatment with RIT in terms of the types of patients with which RIT purports to be useful. However, it is interesting that RIT yielded relatively good results with many of these 'inappropriate' patients.

The real issue, however, is not the overall competitive efficiency of one procedure as compared with another equivalent procedure. RIT and SCARS are relatively different procedures. Although both procedures seek to desensitize anxiety reactions to stimuli, the stimulus materials involved and the level of analysis involved in the behaviour monitoring operations, and hence the training task itself, are relatively different. SCARS seems to represent another tool for use with patients and, as such tools develop, it would seem appropriate to investigate the relative efficiencies of the several tools in the several types of conditions psychologists are called upon to

treat.

It is certainly not clear yet what kinds of patient problems SCARS is particularly suited to treat. However, the present material makes it seem reasonable to hypothesize that (a) SCARS should yield better than no treatment results in many patient groups, since it is shown to be apparently as effective as RIT, which has already been shown elsewhere to have therapeutic efficacy, and (b) SCARS may have some special areas of application in treatment of patients' problems. Moreover, it seems possible that SCARS might turn out to be a relatively efficient procedure, considering the average number of treatment sessions required in the foregoing report to produce reasonably good results. Finally, a somewhat paradoxical statement can be made about SCARS. SCARS permits a fairly wide range of accessory methods to be used in an integrated manner with the basic procedure, and so permits the overall treatment technique to be tailored to a considerable degree to the individual needs of the particular subject. At the same time, SCARS permits sufficient automation and reduction of contact between the operator and the subject that one might be able to argue both that SCARS is a fully automated technique, and that SCARS permits 'relationship' to be kept to a minimum in psychotherapy.

References


Benton, R.G., and Mefferd, R.B., Projector slide changing and focusing as operant reinforcers, Journal of Experimental Analysis of Behaviour, 1967, 10, 479-484.

Fenichel, 0., Psychoanalytic Theory of the Neuroses, New York, W.W. Norton, 1954.

Fenz, W.D., and Epstein, S., Steepness of approach and avoidance gradients in humans as a function of experience: theory and experiment, Journal of Experimental Psychology, 1965, 70, 1-12.

Fenz, W.D., and Epstein, S., Gradients of physiological arousal in parachutists as a function of an approaching jump, Psychosomatic Medicine, 1967, 29, 33-51.

Fisher, S., and Cleveland, S.E., Body Image and Personality, New York, Van Nostrand, l958.

Freud, S., Inhibitions, Symptoms and Anxieties, New York, Appleton-Century-Crofts, 1926.

Gavalas, Rochelle J., Operant reinforcement of an autonomic response: two studies, Journal of Experimental Analysis of Behaviour, 1967, 10, 119-130.

Kimmel, H.D., and Hill, Frances A., Operant conditioning of the GSR, Psychological Reports, 1960, 7, 552-562.

Lang, P.J., and Lazovik, A.D., Experimental desensitization of a phobia, Journal of Abnormal and Social Psychology, 1963, 66, 519-525.

Lang, P.J., Lazovik, A.D., and Reynolds, D.J., Desensitization, Suggestibility, and Pseudotherapy, Journal of Abnormal Psychology, 1965, 70, 395-402.

Lazarus, A.A., Group therapy of phobic disorders by systematic desensitization, Journal of Abnormal and Social Psychology , 1961, 63, 504-510.

Mednick, S .A., A learning theory approach to research in schizophrenia, Psycho1ogical Bulletin,

l958, 55, 316-327.

Mednick, S.A., Schizophrenia and development, Paper presented at University of Toronto Colloquium, 1966.

Paul, G.L., Insight versus Desensitization in Psychotherapy, Stanford, Stanford University Press, 1966.

