

## BEHAVIOUR THERAPY, SPONTANEOUS REMISSION AND TRANSFERENCE IN NEUROTICS

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Any general theory of neurotic behaviour must attempt to account for the main phenomena in this field of psychology, and its acceptability must in part depend on its success in thus creating a "nomological network" within which otherwise isolated events can be ordered and understood. One of the most important, most universal, and most widely acknowledged of these phenomena is that of *spontaneous remission*; as is well documented in several research reports(1, 2, 14, 18) neurotics tend to get better without any form of specific psychiatric treatment. This improvement appears to be a function of time; Eysenck(5) has suggested the following formula as descriptive of the situation:

$$X=100(1-10^{-0.00435N})$$

where X stands for the amount of improvement achieved in percent and N for the number of weeks elapsed. He comments that "while the exact values in this formula should not be taken too seriously, its general form is of course that of the typical learning curve with which psychologists are familiar."

It is also well-known that psychotherapeutic treatment, whether psychoanalytic or eclectic, does not accelerate this rate of recovery(1, 3, 5, 6, 8, 14, 15, 20, 23). Under these circumstances it may be worthwhile to take a closer look at the phenomenon of spontaneous recovery from a theoretical point of view in order to determine possible causative factors; it is clearly impermissible to implicate "time" as such, because it can only be *events* happening in time which can exert a causal influence, and our formula does not tell us very much about the possible nature of these events. It is the purpose of this article to present a theory of "spontaneous remission"; this theory is derived from a general body of knowledge sometimes referred to as "learning theory"(11, 13). It also links up with a rational theory of diagnosis and treatment

in neurosis which has been called "behaviour therapy"(6, 8) and which purports to achieve results superior to those for which "spontaneous remission" can be held responsible(7, 20).

Before proceeding to a discussion of this theory, we may note with some surprise that what may be called the currently prevailing "orthodox" set of psychiatric hypotheses, which are closely identified with psychoanalytic and "dynamic" notions, have nothing to say about spontaneous remission; indeed, they seem to suggest that such remission cannot occur, or that, where it does, it can only be of very short-term duration. This follows directly from the Freudian notion that neurotic behaviour is motivated by some underlying complex or complexes, and that the treatment of the symptom without some form of "uncovering" of the underlying complexes must lead to a recrudescence of the same, or the appearance of some other symptom. The evidence is decisively opposed to this belief(21, 22) and it is notable that no adequate documentation has ever been put forward by psychoanalytic writers who seem entirely to rely on anecdotal evidence, on repetition of doctrinal pronouncements, and on uncontrolled studies incompletely presented. Such an important point, one might have imagined, should have been established a little more securely before being accepted and interpreted as ruling out of court *a fortiori* all nonpsychoanalytic methods of treatment. As the evidence stands now we may perhaps say that the failure of symptoms to recur after spontaneous remission, or after some form of behaviour therapy, is a decisive argument against the Freudian theory.

How does behaviour therapy deal with spontaneous remission? In order to answer this question we must first state the main tenets of the general theory, without however being able here to bring forward all the supporting evidence; this task has been attempted elsewhere(5, 16, 20). For con-

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venience, we may number the points in order. 1. Neurotic behaviour consists of maladaptive conditioned responses of the autonomic system, and of skeletal responses made to reduce the conditioned (sympathetic) reactions. 2. While the term "symptom" may be retained to describe neurotic behaviour, there is no implication that such behaviour is "symptomatic" of anything. 3. It follows that there is no underlying complex or other "dynamic" cause which is responsible for the maladaptive behaviour; all we have to deal with in neurosis is conditioned maladaptive behaviour. 4. Treatment consists of the *deconditioning*, by reciprocal inhibition, extinction, conditioned inhibition, or in some other way, of the maladaptive behaviour, and the *conditioning*, along orthodox lines, of adaptive behaviour. 5. The treatment is a-historical and does not involve any "uncovering" of past events. 6. Conditioning and deconditioning will usually proceed through behavioural channels, but there is no reason why verbal methods should not also be used; there is good evidence that words are conditioned stimuli which have an ascertainable position on the stimulus and response generalization gradients of the patients(19).

Consider now a typical case history involving the establishment and cure of a cat phobia(9). A traumatic event involving the patient's favourite cat produces a conditioned fear of cats; this develops to such an extent that she is effectively home-bound for many years, refusing to go out for fear of encountering cats. Treatment is by means of graduated presentations of cats (first symbolically, *i.e.*, by words and pictures, then bodily, but at a distance, *etc.*) under conditions of relaxation and parasympathetic stimulation (desensitization, reciprocal inhibition). After a few weeks treatment is completely successful, and a permanent cure achieved (no relapse for several years). In this case history there is no spontaneous remission, and we may enquire 1) why such a remission might have occurred, and 2) why in fact it did not do so.

