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Unpredictable and Uncontrollable Events: A New Perspective on Experimental Neurosis

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Recent work has shown that unpredictable and/or uncontrollable events can produce a variety of cognitive, affective, and somatic disturbances to the organism. These disturbances are compared to and found to be quite similar to the symptoms of the classic cases of experimental neurosis described by Pavlov, Gantt, Liddell, Masserman, and Wolpe. The hypothesis is then developed that the common thread running through the entire experimental neurosis literature is that in each case important life events become unpredictable or uncontrollable, or both. This interpretation is contrasted with the earlier physiological, psychodynamic, and behavioral interpretations made by the investigators themselves. The implications of this analysis of experimental neurosis for various issues in the predictability–controllability literature are discussed—for example, the interaction between unpredictability and uncontrollability, the "threshold" for response to lack of predictability or controllability, and the lack versus the loss of predictability and controllability. Finally, the possible clinical relevance of this new perspective on experimental neurosis is discussed.

In 1927 I. P. Pavlov reported an experiment by Shenger-Krestovnikova in which a hungry dog was placed in a harness for what was intended to be a straightforward salivary conditioning experiment. When a circle was presented, meat powder was forthcoming; when an ellipse was shown, the food was withheld. Everything went as usual, and the dog acquired the discrimination rapidly. Then the animal was required to make increasingly fine discriminations, at which he succeeded until the ratio of the semi-axes on the ellipse reached 9:8. Pavlov (1927) described what followed:

> After three weeks of work on this differentiation not only did the discrimination fail to improve, but it became considerably worse, and finally disappeared altogether. At the same time the whole behavior of the animal underwent an abrupt change. The hitherto quiet dog began to squeal in its stand, kept wriggling about, tore off with its teeth the apparatus for mechanical stimulation of the skin, and bit through the tubes connecting the animal's room with the observer, a behavior which had never happened before. On being taken into the experimental room the dog now barked violently, which was also contrary to its usual custom; in short it presented all the symptoms of a condition of acute neurosis. On testing the cruder differentiations they were also found to be destroyed. . . . A fresh development of the latter differentiation up to its previous exactness progressed twice as slowly as at first, but during the re-establishment of this crude differentiation the animal gradually became quieter, returning finally to its normal state. . . . The 9:8 ellipse at its first application was com-

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pletely discriminated from the circle, but from the second application onwards no trace of a discrimination was obtained, and the animal again entered a state of extreme general excitation with the same results as before. (p. 291)

Since Pavlov's classic report, many other psychologists have produced similar "neuroses of the experiment" by means of a variety of procedures. These have included difficult discriminations, increasing delays before reinforcement of a conditioned response, rapid alternation of excitatory and inhibitory conditioned stimuli, increasing numbers of aversive conditioning trials in a day, long periods of restraint and monotony, punishment of an appetitive response, punishment of mistakes on insoluble problems, excessive numbers of unpredictable shocks, excessive numbers of uncontrollable shocks, to name a few. The number of different procedures employed in these studies has almost been matched by the number of theories that have been elaborated to explain the observed phenomena (e.g., Masserman, 1943; Pavlov, 1927; Wolpe, 1958). In part, these interpretive differences may be a function of the sheer variety of procedures used to produce experimental neuroses. In addition, they mirror the historical conflicts between physiological, psychodynamic, and behavioral viewpoints in psychology as a whole.

When one considers the range of procedures that have been used to produce "experimental neurosis," it is somewhat surprising that they have all been classified under one common label. There is in fact no commonly accepted empirical definition of *experimental neurosis,* and yet there do seem to be several general characteristics that all cases have in common. First, some previously learned or adaptive behavior is no longer performed in the test situation as a result of some procedural manipulation, intended or not, made by the experimenter. Second, the behavior that emerges in place of the previously learned or adaptive behavior is generally uncharacteristic of the animal's ordinary behavior and usually shows signs of excessive autonomic arousal.

Despite the fact that these various procedures all produce somewhat similar behavioral disturbances, it has proved extremely difficult to abstract any single common denominator (or even a few) that could serve as a unifying explanatory principle. In one prominent attempt to deal comprehensively with these phenomena, Broadhurst (1961) invoked the Yerkes-Dodson Law, asserting that all the procedures increased motivation beyond optimal drive level, with resulting poor task performance and the release of fear responses. However, in a later review, Broadhurst (1973) retreated from this position and suggested that "the search for the single solution [be] abandoned" (p. 745).

We, however, do not feel that such a search is futile. On the contrary, we believe that a common thread does run through all these experiments. In each case, environmental events of vital importance to the organism become unpredictable, uncontrollable, or both. In this article, we develop this point of view and document it as well as is possible in a necessarily post hoc fashion, by reviewing the classic studies of experimental neurosis.[1] In doing so, we wish to emphasize that the importance of understanding the experimental neurosis literature does not necessarily stem from any very compelling phenotypic or topographic similarities bewteen the disturbances observed by, say, Pavlov and those seen in the psychological clinic. Rather, the relevance of the experimental neurosis literature today derives from the fact that the apparent *sources* or *causes* of experimental neurosis seem highly similar or even identical to certain factors that are also generally considered to be important in human psychopathology.

Predictability and Controllability

Stimuli impinging on an organism may be stressful and produce debilitating emotional, somatic, or cognitive effects because they are intense, frequent, or of long duration or because they produce conflict between approach and avoidance motives. However, even stimuli that do not possess these properties can be stressful or aversive if they are unpredictable or uncontrollable, or both. Over the

[1] Others (e.g., Maier & Seligman, 1976; Seligman, 1975) have made a similar argument, but their accounts are brief and highly selective.

past 10 years, the elaboration of the concepts of predictability and controllability has been a central topic in the psychology of learning and motivation, and this in turn has greatly expanded our understanding of fear, anxiety, stress, and depression (see particularly Seligman, 1975; Seligman, Maier, & Solomon, 1971).