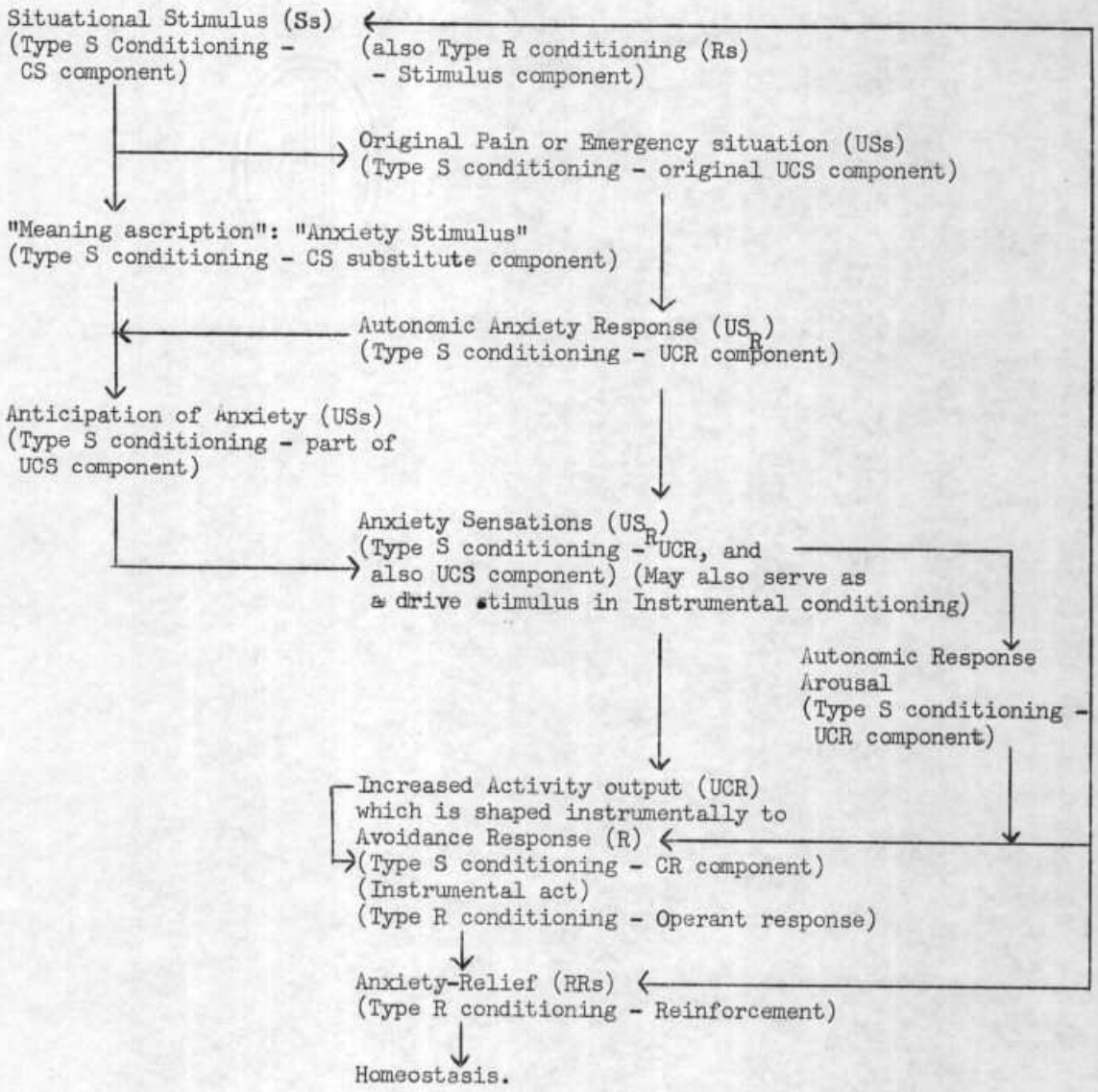
Rachman, S., The role of muscular relaxation in desensitization therapy, Behaviour Research and Therapy, 1968, 6, 159-166.

Stampfl, T.G., and Levis, D.J., Essentials of Implosive Therapy: a learning theory based psychodynamic behavioural therapy, Journal of Abnormal Psychology, 1967, 6, 496-503.

Van Tyyver, H.B., and Kimmel, H.D., Operant conditioning of the GSR with concomitant measurement of two somatic variables, Journal of Experimental Psychology, 1966, 72, 841-846.