First, we have a traumatic event which, by means of classical conditioning, produces a conditioned fear reaction to a

previously neutral set of objects, *i.e.*, cats. It is easy to see how this conditioned fear arose, but it is not so easy to see just why it should have persevered so long. Solomon and Wynne(17), on the basis of their work with dogs, have offered the principle of "partial irreversibility" in avoidance conditioning, but it should be noted that the aversive stimuli in their case were probably stronger than in the case of the patient, and also that they report no single-trial learning, as seems to have occurred in this patient. On general learning-theory principles one would have expected the gradual *extinction* of the conditioned fear response in the course of time. Each time the patient saw a cat (the CS), without a recurrence of the traumatic events which precipitated her original fear (the UCS), this unreinforced presentation of the CS should lead to an increment of inhibition potential leading to extinction. Similarly, each time she discussed her troubles with a sympathetic listener this should have had an effect similar to that of "reciprocal inhibition," also leading to extinction of the fear response. In other words, behaviour theory seems to have no difficulty in explaining the extinction of neurotic symptoms by "spontaneous remission"; this extinction is the natural result of the inevitable recurrence of the CS in the absence of reinforcement. We may thus reinterpret our formula for the time course of spontaneous remission by saying, not that it resembles the typical learning curve, but rather that it resembles (and indeed is nothing but) the typical extinction curve. Our hypothesis, then, is that *all neurotic symptoms are subject to extinction*, and that this process of extinction is reflected in observable behaviour in the form of "spontaneous remission." The theory would appear to fit the facts reasonably well, but it would also appear to assert too much; not all cases of neurosis do in fact remit, and a theory predicting universal remission is clearly in need of an extension.

Such an extension is indeed implied in the first of our numbered postulates of behaviour therapy, given above, in which attention was drawn to the importance of "skeletal responses made to reduce the conditioned (sympathetic) reaction." What is asserted here is that in many cases of

neurosis the original stage of classical conditioning is followed by a stage of instrumental conditioning, and that it is this secondary development which makes impossible the process of extinction by removing the conditions of its occurrence, *i.e.*, the presentation of the CS under conditions of non-reinforcement. Consider the events in the laboratory during the extinction of a conditioned response. The dog, lashed to his stand, is presented with the CS a number of times; his conditioned responses get weaker and weaker until finally they cease altogether. This paradigm differs profoundly from that of our patient encountering a cat in the street after her conditioned fear has been established. The patient is not lashed to a stand, and thus forced to witness the conjunction: CS—non-reinforcement; she is free to turn her back and run away. This course of conduct produces an entirely different paradigm, one favourable to the growth of an instrumental response of running away from cats. Simplifying the situation grossly, we may say that what happens is something like this. The patient approaches the cat and experiences a conditioned sympathetic response (fear) which is profoundly disturbing and (negatively) reinforcing. She turns and runs, thus excluding the cat from her field of vision, and also increasing the distance between herself and the feared object. This behaviour reduces the sympathetic arousal, and is thus reinforced by the resulting lessening of fear. The next time the patient encounters a cat, the newly acquired habit of running away will again, and more easily, be brought into play, until finally an instrumental conditioned response of running away is developed to such an extent that it permanently excludes the possibility of encountering the CS at all. In this way the secondary process of instrumental conditioning “preserves” the primary conditioned response; putting the whole matter into psychiatric terminology, instrumental conditioning makes impossible the “reality testing” of the classically conditioned response.

There is no doubt, of course, that in most cases the situation is much more complex than this. The original conditioning is not always, and perhaps not even usually, a traumatic, single trial event; repeated sub-

traumatic trials may produce an even stronger conditioned fear response than a single traumatic event. Little is known about the precise dynamics of this process in individual cases, largely because psychiatric attention has not usually been directed at these events from the point of view of learning theory. Again, few neuroses are mono-symptomatic, and there may be a very complex interweaving of several different habit-family hierarchies (12, 20), each subject to extinction at different rates, and by exposure to different events (CS's). Lastly, experience indicates, and theory suggests, that extinction of conditioned fear responses in one habit-family hierarchy facilitates (through a process of generalization) extinction in others, whether this extinction is occurring during “spontaneous remission” or during behaviour therapy. To mention these complications, to which many others could have been added, is simply to remind the reader that while in principle the explanation of spontaneous remission here given is perhaps correct, nevertheless much experimental and observational work remains to be done before the details of the process can be said to be at all well understood.