Predictability

Consider two hungry rats bar pressing for food in Skinner boxes who each receive three moderately intense electric shocks over the course of their 2-hour bar-pressing session. One rat consistently receives a 1-minute light preceding each of his shocks. For the second rat, the light is also presented but does not reliably predict the shock. During the first few days of this procedure, both rats show a great deal of generalized suppression of bar pressing. After several days, however, the first rat manifests a typical conditioned emotional response (CER) by suppressing bar pressing only during the light. The second rat, by contrast, continues to show a complete disruption of bar pressing throughout the entire 2-hour session. Apparently his CER is to the experimental situation as a whole. Furthermore, when the rats are sacrificed after 45 days of such conditioning, the second rat shows extensive stomach ulceration but the first rat does not. These are the essential results of an experiment by Seligman (1968) that highlight the importance of the predictability of aversive events.

In any conditioning experiment, two parameters can be varied independently: both $p(\text{UCS/CS})$ and $p(\text{UCS/}\overline{\text{CS}})$ can vary from 0 to 1, where p = probability, UCS = unconditioned stimulus, CS = conditioned stimulus, and $\overline{\text{CS}}$ = the absence of CS. In a traditional continuous reinforcement, classical conditioning procedure, $p(\text{UCS/CS}) = 1$ and $p(\text{UCS/}\overline{\text{CS}}) = 0$. In the past, learning theorists devoted some attention to the first of these two parameters in their studies of partial reinforcement in classical conditioning: They varied $p(\text{UCS/CS})$ but left $p(\text{UCS/}\overline{\text{CS}})$ equal to 0. Only recently has much attention been devoted to the second parameter, as when $0 < p(\text{UCS/}\overline{\text{CS}}) < 1$ (i.e., when the UCS is not reliably predicted). Because the value of these two parameters can be varied independently from 0 to 1, as the values of the two probabilities approach each other, the environment becomes increasingly unpredictable. In the limiting case, $p(\text{UCS/CS}) = p(\text{UCS/}\overline{\text{CS}})$, and the CSs give the organism no information about the occurrence of the UCS. That is, the CSs and UCSs occur randomly with respect to one another (Seligman et al., 1971).

A large body of literature now shows that animals and humans prefer predictable to unpredictable aversive events (Badia, Suter, & Lewis, 1967; Lockard, 1963, 1965; Pervin, 1963) and that they may prefer signalled to unsignalled positive events as well (Cantor & LoLordo, 1970; Prokasy, 1956). In a similar vein, animals and humans seem to prefer immediate over delayed shock (Badia et al., 1967; Knapp, Kause, & Perkins, 1959). In addition, unsignalled aversive events appear to be more stressful than signalled ones (e.g., Badia & Culbertson, 1972). Seligman (1968) and Weiss (1970) both reported extensive stomach ulceration in rats exposed to unpredictable shocks; significantly less ulceration was observed in rats who received the same amount and intensity of predictable shock. MacKintosh (1973) has further reported that experience with random presentations of CSs and UCSs retards the acquisition of a conditioned response (CR) at a later time, when these stimuli are paired. He called this "learned irrelevance." Thus, unpredictable events have profound emotional, somatic, and cognitive effects on the organism.

Controllability

Now consider two dogs, each in a Pavlovian harness, with shock electrodes attached to their feet and panels next to their heads. When the shock is turned on, one dog can terminate the shock by pressing the panel with his head; for the second dog, panel pressing is ineffective in terminating the shock. The two dogs are yoked together so that both receive exactly the same amount of shock at the same time; the only difference lies in the

controllability of the shock. The following day each dog is placed in a shuttle box and given shock avoidance training. The first dog learns the avoidance response quickly. The second dog, by contrast, does not learn the avoidance response and rarely even escapes the shock. When the shock comes on, he lies down on the grid and seems to accept the shock passively. On those occasions when he does make a response, he does not appear to make the association between shuttling and the termination of the shock because he continues to accept the shock on future trials (Overmier & Seligman, 1967; Seligman & Maier, 1967).

As is the case with predictability in classical conditioning, two parameters can be varied independently in an instrumental conditioning situation: p(reinforcement/response) and p(reinforcement/$\overline{\text{response}}$). Again these probabilities vary from 0 to 1. In a simple continuous reinforcement paradigm, p(reinforcement/response) = 1 and p(reinforcement/$\overline{\text{response}}$) = 0. Learning theorists have extensively studied partial reinforcement schedules, in which the first parameter is varied while the second is held constant at zero. Experiments such as those of Overmier and Seligman (1967) and Seligman and Maier (1967) first drew attention to the importance of the second parameter. In particular, when p(reinforcement/response) = p(reinforcement/$\overline{\text{response}}$), we can say that reinforcement is independent of responding, that is, the organism has no control over the reinforcement (Seligman et al., 1971).

There is an extensive literature on the effects of uncontrollable aversive and appetitive events in humans and animals. Subjectively, humans rate controllable noise as less aversive than uncontrollable noise of equal duration and intensity. Controllable noise is also less disruptive to ongoing behavior (Glass & Singer, 1972). In terms of physiological stress effects, Weiss (1971) reported that rats who received controllable shocks showed substantially less stomach ulceration than their yoked partners, who had received the same amount of shock but had not had control over shock termination. In addition to direct stress effects, the behavioral sequelae of exposure to uncontrollable aversive events are numerous and wide ranging. In animals, new associations between responses and reinforcements (same or different) are acquired slowly (e.g., Goodkin, 1976; Maier & Seligman, 1976); in humans, problem-solving tasks, such as anagrams, are performed less efficiently (Hiroto & Seligman, 1975). In general, the subjects of these treatments become more passive. For example, animals have been shown to respond more slowly, to display less shock-elicited aggression, and to lose interest in sex and food (the latter also resulting in weight loss and loss of position in the dominance hierarchy) (Maier, Anderson, & Lieberman, 1972; Miller & Weiss, 1969; Seligman, 1975). Thus, exposure to uncontrollable aversive events can create a variety of profound affective, cognitive, and physiological disturbances for the organism.

