Intracranial stimulation: Performance decrements and a fear-reducing drug[1]

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Four rats were found to have conspicuous discrete-trial performance decrements when reinforced in a runway with intracranial stimulation (ICS). The decrement was reduced among all Ss and across all daily trials, to a level of no decrement, by administering amobarbital sodium (sodium amytal) 20 min prior to the runway trials. The data support the notion that the ICS decrement, when observed, results from an approach-avoidance conflict; some fear-inducing component of the electrical stimulation appears to play a causal role.

Rats reinforced with intracranial stimulation (ICS) are commonly found to exhibit conspicuous discrete-trial performance decrements. Unlike conventional reinforcers, where performance is not drastically affected by manipulations of the intertrial interval, Ss reinforced with ICS commonly exhibit greatly increased reaction times when separated from the stimulation by either time, or perhaps distance. A number of explanations of these performance decrements have been put forth but regardless of which, if any, of the current hypotheses is most correct, it is important to consider the limiting features of ICS as a reinforcer.

One explanation of the decrements was deduced from Deutsch's (1960; Deutsch & Howarth, 1963) structural theory of behavior. Within this framework each ICS excites a "central motivational state" as well as a state of reinforcement. The central motivational state was postulated to decay rapidly when ICS is discontinued, hence the performance decrement is a direct function of time since the last ICS. The Ss are just not "motivated" for ICS.

An alternative and more informal explanation can be derived from the work of Olds (1962; Briese & Olds, 1964). It might be assumed that some real confusion of associational processes occurs as a result of stimulation in reinforcing areas. The emotion or motivation engendered by appetitive or aversive ICS results in a disruption of mnemonic processes or obscures an intact mnemonic mechanism.

Data from our previous studies (Wasden, Reid, & Porter, 1965; Wasden, 1966) have conflicted with both of these explanations. Importantly, not all of the rats we have tested have shown a discrete-trial performance decrement, even following a 10-day intertrial interval. Some rats did indeed show the decrement but the decrement could be reduced by first administering a nonreinforced trial. We also found a high correlation between sites of stimulation and the presence of the decrement. Rats that were stimulated in the medial forebrain bundle showed little or no decrements, while decrements were shown by rats whose stimulation sites were somewhat removed from the medial forebrain bundle. Those sites associated with decrements were in regions Olds & Olds (1963) have shown to be both positively and negatively reinforcing, depending on rather slight changes in parameters of stimulation. These data, plus other observations, such as the decrement frequently becoming apparent after some training rather than throughout training, led to the speculation that the performance decrement is analogous to behavior seen in studies of approach-avoidance conflict. The present study is a further test of this assumption. If it is avoidant behavior, or fear, that is responsible for the decrement, then administering an effective fear-reducing drug should substantially reduce the decrement. Amobarbital sodium (sodium amytal) is such a fear-reducing drug (Miller, 1961).

Method. The Ss were six adult, male, albino rats, each fitted with a chronically indwelling bipolar electrode with the stimulating sites in the lateral hypothalamus. Following recovery from the surgery each S was tested to see if the stimulation was reinforcing and an intensity of stimulation was determined for each S which was used throughout the remainder of the study. The ICS chosen was that which produced high pressing rates without debilitating side effects (range across Ss = 60 to 150 μA of 60 cycle AC with duration determined by the duration of lever depression up to .5 sec). Two Ss were dropped from this experiment at this time because they would not press at sustained rates at any of the tested intensities.

These preliminary tests were given in the goalbox of a runway that served as the apparatus for subsequent tests. The runway was C-shaped, 305 cm long, 12 cm wide, with walls 29 cm high. The startbox was fitted with a sliding gate and the goalbox with a Scientific Prototype rat lever. The apparatus was programmed so that a running time meter was started when the startbox gate was opened and was automatically stopped when the S first pressed the lever.