Wolpe, J., Psychotherapy by Reciprocal Inhibition, Stanford, Stanford University Press, l958.

Illustration I: The Neurotic Chain



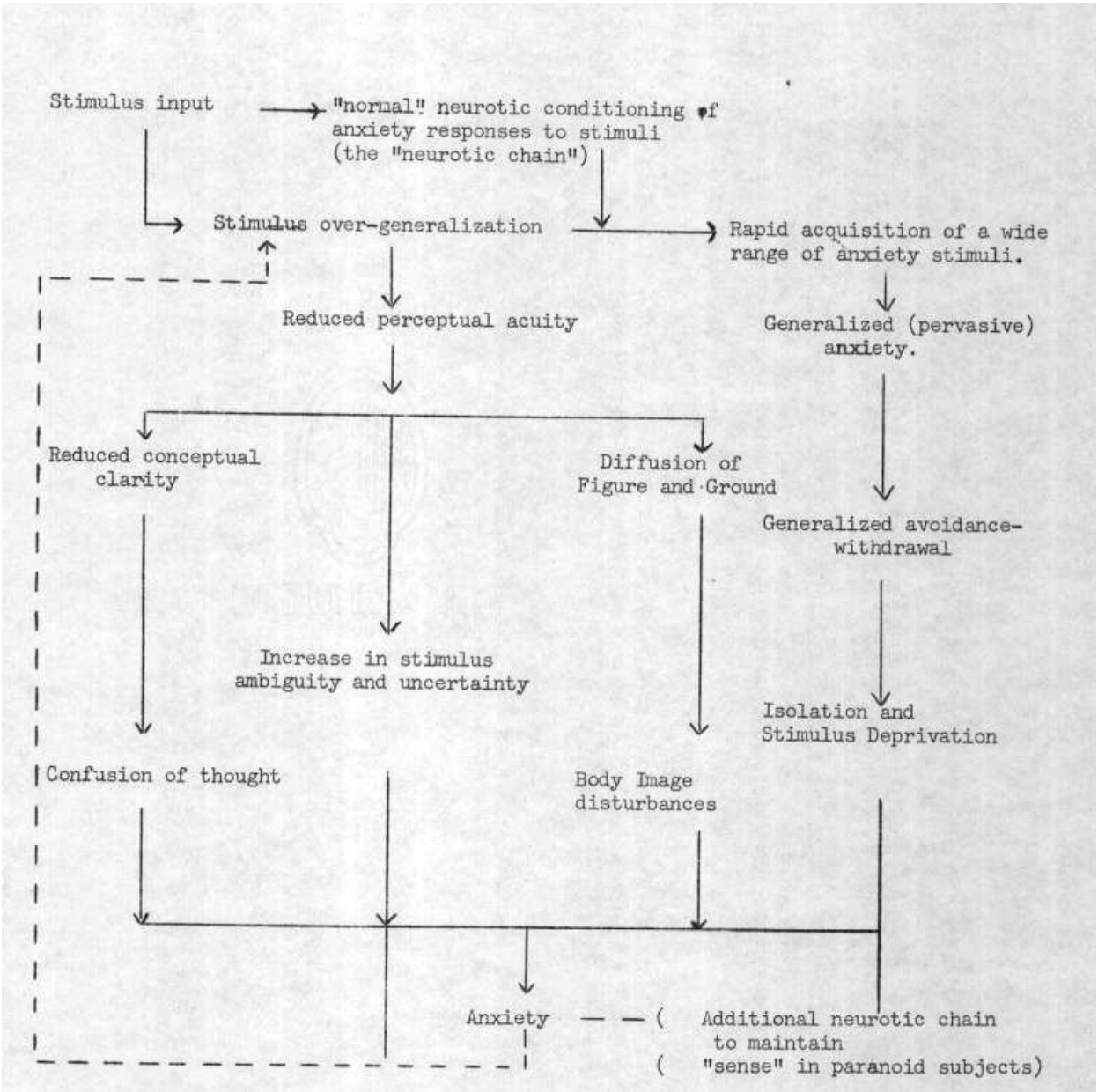



Illustration II: Schematic representation of some of the components of the psychotic chain.