Although the literature on the effects of uncontrollable *appetitive* events is much less extensive than that on aversive events, there do appear to be parallel cognitive and motivational deficits produced by exposure to such events. Welker (1976) and Wheatley, Welker, and Miles (1977) have found that pigeons and rats given exposure to response-independent food were later retarded at learning to make a response to deliver the food to themselves. Goodkin (1976) also found that rats given pretreatment with inescapable shock or free food were later retarded at acquisition of a shuttle box escape response. And in humans, Kurlander, Miller, and Seligman (cited in Seligman, 1975) found that exposure to insoluble discrimination problems (hence uncontrollable reward) resulted in a reduced level of competitiveness when the subjects were later required to play a Prisoner's Dilemma Game.

Overlap Between Predictability and Controllability

It is, of course, logically impossible to manipulate the factors of controllability and predictability completely independently. Although predictable events are not necessarily controllable (as in a classical conditioning paradigm), controllable events necessarily involve a certain amount of predictability, at

least over stimulus offset. A number of theorists (e.g., Averill, 1973) have asserted that it is this element of predictability inherent in controllability that is the critical element accounting for the importance of control. Others (e.g., Seligman, 1975) have argued that control is important for additional reasons having to do with the sense of mastery or competence that it teaches the organism. At present this issue remains unresolved (see Miller, in press, for the most recent review of this issue).

Despite the difficulty in making clear-cut interpretations of what effects are due to which factor, it is apparent from the preceding brief review that the unpredictability and/or uncontrollability of aversive events can have many deleterious consequences on affect and cognition as evidenced by behavioral and somatic changes in the organism. Similar, although perhaps less pronounced, cognitive and motivational deficits also appear to follow experience with uncontrollable and/or unpredictable appetitive events, although the relevant literature on this topic is sparse. In sum then, the lines of evidence just reviewed converge on the proposition that it is important for the organism that certain events (e.g., acquisition of food, escape from pain) occur in a predictable and/or controllable manner.

Symptoms of Experimental Neurosis

Thomas and Dewald (1977) recently compared the symptoms that follow exposure to an experimental neurosis paradigm with those following exposure to uncontrollable shock. Using a variant of the Shenger-Krestovnikova procedure in cats, Thomas found that as the discrimination became increasingly difficult, his cats ceased to initiate their own Pavlovian conditioning trials. Furthermore, their behavior became highly agitated and included "sudden vigorous outbursts of escape and aggressive behaviors." This agitated state was followed several days later by a stage of depression and lethargy. Virtually identical behavioral disturbances resulted from exposure to uncontrollable electric shock. During the first few inescapable shocks, the cats struggled and were highly agitated. Soon, however, the agitation disappeared and the animals displayed virtually no reaction to the shock. This "striking lack of affect or overt signs of fear" (p. 224) carried over into the test session the next day. So "both syndromes appear to follow a similar course—a period of intense agitation followed by a stage of lethargy and depression" (p. 225). Furthermore, similar loci in the brain seem to be implicated in the two syndromes, and rewarding brain stimulation can be used to restore normal behavior for both.

No other direct comparisons have been made between the symptoms of experimental neurosis and those created in situations of unpredictability or uncontrollability. Nevertheless, the cognitive and motivational deficits and the affective and somatic disturbances that pervaded the predictability–controllability literature also stood out in our review of the experimental neurosis literature. First, we noted the frequency with which the investigators of experimental neurosis stated that their animals seemed to have lost their ability or motivation to perform even the simplest of learning tasks. Such cognitive and/or motivational deficits parallel those noted above in our discussion of predictability and controllability. Second, we noted that experimentally neurotic animals generally showed affective and somatic changes in one of two directions. Some became very agitated, with increases in general activity level and signs of high autonomic arousal (increased breathing rate, piloerection, struggling, howling, etc.). Others showed decreased activity levels and generally looked passive and withdrawn, sometimes becoming socially isolated from their conspecifics. Some animals passed through both of these stages at different times. Nearly all animals showed feeding disturbances, at least in the experimental situation. These stages appear to resemble, respectively, the state of chronic fear or "anxiety" and the state of passivity and "depression" described above as occurring in situations of unpredictability or uncontrollability (Seligman, 1975).

The experimental neurosis literature and the predictability–controllability literature probably represent the two most salient examples of gross behavioral and physiological disturbance ever produced by purely behav-

ioral manipulations. Although direct comparison of the kinds of disturbances created is impossible to perform retrospectively, many compelling similarities do appear to exist (see especially Thomas & Dewald, 1977). The natural question to follow this observation, then, is: Have similar factors *caused* the similar disturbances described in the experimental neurosis and the predictability–controllability literature? In the next section we explore this possibility by analyzing the classic demonstrations of experimental neurosis. In each case we argue that the behavioral disruptions observed can be traced to the inability of the subject to predict and/or control certain events of importance to him.

Reanalysis of Classic Demonstrations of Experimental Neurosis

We now turn to those studies of experimental neurosis that have gained special prominence in the literature. We restrict our attention to the studies of Pavlov, Lidell, Gantt, Masserman, and Wolpe because all other studies of experimental neurosis have involved minor variations on their procedures. Within this domain, however, we aim to be comprehensive. We recognize that our analysis is post hoc because none of the experiments was designed with our hypothesis in mind. In fact, with the exception of Masserman and Wolpe, most investigators were content merely to demonstrate the experimental neurosis phenomenon. By and large they did not proceed with the next step, a rigorous experimental analysis, and hence our job of interpretation is all the more difficult. We consider our account valid to the extent that it can be compellingly applied to each individual study. At various points we offer suggestions about how a more rigorous experimental test of our hypothesis might be conducted.

