Tests of Lenzer's model of intracranial reinforcement*

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Rats were fixed with chronically indwelling electrodes to test Lenzer's model of positive intracranial reinforcement. The model stresses the associative bonding supposedly occurring between stimuli of the brain stimulation and stimuli of the environment where self-stimulation can occur. Rats ran an alley just as efficiently when the opportunities for associative bonding were maximized as when they were minimized. Therefore, it was concluded that the model was not confirmed. It was shown, again, that sodium amytal decreased running time for brain stimulation, a finding that is also incompatible with the model.

The latest attempt to explain the reputed differences between reinforcement by intracranial stimulation (ICS) and conventional reinforcement is Lenzer's (1972) associative model. The model is similar to Deutsch's (Deutsch & Deutsch. 1966). but Lenzer postulates, instead of drive decay. stimulus decay. Reputed discriminative aspects of ICS become associated with stimuli of the environment, and it is this complex of stimuli that elicits running a maze. pressing a bar. or performing discriminations for ICS. To explain the anomalies of ICS. such as the spaced-trial performance decrement. it is postulated that the cue properties of ICS fade with time since last ICS. Because Lenzer's model is an associative model. it demands contiguity between the postulated cues of ICS. which are supposed to be short-lived. and the cues of the environment in which a rat is given an opportunity to respond for ICS. Therefore. according to Lenzer. if ICS (priming ICS) is given minutes prior to the opportunity to run for ICS in a different place. then the priming ICS should have negligible effects on running for ICS. This would hold. according to Lenzer. because the cues of the priming ICS fade rapidly and therefore would not have an opportunity to combine with the stimuli of the place of running for ICS. Consequently. we tested rats after priming ICS in a room away from where they were subsequently allowed to run for ICS.

Lenzer (1972) said that results of our studies (Reid. Wasden, & Courtney. 1970: Wasden & Reid. 1968). showing that sodium amytal reduced spaced-trial performance decrements. "remain to be confirmed [p. 110]." These data. showing a strong effect of a fear-reducing drug on the overnight decrement. are not compatible with a theory that stresses associative processes. Consequently. we again replicated our finding that sodium amytal injections reduced spaced-trial performance decrements.

METHOD
Subjects and Apparatus

Ten adult male albino rats were fixed in standard ways with chronically indwelling electrodes. Electrodes were bipolar. stainless steel. and insulated except at the cross-section of the stimulating tips. All electrodes were aimed for the lateral hypothalamus-medial forebrain bundle. Direct inspection of sections of the brains of seven rats. aided by enlarged photographs of sections. indicated that all stimulation sites were in the area of the medial forebrain bundle: however. the bundle was not stimulated exclusively. In different rats. areas either medial or lateral to the bundle were probably activated.

Testing for responsiveness to ICS was in a C-shaped alley. 305 cm long. 12 cm wide. with walls 29 cm high. (We have been asked why we use a C-shaped runway. Well. the startbox and the goalbox are close to the E. who sits in the opening of the C and therefore does not have to run after the rat but can merely sit and watch as the rat runs around and back to him.) A timer started when the startbox was opened and stopped with the first leverpress. Each leverpress resulted in one ICS of 60-Hz sine waves of 0.25 sec duration. with an intensity determined for each rat.

A square box of clear Lucite in a room down a hall from the room of the alley was also used. This box was fixed to deliver the same duration and intensity of ICS that rats could press for in the alley.

Procedure

After recovery from surgery. rats pressed for various intensities of ICSs in the goalbox of the alley. An intensity that sustained high rates of pressing was selected for each rat. The selected ICSs. which ranged from 30 to 75 microA. was used throughout the study. For each of 6 days thereafter. rats were allowed to travel the alley twice for the opportunity to press for 25 ICSs. The time between daily runs was 10 sec: consequently. the first trial was preceded by about 24 h without ICS and the second trial by 10 sec without ICS.

The basic regimen of two runs a day was continued as a schedule of priming was introduced following an ABBA design. On A days. rats received no priming before being tested in the alley. i.e.. they were run as during the first days of testing. On B days. rats received priming before they were tested in the alley. On the B days of the first ABBA sequence. six rats were primed in the box away from the alley 15 min before being tested in the alley. Each rat was carried to the room. placed in the box. and given its 50 ICSs. The priming ICS was at the rat's selected intensity and was .25 sec long with 2 sec between ICSs. After priming. the rat was returned to its home cage. and 15 min later it was taken to the alley and tested as it was every other day. The six rats plus four others then received two ABBA sequences with 5 min intervening between priming and tests on B days.

All 10 rats were then given. in counterbalanced order (5 rats with ABBA order and 5 with a BAAB order). 2 days of injections with 20-mg kg doses of sodium amytal (or physiologic saline) 20 min prior to trials in the alley.

The last sequence. which included only nine rats because one rat lost an electrode. was again in an ABBA order. but this time priming occurred in the startbox of the alley. Rats were taken on B days to the startbox and given 50 ICSs and then returned to their home cages. Five minutes later. they were returned to the startbox to begin the typical two trials a day.

If a rat failed to run and begin taking ICS within 100 sec after the startbox was opened. it was placed before the lever and its running time was recorded as 100 sec. Being placed before the lever was nearly always sufficient to elicit pressing for ICS. On the rare occasions when pressing did not begin just after being

placed before the bar, one or two E-delivered ICSs started vigorous pressing.

