Scopolamine and reinforcement omission on fixed-interval schedules¹

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Two rats were run on a fixed-interval schedule with occasional blackouts (2-32 sec) substituted for reinforcement. Injections of scopolamine eliminated the fixed-interval scallop and substantially reduced the elevation in response rate which typically follows reinforcement omission. This result is in agreement with a nonmotivational interpretation of interval-schedule "frustration" effects.

On some reinforcement schedules (e.g., fixed-interval, DRL) behavior comes primarily under the control of temporal factors in the situation. Scopolamine is a drug which, administered in appropriate doses, eliminates temporal control of behavior without eliminating the behavior (e.g., bar-pressing) itself (Boren & Navarro, 1959; Herrnstein, 1959; Carlton, 1963).

This experiment investigated the factors controlling the elevation in response rate which occurs following reinforcement omission on fixed-interval schedules (Staddon & Innis, 1966). It has been suggested (Staddon, 1967) that this effect is due to selective control of responding by reinforcement and time since reinforcement, i.e., to purely associative processes. The effect of reinforcement omission has also been studied in double runway experiments (Amsel & Roussel, 1952; Amsel, 1958; Wagner, 1959). However, the elevation in running rate observed in this situation and in analogous free-operant situations (Davenport & Thompson, 1965; Davenport, Flaherty, & Dyrd, 1966) has been discussed in terms of motivational variables (the *frustration effect*).

By administering scopolamine to rats run on a fixed-interval schedule with occasional reinforcement omissions we hoped to eliminate the temporal control of responding in this situation and thus be better able to evaluate the role, if any, of motivational factors. The experiment included control sessions of three types: (1) injection of methyl scopolamine, a compound similar to scopolamine in its peripheral effects but which does not readily pass the blood-brain barrier (Carlton, 1968), (2) injection of an isotonic saline solution, and (3) no injection.

SUBJECTS

Two male hooded rats from the University of Toronto colony were maintained at 80% of their ad lib weights: B-3, 305 g; B-4, 320 g. The rats had previous training on the procedure used here and were approximately seven months old.

APPARATUS

The experiment was conducted in a single-layer Scientific Prototype rat chamber, modified to contain a dipper feeder from which reinforcement (3-sec access to a 2:1 water and sweetened condensed milk solution) was available. This chamber was housed in a large insulated box. White noise and the noise of the ventilating fan also helped to mask extraneous sounds. A houselight illuminated the box except during reinforcement and time-out periods. Each effective bar-press (greater than about 20 g) produced a feedback click from a relay mounted in the box.

Programming and recording equipment were in an adjacent room. A Stoelting optical film reader, relays and timers were used to program the experiment. Data were recorded on digital counters and a cumulative recorder.

PROCEDURE

Each daily session consisted of 60 2-min fixed intervals, alternately designated C-1 and C-2 (i.e., Interval 1, C-1; Interval 2, C-2; Interval 3, C-1; ... Interval 60, C-2). Responses at the end of C-2 intervals were always reinforced. Responses at the end of C-1 intervals led to reinforcement 50% of the time (according to an irregular sequence) or time-out (TO). The TO was one of five durations (2, 4, 8, 16, or 32 sec), each duration occurring three times per session. Within C-2, fixed intervals preceded by a reinforcement are designated C-2R, those preceded by a time-out, C-2N.

Of the 21 sessions comprising the experiment, scopolamine (S) was administered on eight days, methyl scopolamine (M) on two, and saline (A) on six; on the remaining five days (C) there were no injections. The sequence of experimental conditions was as follows: C C C S A S A S A S A S A S C C S M S M. All injections (intraperitoneal) were made at the home cage 20 min before the beginning of the session and consisted of approximately .31 cc of fluid. A .5-mg/kg solution of scopolamine hydrobromide dissolved in isotonic saline and an equimolar solution of methyl scopolamine were the drug doses used.