The Shenger-Krestovnikova Study

The importance of predictability is particularly apparent in the study of Shenger-Krestovnikova described at the outset (cited in Pavlov, 1927, pp. 290–293). A hungry dog receives food in the presence of one CS (circle) and not in the presence of another CS (ellipse). The dog rapidly learns to predict when food will occur. As the ellipse becomes more and more circular and the discrimination increasingly difficult, there comes a point when the dog can no longer reliably predict whether a given CS will be followed by food. This state of unpredictability persisting over 3 weeks must be very stressful for a hungry dog, and the resulting disturbance in his behavior is not surprising. When the discrimination is made easy again and predictability restored, his behavioral disturbance disappears. That he initially has difficulty relearning the easy discrimination is in line with the results of MacKintosh's (1973) study on learned irrelevance and Seligman's (1968) study of associative retardation. When the semi-axes of the ellipse again occur in the ratio of 9:8, the behavioral disturbance reappears, reflecting the loss of predictability that has occurred, now for the second time.

It may be argued that if the two CSs are functionally equivalent, then $p(\text{UCS/CS}+) = .5$ and $p(\text{UCS/}\overline{\text{CS}}) = 0$, and the appearance of food is not really unpredictable in the strict sense at all. According to this argument, the Shenger-Krestovnikova experiment simply involves partial reinforcement, and the eruption of experimental neurosis is truly surprising. However, this objection fails to recognize that for an animal who has been in a discriminative conditioning situation for a long time, the two most salient parameters are not $p(\text{UCS/CS}+)$ and $p(\text{UCS/}\overline{\text{CS}})$ but rather $p(\text{UCS/CS}+)$ and $p(\text{UCS/CS}-)$. In a typical discriminative conditioning experiment, $p(\text{UCS/CS}+) = 1$, $p(\text{UCS/CS}-) = 0$, and $p(\text{UCS/}\overline{\text{CS}}) = 0$. As long as these two most salient parameters are unequal, onset of the UCS is at least somewhat predictable. In the Shenger-Krestovnikova study, however, at the point where the circle and the ellipse can no longer be distinguished, $p(\text{UCS/CS}+) = p(\text{UCS/CS}-) = .5$. Thus, in terms of the parameters that are salient for the animal, the situation is subjectively one of unpredictability rather than one of partial reinforcement.

We speculate that the important variable resulting in the behavioral disturbance is the

loss of predictability in an animal who once possessed it in the context of discriminative conditioning. In principle, if a dog with no extensive previous history of discrimination learning were simply presented with a circle and a 9:8 ellipse and only reinforced for the circle, even over a period of many weeks, experimental neurosis would undoubtedly not emerge (see Kimmel, 1975, for a similar argument). In view of the complete lack of research on the loss of predictability, as opposed to the simple lack of it, we feel that the Shenger-Krestovnikova study illuminates an important topic for future study.

Other Studies from Pavlov's Laboratory

In a strict historical sense, priority in the discovery of experimental neurosis goes to Erofëeva in 1912 (cited in Pavlov, 1927, pp. 289–290). In her experiment, a dog was initially presented with a mild electric shock to the skin as a CS in a salivary conditioning experiment. Gradually the intensity of the CS was increased until it was "extremely powerful" and a stable conditioned response was maintained over several months. An attempt was then made to generalize the CR by applying the CS (strong shock) to new places on the skin. After the CR had been generalized to a number of new places, suddenly a "violent defense reaction" occurred, and all traces of the salivary CR, even to the original location and intensity of the CS, disappeared. (Two other dogs were run in similar procedures, and both showed the same sort of disturbance.) Here the CS is a traumatic stimulus in its own right, and it seems likely that it would be important for the animal to be able to predict where on his body that stimulus will be applied. In the absence of this kind of predictability, the animal cannot make any kind of postural adjustment or preparatory response to mitigate the effects of the shock. What appears to be critical, then, in producing this disturbance is that the animale loses his ability to predict the point on his body where the traumatic stimulus will in fact be applied. Note that our emphasis here is on the importance of being able to predict *where* a traumatic stimulus will be applied. In the past the importance of this variable has not been investigated because experimenters have exclusively manipulated predictability over *when* a traumatic stimulus will be applied. The results of the Erofëeva study suggest an important topic for future research.

In 1913 Petrova (cited in Pavlov, 1927, pp. 293–294) conditioned a dog to make salivary responses to each of six different stimuli. Initially the interval between any of the CSs and the UCS was 5 sec; gradually the intervals were increased by 5 sec per day. When the CS–UCS interval reached 120 sec, behavioral disturbances began to appear, and when the interval was further extended to 180 sec, "the animal became quite crazy" (p. 294). Here we are reminded of the studies cited earlier, which indicate that, at least for aversive events, subjects prefer short to long interstimulus intervals (Badia et al., 1967; Knapp et al., 1959), presumably because they can predict events more accurately over the short run. If so, then increasing the CS–UCS delay effectively decreases the animal's ability to predict the occurrence of the UCS. Furthermore, if one considers that a long-delay CS has several components, some of which are excitatory, some relatively neutral, and others inhibitory (Pavlov, 1927; Rescorla, 1967), then the predicament of this animal becomes more apparent. Each day, what was previously the excitatory part of the CS now must become inhibitory (i.e., that part of the CS which previously informed the animal that food was about to appear now no longer predicts food). Konorski (1967) has documented that the transformation of CRs from excitatory to inhibitory, or vice versa, is always a difficult process, and the results are generally unstable. He, in fact, has observed experimental neurosis developing during the transformation of an excitatory CR into an inhibitory one (1967, p. 333). In the Petrova experiment, this state of shifting predictions persists day after day and is compounded by the unreliability of the animal's internal clock, which presumably increases as the delay increases. According to our theory, neurosis would not have resulted if long-delay CSs had been used from the start of conditioning. In this case there would have been no

shifting predictions from day to day and no *loss* of a capacity to predict the UCS. In fact, Pavlov sometimes used this procedure and did not report any instance of experimental neurosis developing.