TABLE 1

Types of Subjects treated by three types of Treatments, and Results obtained

| Subject Diagnoses (N) | RIT | | | | | SCARS | | | | | Both | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | R | M | So | Sl | NC | R | M | So | Sl | NC | R | M | So | Sl | NC |
| Catatonic Schizophrenia (5) | 0 | 0 | 0 | 1 | 1 | 0 | 2 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| Paranoid Schizophrenia (4) | 3 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Simple Schizophrenia (2) | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 0 |
| Manic-Depressive psychosis (5) | 1 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 0 | 0 |
| Borderline Schizophrenia (6) | 0 | 0 | 0 | 0 | 0 | 0 | 2 | 1 | 0 | 0 | 1 | 1 | 1 | 0 | 0 |
| Obsessive-Compulsive Neurosis (6) | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 0 |
| Obsessive Anxieties & Fears (6) | 1 | 1 | 0 | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Compulsive Depression (7) | 2 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 0 | 3 | 0 | 0 | 0 | 0 |
| Neurotic Depression (6) | 0 | 0 | 0 | 1 | 0 | 2 | 1 | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 |
| Post-traumatic Neuroses (11) | 1 | 4 | 3 | 1 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| Somatic Pain Neuroses (3) | 0 | 0 | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Hysteria (1) | 0 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Homosexuality (&Fear of) (8) | 0 | 2 | 1 | 1 | 0 | 1 | 2 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 |
| Sexual perversions (4) | 0 | 0 | 1 | 0 | 1 | 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Tic and Tremor (5) | 0 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 2 | 0 | 0 | 0 | 0 | 0 | 0 |
| Complex Phobias (6) | 0 | 0 | 0 | 0 | 0 | 1 | 2 | 2 | 0 | 0 | 0 | 1 | 0 | 0 | 0 |
| Mutiple Phobias (2) | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| Simple Phobias (10) | 0 | 0 | 0 | 3 | 0 | 3 | 3 | 0 | 1 | 0 | 0 | 0 | 0 | 0 | 0 |
| Total (97) | 10 | 9 | 8 | 9 | 4 | 14 | 15 | 5 | 5 | 1 | 6 | 6 | 5 | 0 | 0 |

Table 2

Cumulative Percentages of Success in three types of Treatments

| Outcome Category | Wolpe RIT | | SCARS | | Both | |
|---|---|---|---|---|---|---|
| | Number | Cumulative % | Number | Cumulative % | Number | Cumulative % |
| Recovered | 10 | 25 | 14 | 35 | 6 | 35 |
| Much Improved | 9 | 47 | 15 | 72 | 6 | 70 |
| Some Improved | 8 | 67 | 5 | 85 | 5 | 99 |
| Slight Improved | 9 | 90 | 5 | 97 | 0 | 100 |
| No Change | 4 | 100 | 1 | 100 | 0 | 100 |

Table 3

Number of Treatment Sessions (Average) per Subject per Treatment Type

| Outcome Category | RIT | SCARS | Both |
|---|---|---|---|
| | Sessions/Subject | Sessions/Subject | Sessions/Subject |
| Recovered | 41 sess./S | 22 sess./S | 89 sess./S |
| Much Improved | 35 sess./S | 23 sess./S | 88 sess./S |
| Some Improved | 24 sess./S | 19 sess./S | 70 sess./S |
| Slight Improved | 19 sess./S | 12 sess./S | 0 |
| No Change | 28 sess./S | 7 sess./S | 0 |

Table 4

Average number of years of Illness per Subject for each

Outcome category of each Type of Treatment employed.

| Outcome Category | RIT | SCARS | Both |
|---|---|---|---|
| | Years/Subject | Years/Subject | Years/Subject |
| Recovered | 9 years/S | 7 years/S | 9 years/S |
| Much Improved | 13 years/S | 8 years/S | 13 years/S |
| Some Improved | 6 years/S | 7 years/S | 16 years/S |
| Slight Improved | 7 years/S | 3 years/S | -------- |
| No Change | 12 years/S | 3 years/S | -------- |