Fig. 1. Cumulative records of the first 20 intervals of a session from each of the four conditions for both rats. The response pen reset after each fixed-interval; a blip at the point of reset indicates that the interval was reinforced. Duration of reinforced and unreinforced blackouts at the end of C-1 are indicated by the pen-depressions on the line directly below the response record.

(Continued on page 45)

administered intraperitoneally to the recipient animal and finally absorbed by the brain of the recipient. But this mechanism is highly improbable. Moreover, intraperitoneal injection of 40 µg of Puromycin does not affect the performance of animals in our experimental situation. It was found in our control experiments when we tested the correct doses of Puromycin.

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NOTES

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(Continued from page 43)

Table 1
Mean Relative Frequency of Responses in C-2N, Range of Relative Frequencies, and Number of Sessions

Condition	No. of sessions	B-3		B-4	
		Mean	Range	Mean	Range
Scopolamine	8	.54	.51-.57	.50*	.45-.53
Methyl scopolamine	2	.66	.64-.67	.61*	
Saline	6	.60*	.54-.66	.59*	.53-.68
No injection	5	.63*	.58-.67	.68	.63-.72

* Data from one session of these conditions were not included due to equipment failure or failure of the S to respond during the session.

RESULTS

Figure 1 compares cumulative records from each of the four experimental conditions for both rats. The records from the no-injection control sessions show the pattern of responding typical of this reinforcement schedule: a pause after reinforcement followed by responding until reinforcement occurs again. Following TO (i.e., nonreinforcement) the pause was generally shorter, yielding a higher overall response rate during intervals following reinforcement omission. In contrast, the records for the scopolamine condition reveal no temporal discrimination during the fixed intervals; both rats began responding as soon as reinforcement or TO was over and continued to respond at a fairly steady, low rate. The actual "running" rate on scopolamine days was lower as the records show. The data from the saline and methyl scopolamine conditions resembled the no-injection sessions showing the fixed-interval scallop indicative of temporal control. There was no systematic effect of time-out duration across the four experimental conditions.

If we consider the ratio of responses in C-2 following reinforcement omission to the total number of responses during C-2 (C-2N/C-2) we have an index of the effect of reinforcement omission. If there is no effect, responding should be equal across all C-2 intervals and the ratio should be .5; the greater the omission effect, the larger the ratio. Table 1 records the average relative frequency of C-2N responses for each of the four experimental conditions. Rat B-4 showed no omission effect under the scopolamine condition, while B-3 showed a slight effect (.54). Much larger omission effects were obtained in the other three conditions for both animals.

DISCUSSION

The results of this study are in agreement with the findings of Herrnstein (1959) and Boren & Navarro (1959); administration of scopolamine eliminates the typical fixed-interval scallop. The rats responded throughout the entire fixed interval rather than pausing for a period of time after reinforcement before beginning to respond. The fact that the pattern of responding during the methyl scopolamine condition did not differ from the pattern during the other

control conditions (see Fig. 1) is evidence that the fixed-interval scallop is controlled by a central rather than a peripheral mechanism. Thus, bar-pressing following reinforcement on FI schedules can be described as centrally inhibited; as this inhibition slowly decays over time bar-pressing begins again.

The omission effect was eliminated (B-4) or substantially reduced (B-3) by the administration of scopolamine (cf. Table 1). If the omission of reinforcement results in a motivational reaction (an increase in drive in Hullian terms) then it might be expected that the resulting increase in response rate would be observed even when the rats were under the influence of scopolamine. On the other hand, if the elevation in response rate following reinforcement omission on fixed-interval schedules is due to absence of the usual *inhibitory* effect of reinforcement, as has been previously suggested elsewhere (Staddon, 1967), then the simultaneous elimination both of the FI scallop and the omission effect seen here is quite predictable, since both involve the same inhibitory mechanism. The present results, therefore, give some support to a nonmotivational analysis of interval schedule "frustration" effects.

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NOTE

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