A fourth demonstration of experimental neurosis, noted only briefly by Pavlov (1927, pp. 301–302), occurred when a dog suddenly received a previously established CS+ immediately following a CS− for a salivary conditioned reflex. Although the details of this experiment are lacking, it seems likely once again that this was a situation in which the dog's capacity to predict what would happen next was exceeded. Generally in a discriminative conditioning experiment, $p(\text{UCS}/\text{CS}+) = 1$, $p(\text{UCS}/\text{CS}-) = 0$, and $p(\text{UCS}/\overline{\text{CS}}) = 0$. Because CS− is normally followed by an interval of $\overline{\text{CS}}$ that is also a reliable predictor of $\overline{\text{UCS}}$, the sudden appearance of CS+ immediately following CS− presents a contradiction similar to the one noted earlier. A "stimulus" (interval following CS−) that once reliably predicted $\overline{\text{UCS}}$, is now filled with CS+, a reliable predictor of UCS. The dog's capacity to predict when food will occur is strained or exceeded. In another account of this experiment, Pavlov (1961, p. 78) noted that when this procedure was repeated many times, the intensity of the disturbance gradually decreased. Eventually this procedure no longer evoked any disturbance. By our analysis, this is not surprising because the interval following the CS− by this point no longer predicts $\overline{\text{UCS}}$. In fact, there is now a sequential compound CS (CS− CS+) that reliably predicts UCS. Such a compound CS would probably function like any long-delay CS, with the initial parts being inhibitory and the later parts excitatory.

So although Pavlov attributed his dogs' agitated and disturbed behaviors in each of these situations to a "clash" between cortical excitation and inhibition, we believe that the antecedent conditions can each be characterized as having created a loss of predictability. Actually, from an operational and functional standpoint, the situations that create clashes of excitation and inhibition are generally identical to those that create a loss of predictability. The advantage that our theory has over Pavlov's is that it does not rest on outmoded, quasi-neurophysiological concepts.

Gantt

W. Horsely Gantt, who spent some time in Pavlov's laboratory, helped to introduce the concept of experimental neurosis to this country by virtue of his translation of Pavlov's (1928) lectures. His own experiments followed Shenger-Krestovnikova's procedure for inducing experimental neurosis, by employing difficult discriminations in salivary conditioning with dogs (Gantt, 1944). Following Pavlovian theory, he has interpreted the behavioral disorders that he observed to be the result of a clash of excitation and inhibition (Gantt, 1944, 1971). Following our earlier analysis of Shenger-Krestovnikova's study, however, we believe that the experimental neurosis is related to the loss of predictability that occurs when discriminations become extremely difficult or impossible.

Aside from replicating and extending Pavlov's procedures, Gantt made several unique contributions to our knowledge of experimental neurosis, stemming from the many years of careful observations that were made in his laboratory. For example, the case histories of his dogs Peter, Fritz, and Nick (Gantt, 1944) underscore Pavlov's earlier emphasis on the contribution of individual differences in temperament to susceptibility to experimental neurosis, to symptomatic manifestations of breakdown, and to prognosis for recovery. Furthermore, his observations of long-term symptomatic changes led him to originate the concepts of schizokinesis and autokinesis (Gantt, 1937, 1953). *Autokinesis* refers to the spontaneous development of new symptoms over time, even after the discontinuation of the experimental procedure. In *schizokinesis*, separate components of a conditioned reflex develop at different rates and persist for different periods of time, resulting in a disharmony or cleavage in behavioral, emotional, and psychophysiological response systems, which is an important aspect of our current understanding of fear (e.g., Lang, 1968). The importance of

these two concepts in interaction with predictability and controllability has not yet been investigated but would appear to be an important topic for future research.

Liddell

Perhaps the most prolific investigators of experimental neurosis were Liddell and his coworkers at Cornell, who over many years studied experimental neurosis in sheep, goats, and pigs, as well as in dogs (Anderson & Liddell, 1935; Anderson & Parmenter, 1941; Liddell, 1944). Their procedures were also Pavlovian, although they generally employed a defensive leg-flexion CR, with shock as the UCS, rather than a salivary CR. This difference is important because in defensive classical conditioning, the subject is clearly placed in a situation in which he/she has no control over the occurrence of aversive events. Moreover, it was often the case that the interval between the CS and the UCS was variable (e.g., Anderson & Liddell, 1935, Sheeps 2 and 3). Thus, the onset of shock was often somewhat unpredictable, as well as uncontrollable. These threads of unpredictability and uncontrollability run throughout the Cornell group's studies, and are further compounded by other aspects of the procedures employed.

Five different procedures were found to produce experimental neurosis (Anderson & Parmenter, 1941; Liddell, 1944). Often several of these were employed concurrently, making it difficult to assign definite responsibility in each particular case. Two of these procedures involved variants of the difficult discrimination and increasing delay of the CS paradigms studied in Pavlov's laboratory. As discussed earlier, these are both situations in which the animal loses its ability to predict when the UCS will occur, with results that are even less surprising in view of the fact that the UCS employed was shock rather than food. A third procedure, which was accidentally found to produce experimental neurosis, involved increasing substantially the number of conditioning trials presented each day. If the experimental situation was already one in which the controllability and sometimes the predictability of aversive events were absent, increasing the number of conditioning trials and consequently the duration or density of the experimental session may conceivably have multiplied the stress experienced by the animals.

The two remaining procedures involved, respectively, alternating the presentation of CS+ and CS− for shock according to a rigid time schedule and presenting a long series of CSs− in order to establish a simple discrimination (extinciton method). Frankly, from the point of view adopted in this article, these procedures alone should not have produced behavioral disorders. In both cases, the occurrence of environmental events seems perfectly predictable. We suspect that these last two procedures, neutral or only moderately aversive in and of themselves, were compounded by other factors, both procedural and constitutional, which were actually responsible for the disturbed behavior. For example, Anderson and Parmenter's (1941) Sheep D showed signs of experimental neurosis following a long series of CS− trials presented in an attempt to establish an easy discrimination. The authors remarked, however, that this sheep was extremely nervous and "skittish" even before he was introduced to the laboratory. Additionally, some sheep (e.g., Sheep 8) showed signs of experimental neurosis when rhythmic alternation of CS+ and CS− was introduced, but others (e.g., Sheep 11) showed no signs of experimental neurosis even over several years of similar experience. In both instances, it seems that constitutional factors, rather than the experimental procedures themselves, may have played a primary role in the breakdown observed.

