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SINGLE-TRIAL CONDITIONING, NEUROSIS, AND THE NAPALKOV PHENOMENON

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THE BELIEF that many if not all phobic and anxiety reactions of a neurotic kind are due to conditioning, either through single-trial traumatic or many-trial sub-traumatic learning, forms the basis of behaviour therapy, and there is much evidence to support it (Eysenck and Rachman, 1965). Similarly, spontaneous remission of such reactions is often attributed to extinction; repeated exposure to the conditioned stimulus without reinforcement should, according to the precepts of modern learning theory, lead to extinction (Eysenck, 1964). There are certain facts which pose difficulties in relation to both these general statements. Occasionally phobic patients are found in which the original traumatic event is not immediately followed by a strong conditioned fear of the CS, but where this fear seems to grow in time, so that exposure to the unreinforced CS does not seem to lead to extinction, but rather to an increase in the severity of the conditioned response. Again not all patients show spontaneous remission; a fair proportion either remain ill or even get worse with time, in spite of the fact that no further reinforcement (pairing of CS and UCS) occurs. It is possible to account for some of these facts in terms of operant conditioning superimposed upon classical conditioning (Eysenck, 1964), i.e. voluntary avoidance of the CS is reinforced through lessening of autonomic reactions, but it may be doubtful if this mechanism can explain all the relevant facts. It is the purpose of this paper to draw attention to some laboratory experiments reported by A. V. Napalkov (1963); these are concerned with a phenomenon which may be very relevant to the problems just mentioned.

Working with dogs, Napalkov inflicted various nocive stimuli on these animals (electric shocks, flashes of light, firing of toy pistols, etc.); the result was an increase in level of blood pressure of between 30 and 50 mm, lasting for 10–15 min. After twenty to thirty such applications, no rise in blood pressure was observed; in other words, adaptation of the dogs to the physical stimuli was complete. No such adaptation was found, however, in another type of experiment, in which the nocive stimulus was only administered *once*, preceded by a conditioned stimulus. The conditioned stimuli were then applied repeatedly, at intervals of 3–5 min, without any reinforcement. "The first application of a conditioned stimulus resulted in a 30–40 mm rise in blood pressure in all five dogs. After that the intensity of the reaction to the application of the stimulus continued to grow. The sixth and seventh applications brought about 50–60 mm increase in the maximal blood pressure. This process was going on during the subsequent days as well, and soon the blood pressure rose to 190–230 mm. But even this was not the limit;

the hypertensive state of the dogs showed a further increase . . . The hypertensive state persisted in the dogs for many months (the dogs were observed for a period of 1 yr and 4 months). The blood pressure did not fall even after the dogs had been excluded from the experiments for a period of 5 months (during this period they were kept in the kennel)."

Napalkov explains these phenomena in terms of an ever-widening system of stimulus generalization. "An analysis of the factors which caused the development of this pathological state showed that it was due to the formation of new pathological programmes of the level of blood pressure. These control programmes proved to include a considerable number of external signals which provoke a protracted and stable rise of the blood pressure. Many of these signals were present in the surrounding medium for a long time, and this resulted in the stability of the pathological level." While it is of course possible that extensive stimulus generalization took place under the conditions of this experiment, the question remains why such higher-order conditioning should have taken place at all, instead of extinction; in the laboratory, higher order conditioning is usually difficult to achieve, and extinction is quick. Napalkov's results may have some relation with Solomon and Wynne's (1954) principle of partial irreversibility, but they seem to go well beyond anything previously reported in the literature. Napalkov appeals to cybernetics for an explanation, but it cannot be said that his account is very enlightening; an explanation must be found in psychological or physiological language for the very important phenomenon he discusses. Unfortunately, no such explanation seems in sight, and in any case Napalkov's account is not systematic enough to make it easy for the reader to try out different hypotheses against the facts.

It may be possible to account for the facts of the case along the following lines. The CS is followed by a nocive stimulus, which gives rise to an autonomic UCR. When the CS is next produced (under conditions of no reinforcement) it gives rise to a CR which is perceived as nocive in its turn, so that now we have CS reinforced by CR_1. This reinforcement is much weaker than that given by the UCS but it obeys the same laws as any reinforcement, and keeps alive the link between CS and UCR. Furthermore, repetition of the CS–CR association should occur each time the CS is exposed, and the strength of the CR should increase, as argued in the case of a somewhat parallel phenomenon by Wolpe (1958, pp. 63–64). Wolpe made use of an argument of this kind in order to explain "the learning process in neuroses produced by mild shock"; it seems possible that the Napalkov phenomenon also might be explained along these lines. If such an explanation is anywhere near the right lines, then we would expect to be faced with certain quantitive questions crying out for an answer. Repeated presentation of CS and UCS, even under conditions of reinforcement, produces inhibition ("inhibition with reinforcement"); similarly presentation of CS, even when followed by CR as a reinforcer, is likely to accumulate inhibition. There must be a very definite dividing line between cases where inhibition wins out, and the CR is too weak to persist, and cases where CR is strong enough to overcome this inhibition, and grow in strength sufficiently quickly to set up a very persistent system of CS–CR links, leading to the stimulus generalization observed by Napalkov.

It is not argued that this account is necessarily true; it is merely suggested that the simple rule of "no reinforcement→extinction" does not always hold in relation to the growth of autonomic fear reactions, and that this fact may have a very important bearing on the origin and perpetuation of neurotic disorders. A direct experimental attack on this problem would seem overdue, guided perhaps by some rather vague hypotheses as

have been suggested here. Additional points worth investigating relate to the influence of innate differences in emotionality; all writers from Pavlov to Gantt and Liddell (both of whom have published investigations not dissimilar in outcome to Napalkov) insist on the importance of individual differences in this respect.

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