The comparisons of interest are the first-trial running times following either priming or no priming (or drugged and without drug for one comparison), or the mean (M) running time of the A days' first trial compared to the M of B days' first trial of an ABBA sequence. Since the time to press for 25 ICSs at the end of the first trial parallels running time on first trial (West, Hunsicker, & Reid, 1971; Reid, Hunsicker, Kent, Lindsey, & Gallistel, 1973), and since the same conclusions are reached regardless of whether pressing times or running times are analyzed, only running times are reported and discussed here.

RESULTS

Without priming, all rats ran faster on the second trial of a day than they did on the first. Some rats consistently ran the alley in less than 10 sec on the second trial, but typically did not run the first trial within the 100-sec limit. M seconds of all rats to run the first trials on days without priming or injections = 66.1; M of the second trials = 19.4. M seconds to take 20 ICSs after the first trial = 47.8; after the second trial, = 26.4. As is typical in this type of study (Reid et al. 1973), there were large individual differences in performance among rats while a given rat's performance remained stable except when primed or drugged. Some showed a remarkable spaced-trial decrement (a 90-sec, or more, difference between running times of the first and second daily trial), whereas others did not (less than a 5-sec difference). It is the dramatic difference in running between first and second trials that the associative model attempts to explain.

The spaced-trial decrement is not highly correlated with pressing rates once pressing has begun. All rats of this study pressed rapidly once they had begun; M seconds to take 25 ICSs after the second trial was 26.4, with limits of the range being 20.0 and 36.0 sec. The rank order correlation between M time to take 25 ICSs second trial and M first-trial running times was 0.40 for all days without priming or injection. A moderate positive correlation is typical (Reid et al. 1973).

Priming away from the alley should, according to our prediction from Lenzer's theory, have little effect on alley performance, because opportunities are minimal for ICS cues and alley cues to be associated. Priming in a room away from the alley, however, did facilitate alley performance. In the sequence when priming was 15 min prior to tests, the M seconds to run the alley for the first ICS was 57.4; on the days of the sequence with no priming, the M was 60.2. For four rats, priming 15 min prior to testing made little difference in running time, while for two rats, the M difference between running time on days with priming and without priming was 11.9 and 7.4 sec. When priming was 5 min prior to testing, all rats ran, on the average, either faster (seven rats) or at the same rate (three rats) following priming than they did on trials without priming. Rats ran with a M of 10 sec faster following priming than they ran without priming. Consequently, it can be concluded that priming away from the alley facilitated running. West et al

(1971) showed that priming in the alley was uniformly effective when time between priming and testing was at or less than 5 min, but that priming was effective with only some rats when the time between priming and testing was greater than 5 min. These findings parallel West et al's (1971) results.

From Lenzer's model, it follows that priming in the alley would be more effective in eliciting running for ICS than would priming away from the alley. M seconds of all first trials preceded by priming by 5 min in the box away from the alley was 53.1, and M after priming in the startbox was 52.7 (N = 9 in this comparison). The difference is hardly large enough to be reliable. For each rat, the M difference between the days of priming 5 min before each run in the box and no priming was compared to the M difference between priming in the startbox and no priming. From the associative model, it would be predicted that the difference would always be greater when priming was given in the alley, but that result occurred with only three out of nine rats. Two rats' performances did not change, and four rats' performances changed in the direction contrary to prediction. An essential dynamic of Lenzer's theory, the opportunity to associate priming ICS cues with alley cues, appears irrelevant.

Injections of sodium amytal lead to decreased running times. When drugged, eight of the rats with amytal ran faster on the first trial of a day than on all other days of testing.

Two rats who had never run the first trial of the day in less than 100 sec when not drugged ran, while under the influence of the first injection of amytal, in 92.1 and 29.8 sec. The M difference between first-trial running times on placebo days and on days with sodium amytal was 26.5 sec.

DISCUSSION

There is no doubt that priming will generally facilitate running for ICS if that priming ICS, or self-delivered ICS, is given seconds or minutes before the opportunity to run (Reid et al. 1973). The question that Lenzer and others have attempted to answer is why priming facilitates running, and furthermore, why priming effects are so time-dependent. Priming, according to Lenzer, allows associative bonding. Accordingly, if priming is given in another place, no associations should be formed and priming should have no effect. One might even predict negative transfer with priming away from the alley because new associative bonds would be formed. The data, however, indicated that priming in another place facilitated subsequent running and that this priming in another place was of nearly equal facilitory effect as priming in the alley.

Although we believe that these demonstrations are tests of Lenzer's model, they can be disputed. Since Lenzer allows the postulation of internal and unobservable cues, there is no reason why other cues cannot be postulated to explain these data. If cues can be hypothesized without the possibility of verification, then there remains no way to test the self-fulfilling theory of Lenzer.

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Habituation of cardiac components of the orienting reflex to stimuli repeated at fixed and variable intervals

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The purpose of this study was to determine the effects of fixed-interval (FI) stimulus repetition on cardiac components of the orienting reflex (OR). It was predicted that variable stimulus repetition would lead to habituation of the OR, whereas a fixed (temporal conditioning) interval paradigm would inhibit such habituation. Thirty-six Ss were employed. The main hypothesis concerning the effects of FI stimulus repetition was confirmed. In addition, the data supported the use of Lang & Hnatiow's (1962) peak-to-valley measure of the cardiac response, although the response appeared to be monophasically decelerative, not diphasic. The results suggest that stimuli having "signal functions" will continually elicit the OR, whereas stimuli not having such functions eventually lose their value as OR elicitors.

Experimental studies of heart rate (HR) generally have yielded contradictory findings concerning the