Initially Liddell's group attributed experimental neurosis to a variety of neuroendocrine changes (e.g., Anderson & Parmenter, 1941). Later, however, Liddell (1944, p. 393) offered an interpretation very much in accord with our own, noting that the domesticated animal already lives under conditions of considerable restraint—a condition that is exacerbated by the exigencies of the laboratory experiment. Restraint, or loss of control, in either situation alone may be disturbing to the animal, and the two in conjunction are likely to be even more stressful. Liddell often

emphasized that laboratory experiments which did not involve restraint of movement (as did the Pavlovian harness), such as insoluble mazes, did not produce experimental neurosis. Moreover, we noted that in the case of Sheep 8 (Anderson & Parmenter, 1941), signs of experimental neurosis disappeared when the defensive conditioning was carried out in a situation that permitted freedom of movement. Although Liddell's experiments were not conducted with the operational concepts of controllability and predictability in mind, it is clear that he considered similar concepts (especially control) to be critical.

Masserman

In procedural terms, the work of Masserman contrasts sharply with that of Gantt and Liddell, which was conducted around the same time. Whereas Gantt's and Liddell's methods were closely tied to Pavlov's, Masserman used a punishment procedure to induce "motivational conflict" in his cats and monkeys. In his early experiments, cats were trained to associate a signal with the availability of food. After the cats reliably opened the lid of the food container upon presentation of the signal, a variety of procedural changes were introduced. For example, some cats were prevented from reaching the food box, others were put on a partial reinforcement schedule, and still others were punished with an air blast or electric shock the moment they opened the lid to feed. Only the punishment of feeding resulted in profound and persistent behavioral changes. Complete cessation of feeding in the box generally ensued rapidly in the punished animals. Moreover, the animals showed marked changes in activity levels, strong behavioral and psychophysiological signs of fear of the signal, stereotyped defensive reactions, alterations in the dominance hierarchy, and displacement activity (Masserman, 1943, pp. 67–71). These behavioral changes persisted over many months, despite the fact that the punishments were discontinued. Furthermore, the behavioral disturbances were highly resistant to most therapeutic attempts.

That the cats reacted in this way is not particularly surprising because they were being unexpectedly punished for an instinctive consummatory response (Solomon, 1964). From the point of view being developed here, however, what is especially interesting is the procedure that was the most effective in ultimately diminishing the symptoms of experimental neurosis. Some of Masserman's cats were initially trained to press a switch to activate the signal that was followed by food (i.e., they had control over the presentation of food to themselves). When punishment was introduced at the moment of feeding, these cats still developed all the "neurotic" symptoms, although in a somewhat milder form. More important is the fact that during extinction (i.e., when punishment was discontinued), these cats showed by far the most rapid return to normal behavior patterns. These results are interesting from our viewpoint because they indicate that when an animal has prior experience with control over food presentation, it seems to be "immunized" against the long-term, neurosis-inducing effects of punishment. Control may serve both to attenuate the effects of the punishment per se and to help break up the neurotic symptoms once they have been established. Similar conclusions are suggested by the observations of Masserman and Pechtel (1953).

Masserman (1943) originally interpreted his findings in psychodynamic terms, attributing the neurotic behavior patterns to conflict between approach (feeding) and avoidance (fear) motives. Recently, however, Masserman (1971) himself has offered a modified account that is similar to, though less formalized than, the one developed here. He concluded that "the various forms of 'conflict' . . . may be subsumed under a broader etiological rubric: namely, that in each instance the organism apprehends a failure to predict and control events important to its welfare" (1971, p. 9).

Wolpe

In an attempt to clarify certain aspects of Masserman's work, Wolpe (1952, 1958) introduced an additional comparison group. Paralleling Masserman's procedure, cats in one condition were first trained to feed at a buzzer signal from a box and later subjected

to footshocks at the moment of feeding. An average of four shocks was needed to completely suppress feeding in the situation. The other group of cats was simply given classical fear-conditioning trials, in which a horn was followed by footshock. This group received an average of 10–20 shocks over two sessions. Both groups subsequently showed signs of experimental neurosis, including generalized anxiety inside and outside the experimental situation, activity-level changes, increased startle responses, and refusal to feed in the experimental chamber even when very hungry. These effects persisted over many sessions despite the fact that no more shocks were given. Because essentially identical behavior was observed in both groups, Wolpe explained the development of experimental neurosis as a simple example of fear conditioning, in contrast to the dynamic account provided by Masserman. Although more shocks were given to Wolpe's simple fear-conditioning group, Smart (1965) confirmed Wolpe's observations in an experiment that equated the shocks received by the conflict and no-conflict groups.

Wolpe (1958) extended his theory to account for all the examples of experimental neurosis that have been discussed here. He asserted that in all cases, fear or anxiety was aroused and conditioned to situational cues, thus accounting for the persistence of the behavioral disturbances even when the precipitating conditions had been removed. Wolpe's theory concentrates on the internal state of the animal (anxiety) and the conditioning of that state to neutral cues. His treatment of the precipitating factors of anxiety is secondary (noxious or ambivalent stimuli impinging on the organism) and somewhat post hoc. For example, if difficult discriminations had not produced neuroses, Wolpe would not have looked for evidence to argue that ambivalent stimulation produces anxiety (e.g., his citation of Fonberg, 1956). In essence, any procedural manipulation that resulted in experimental neurosis would be assumed also to cause anxiety, often without any independent means of verifying that fact. For Wolpe, experimental neurosis is conditioned anxiety, more or less by definition.