The Ss were then trained to traverse the runway to obtain access to the lever in the goalbox. They were allowed 25 reinforced presses on each trial and were given five trials per day, with a 1 min intertrial interval. Ss that failed to reach the goalbox within 200 sec were placed there and allowed the standard 25 reinforced presses before beginning the next trial. Training trials continued for 13 days. On three occasions during the 13 days of training Ss were injected with sodium amytal, at the dosage subsequently used, to observe, among other things, the effects of the drug on seizure production. The data reported here are from a more formal test that followed these training trials; the preliminary data are in no way inconsistent with those reported here.

The Ss were then given five days of standard trials, but they were now permitted only 15 reinforced lever presses on each trial. This was initiated to prevent seizures in two of the Ss. Other conditions were the same as the previous training trials. The five days of standard trials were followed by three days of trials in which Ss were injected with amobarbital sodium (sodium amytal), 20 mg/kg, 20 min before beginning the first daily trial. This was the same dosage and time-interval used by Miller (1961) in demonstrating the fear-reducing properties of sodium amytal and produced a flaccid, drunken-like state in the Ss. This same dosage was also tested with water-deprived rats in a straight-way maze and found to increase running times over nondrug conditions. Drug-trials were followed by three days of trials under the standard (no drug) conditions and then three days in which Ss were injected with physiological saline 20 min prior to the first daily trial. The final phase was three more days of trials under the influence of sodium amytal.

Results and Discussion. The minimum running time in the runway is approximately 6 sec; times substantially longer than this can be considered a performance decrement. All of the four Ss exhibited unduly long running times on all five of the daily trials under standard conditions, thus showing intertrial as well as 24-h

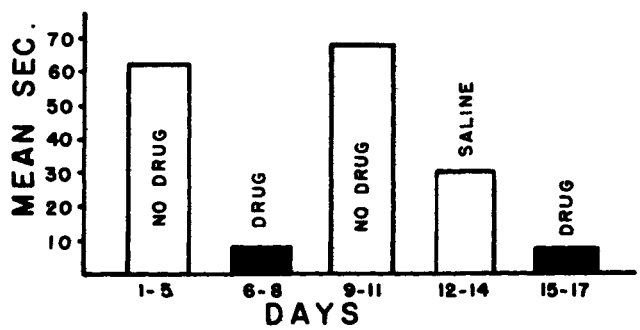

Fig. 1. Mean first-trial running times for four Ss showing effects of intraperitoneal injection of 20 mg/kg of sodium amytal 20 min before testing under drug conditions. The ranges and interquartile ranges of all first-trial scores for each condition were, reading from left to right, (a) 7-200, 16.5-85.5, (b) 6-12, 6.0-10.2, (c) 11-200, 28.5-84.5, (d) 11-70, 14.0-41.5, (e) 5-11, 7.0-9.2.

decrements. The sodium amytal was effective in reducing the running times across all daily trials to a level of no decrement. Fig. 1 shows the results for the first trial of a day's sessions for each condition; data for Trials 2-5 were quite similar to first-trial data across all conditions. The mean first-trial running times for each of these Ss was 19, 31, 75, and 125 sec for the first five-day series of no drug (standard) conditions, and 8, 6, 11, and 8 sec, respectively, for the first three-day series of drug conditions. Differences between drug and standard conditions are statistically reliable since there was no overlap in distributions. Physiological saline as a placebo had a similar, but smaller, effect than did the sodium amytal. The placebo effect was most likely the result of respondent conditioning and a gradual weakening of the effect was noted across the three days of trials.

Neither a theory of time dependent drive decay, or the notion that the electrical stimulation disrupts mnemonic processes, can adequately account for the decrement reducing effects of the sodium amytal. However, the data do support the notion that the ICS decrement results from an approach-avoidance conflict. ICS reinforcement does not always result in performance decrements (Wasden, Reid, & Porter, 1965), but in those instances in which it does, some fear-inducing component of the electrical stimulation appears to play a causal role.

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NOTES

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