It is interesting to note that several observationally well attested phenomena can be brought into this theoretical framework. Consider the pilot who has crashed his plane, or the cowboy who has been thrown by his horse. It has often been stated that if the pilot, or the cowboy, is allowed to walk away from the plane, or the horse, he will never fly, or ride, again. If, however, he makes himself fly or ride again immediately, then there will be no such disastrous after effect. We may regard the original event as productive of a conditioned fear reaction to planes or horses; this by itself would not be strong enough to preclude future resumption of the particular activity which produced the traumatic event. However, bodily removal from the now feared object produces instrumental conditioning, along the lines indicated above, and it is this additional process of avoidance conditioning which, when superadded to the original classical conditioning, makes the total aversive forces too great to be overcome.

Much the same explanation could be given to the well-known fact that psychiatric casualties during the war tend to go back to combat easily and readily if treated in front-line conditions, but hardly ever if sent back to base hospitals first and then treated. Here also the part played by classical conditioning is fundamental, but can be counteracted by a process of extinction in the front-line situation; if instrumental conditioning is allowed to add its share, *i.e.*, through removal of the patient to a base-line hospital, prognosis is poor because now *extinction is made almost impossible!* Other applications of this general theory will easily occur to the reader.

This may be an appropriate place to consider another event which is frequently claimed to be an almost invariable concomitant of the therapeutic process, namely *transference*. Here there is indeed a psychoanalytical theory to explain the phenomena alleged to occur, namely, the development of certain strong emotional feelings on the part of the patient for the analyst (and perhaps vice versa). This theory depends on the *transfer* of certain childhood emotions originally attached to the parents; these, it is suggested, are transferred to the analyst. Now there is little doubt that such emotional dependence does in fact occur, although there is very little well-established evidence to suggest just how frequent, how strong and how lasting such emotions are. Indeed, similar facts have been known to occur in the Catholic confession for many hundreds of years, and the priest taking the confessional is taught how to deal with these feelings. How does behaviour theory account for the facts?

In the first place, it is important to dissociate  $T_F$  (the facts conveniently summarized under the heading "transference" and  $T_T$  (the psychoanalytic theory of literal "transference"). The writer would hold that  $T_F$  is a real phenomenon requiring an explanation, but that  $T_T$  is a speculative theory without any sound experimental background. It is unfortunate that the name for  $T_F$  immediately suggests the truth of  $T_T$ ; it might be better if a more neutral name were to be chosen. In any case, it will be clear from what has been said that when it is stated that behaviour therapists

reject the notion of transference, what is meant is a rejection of the speculative theory, and not of the facts themselves.

As for an alternative theory, consider the position of the therapist in his relation to the patient. Whether because of spontaneous remission, or because of the reciprocal inhibition produced by the permissive attitude of the therapist, there is a tendency for the patient to improve. Consider the therapist as a CS in this situation, consider the unknown cause of the improvement as the UCS, and consider the improvement and its attendant emotions and feelings as the response. It will be clear that there will be a tendency for the therapist to be credited with the properties of the UCS, through a process of classical conditioning, and that attitudes and emotions appropriate to the latter are shifted to the former. A well-known example is given by Pavlov, who reports that when an electric light was used as the CS for a feeding-salivation experiment, the dog after a while licked the light bulb! In other words, there is a transfer of reactions appropriate to the UCS to the CS. As an example of such a transfer in a human subject, consider Connie, a 5-year-old girl being treated for enuresis by means of the well-known bell-and-blanket method (10). When the first signs of a cure became noticeable after 4 applications, she spontaneously kissed and hugged the red light on the apparatus which illuminated the switch activating the bell, saying "The ting-a-ling is my best friend." No doubt it would seem almost sacrilegious to many psychiatrists to consider this analogous to full-blown "transference," but the fundamental identity or lack of identity of the processes involved must be established on a more experimental basis than mere shocked disbelief. The explanation here given accounts for the facts as well as does the Freudian, and in addition it is based on well-documented laboratory experiments; nevertheless it would be most desirable to submit it to direct experimental investigation before regarding it as anything but an hypothesis.

This paper has been kept short on purpose, being purely theoretical in the first place, and lacking direct experimental support in the second. It would be idle guess-

work to extend speculation beyond the points raised, although promising extensions do suggest themselves in considerable number. Its primary purpose, however, will have been served if it reawakens interest in the phenomena of spontaneous remission and transference, and leads to more experimental investigations of these interesting and perhaps even crucial events in the life-history of the neurotic. The formulation of an explanation in terms of learning theory here given is not the only one possible, and it may not be the one preferred by other psychologists; it may nevertheless repay investigation. But primarily it is hoped that the reconsideration of these phenomena will serve to raise doubts about the adequacy of that "premature crystallization of spurious orthodoxy" which is present-day psychoanalytic theory.

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