Our approach differs from Wolpe's in that we emphasize the precipitating conditions that lead to experimental neurosis: When important life events become unpredictable or uncontrollable, or both, behavioral disturbances result that have sometimes been labelled *experimental neurosis*. The state of the organism induced by such manipulations of controllability or predictability may *sometimes* be what Wolpe labelled "anxiety," and when it is, we would expect that anxiety to be conditioned to the experimental situation. If, however, some other state, not easily characterized as anxiety, is induced (e.g., passivity and depression), then we may also call this experimental neurosis, whereas Wolpe would not. The advantages of our approach are twofold. First, it is more easily testable than Wolpe's because the precipitating conditions can be operationalized. Second, it is not concerned with whether the state of the organism in every case of experimental neurosis can always be characterized as anxiety.

We now illustrate the testability of our theory with regard to Wolpe's own paradigm for experimental neurosis. We have argued that predictable aversive events are considerably less aversive than unpredictable ones (e.g., Seligman, 1968). Thus, it is not immediately clear why Wolpe's cats who received classical fear-conditioning trials developed generalized neurosis whereas Seligman's rats who received classical fear conditioning did not. However, if we look at Seligman's rats on their first 6 days of conditioning, we find that their behavior was as suppressed as that of the unpredictable group (i.e., they were exhibiting a CER to the situation as a whole). Only on Day 7 did their CER start to become specific to the CS. This is what would be expected by current models of Pavlovian conditioning (e.g., Rescorla & Wagner, 1972). Because Wolpe's cats received only 10–20 fear-conditioning trials over only 2 days, it is not surprising that their fear was conditioned to the situation as a whole rather than specifically to the CS. Our prediction, then, is that further CS–UCS pairings would have made Wolpe's cats *less* generally neurotic (i.e., their fear would have become specific to the CS) rather than more neurotic, as Wolpe

would predict.[2] In other words, predictability can only mitigate anxiety if the animal has had a chance to learn that there is a reliable predictor of the UCS.

Implications

We have shown that many of the behavioral effects of exposure to unpredictable and/or uncontrollable shock are mirrored in the symptoms of experimental neurosis—for example, cognitive deficits, agitation, passivity, disturbance in feeding and in patterns of dominance (see especially Thomas & Dewald, 1977). These parallels suggest that the procedures employed for inducing experimental neurosis may be amenable to analysis in terms of the predictability and controllability of important environmental events. Our retrospective analysis of the classic studies performed by Pavlov, Gantt, Liddell, Masserman, and Wolpe, and their associates, suggests that these factors did play an important and perhaps central role in their procedures and in the outcomes that were observed. In light of the wide ranges of procedures employed to produce experimental neurosis, the fact that two common features can be distilled is particularly striking. Our account seems especially compelling for the studies of Pavlov, Gantt, Masserman, and Wolpe. Liddell's work occasionally presents some difficulties, but even there we have been able to discern elements of unpredictability and uncontrollability (especially the latter). We also noted that some of these investigators themselves anticipated our account by referring to problems of expectations and restraint posed by their procedures.

Questions for Further Research

Our analysis raises as many questions as it suggests answers. First, we are impressed by the striking individual differences in subjects' reactions to any particular procedure used to induce experimental neurosis. Some animals become passive, but others become diffusely agitated; still others develop specific defensive reactions of many kinds. Further, the Pavlov and Liddell studies remind us that some animals break down under what appears to be relatively moderate stress, whereas some require extraordinary treatment. It is important to remember that many animals do not develop experimental neurosis at all. Some of the relevant factors here probably relate to enduring temperamental features of the animals. We certainly need further research on the genetic–constitutional, as well as the experiential, factors that predispose an organism to react in a certain way to situations in which it has lost predictability and controllability. Attempts to isolate the genetic factors responsible for behavioral differences in fear and avoidance conditioning have been reported by Broadhurst (1969), Katzev and Mills (1974), and Gray (1971), among others, but their research has not yet dealt with the issues of predictability and controllability. Seligman (1975) and his associates, furthermore, have speculated extensively on those aspects of experience, especially early in the course of development, that many immunize the organism to the loss of controllability, but this theory at present lacks specificity.

Still another issue highlighted by the experimental neurosis literature pertains to the relationship between predictability and controllability, and the ways in which the lack of one can compound the effects of the lack of the other. In the experimental neurosis literature, nearly all the paradigms are variants on classical conditioning paradigms (with the exception of Masserman's punishment procedure), and hence involve uncontrollable events with varying degrees of unpredictability. The extent to which the effects of uncontrollability are compounded by these varying degrees of unpredictability is not yet clear because few studies have independently manipulated predictability of stimulus onset and controllability of stimulus offset. In one such study, using

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[2] Interestingly, Wolpe has data from one cat that tends to support our prediction. His Cat 14 (1958, p. 54) was given extensive discriminative conditioning in the following paradigm: Food pellets preceded by a buzzer (CS−) were not followed by shock, whereas food pellets preceded by no buzzer (CS+) were followed by shock. Over 21 sessions the cat learned the discrimination and only showed signs of fear when pellets not preceded by a buzzer (CS+) were dropped into the box. By this time he did not show signs of experimental neurosis, although in the earlier sessions there is some indication that he did.

a stress-induced ulceration measure, Weiss (1971) found signalled escapable shock to be least stressful, unsignalled escapable and signalled inescapable shock to be equally and somewhat more stressful, and unsignalled inescapable shock to be most stressful.

A third matter pertains to what might be called the *threshold* for response to the lack of predictability and/or controllability. We noted earlier that punishment and fear conditioning—situations in which aversive stimulation is inherently uncontrollable—do not ordinarily produce gross behavioral disturbances of the kind observed by Masserman (1943) and Wolpe (1958). Obviously some other factors must contribute in important ways to the production of experimental neurosis. For example, the punishment of a consummatory response may be especially stressful for an organism because in essence he loses a substantial degree of control over a vital life function. In addition, predictability and controllability may be especially salient features of an environment when aversive stimulation is intense, frequent, or prolonged, but not when it is mild or infrequent. Future research should attend more closely to differentiating those situations in which lack or loss of prediction and control matters from those in which these factors are of minimal importance. In one such recent attempt, Wortman and Brehm (1975) have argued that the initial response to loss of control is reactance or motivational arousal. Repeated or prolonged exposure to the same uncontrollable events, however, eventually results in the cognitive, affective, and motivational deficits labelled *learned helplessness* by Seligman, Maier, and their colleagues. Furthermore, the more important the uncontrollable outcome is to the subject, the more helpless he will eventually become (see Roth & Kubal, 1975, for recent empirical support of this integration of reactance theory with learned helplessness theory).

A fourth issue highlighted by our considerations of the experimental neurosis literature pertains to the importance of lack versus loss of controllability and predictability. Most research reviewed in our discussion of these two concepts has emphasized the importance of lack of control or predictability. In fact, with regard to controllability, Seligman (1975) has argued that a prior history of control should immunize against the effects of loss of control. Intuitively, however, it seems obvious that there will be cases in which loss of control, given some prior history of control, *will* have profound effects like those we have discussed in this article. There may in fact be situations in which loss of control is more stressful than never having had it (Hanson, Larson, & Snowdon, 1976; Stroebel, 1969). With regard to predictability, we have indicated earlier that there seems to be a complete lack of research on the results of loss of predictability. No concept parallel to immunization has been suggested for predictability, and our analysis of Pavlov's and Gantt's cases of experimental neurosis would lead us to expect that loss of predictability in an organism who once had it may sometimes produce more profound disturbances than mere lack of predictability in an organism who never had it.

Clinical Relevance of Experimental Neurosis

In this article, we have intentionally ignored the issue of whether the symptoms of experimental neurosis directly resemble those of neuroses seen in the psychological clinic. This matter has been dealt with by others (e.g., Abramson & Seligman, 1977; Broadhurst, 1961; Hebb, 1947), who have generally concluded that the parallel between experimental and clinical neuroses should not be pressed too far. The reason for such reservations does not stem from any specific evidence that the symptoms are not parallel. Rather, such reservations derive from the fact that few systematic empirical comparisons between experimental animal and human clinical neuroses have ever been made. Within the framework of current approaches to animal models of human psychopathology (e.g., Abramson & Seligman, 1977; McKinney, 1974), it would, in principle, be possible to determine if experimental neurosis is a good model of any form of human psychopathology. Certainly our review suggests that there are some important commonalities between the *sources* of experimental neuroses and the factors known to be important in the etiology and maintenance of certain forms of clinical psychopathology.

It does seem unlikely that all the cases of experimental neurosis resulting from the myriad procedures we have discussed here would all model one form of psychopathology. Some of the symptoms as described by the investigators resemble certain symptoms of anxiety —hypersensitivity, howling, rapid respiration, piloerection, muscular tension, mydriasis (e.g., Wolpe, 1958). Other symptoms resemble symptoms of depression—passivity, lethargy, smooth hair, anorexia (e.g., Thomas & Dewald, 1977). Interestingly, some animals seem to exhibit first the anxiety symptoms and later the depressive symptoms (e.g., Thomas & Dewald, 1977). Some investigators have suggested that excessive amounts of unpredictability generally lead to anxiety, whereas excessive amounts of uncontrollability generally lead to depression (e.g., Seligman, 1975). But because lack of predictability and lack of controllability are intertwined in complex ways, this point of view suggests a distinction between anxiety and depression that may be oversimplified. We note, for example, that anxiety and depression often occur together, especially in neurotics (e.g., Derogatis, Klerman, & Lipman, 1972; Mendels & Weinstein, 1972), and that prolonged anxiety states generally end in depression (Freud, 1926/1959).

These considerations of predictability and controllability may allow future investigators to spell out some of the possible relationships between anxiety and depression in terms that are more adequately operationalized than those used in the past. Environmental events must in principle be either predictable or unpredictable and either controllable or uncontrollable, generating four possible combinations of predictability and controllability. Moreover, an organism may often not be able to find the correct coping response necessary to gain control; hence, events that are in principle controllable may be *perceived* as uncontrollable.[3] This could occur either because the person may actually not see the relationship between the possible response and a favorable outcome or, alternatively, because he may perceive the response–outcome relationship but perceive himself to be incapable of performing the response (Bandura, 1977). These three variables then—unpredictability, veridical uncontrollability, and perceived (nonveridical) uncontrollability—may in fact generate six possible "mixes" of anxiety and depression.

Conclusion

We obviously believe that experimental neurosis is more than a historical curiosity or an academic puzzle. In the first place, it has raised many interesting new questions about the behavioral and cognitive effects of unpredictable and/or uncontrollable environmental events. Moreover, it seems that there are some important similarities between experimental neurosis and human psychopathology. In this latter point we concur with Thomas and Dewald (1977), who have noted striking parallels between experimental neurosis and learned helplessness following exposure to uncontrollable shock, which in turn has been linked to depression (Seligman, 1975). Their emphasis has been on comparing symptomatology, associated brain mechanisms, and cure. However, an adequate laboratory model must go beyond these points of similarity and explore etiological commonalities as well (Abramson & Seligman, 1977; Seligman, 1975).

In this article we have tried to document two threads that run through the experimental neurosis literature—unpredictability and uncontrollability. These factors have also been found to be important elements underlying certain behavioral disorders that are observed clinically. It is our hope that this analysis will lead to further empirical attempts both to understand and to learn from the phenomena of experimental neurosis.

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[3] Logically, the possibility also exists that uncontrollable events are actually thought to be controllable (e.g., through superstitious learning). This possibility will not be dealt with here, because it is probably not important in generating the kinds of psychopathological symptoms we deal with.

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