Table 2
Differences in Mean Trials to 95% Efficiency
in Pursuit-Rotor Tracking

Subgroup	Right Hand	Left Hand
Cardiac	7.3	7.5
Control	3.8	4.6
t (df = 28)	4.60*	8.17*

* $p < .001$

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The effects of scopolamine on the extinction of a continuously reinforced response*

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Thirty-five rats were injected with varying doses of scopolamine, methyl scopolamine, and saline before extinction of a continuously reinforced response. Methyl scopolamine and low doses of scopolamine speeded extinction, whereas higher doses of scopolamine led to responding similar to that of saline controls. It is suggested that the relatively weak tendency to respond after CRF training and the drastic switch to extinction from CRF maximized the disruptive peripheral effects of scopolamine, and as a result higher doses were required to show prolongation of extinction.

One of the more interesting effects of anticholinergic drugs on behavior is that they retard or block the extinction of a previously reinforced response. Herrnstein (1958), after injecting rats with doses of scopolamine from .05 mg/kg to 1.6 mg/kg, found increased responding during two phases of a four-ply multiple schedule that had never been associated with reinforcement. Stone (1964) and Carlton (1963) both examined the effects of scopolamine on the extinction of a Sidman avoidance response. Stone used an alternating avoidance-acquisition procedure and with dosages ranging from 1 to 20 mg/kg, demonstrated increased responding during the extinction periods.

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Carlton, who examined terminal extinction, injected his rats with .6 mg/kg before each extinction session and found no decrease in response rate after 10 sessions. Hearst (1959) used doses of .6 mg/kg of scopolamine given before each session and found no evidence of a decrease in response rate in a discrete-trials discrimination task, even after food had been withheld for 40 sessions.

The above studies all involve either some situation in which the organism has had experience with nonreinforcement or the use of shock motivated behavior; both procedures are known to increase resistance to extinction (Jenkins & Stanley, 1950; Solomon & Wynne, 1954). The purpose of the present study was to extend these earlier results with a procedure involving terminal extinction of a previously continuously food-reinforced response. Dose-response relationships for this drug effect were also examined.

It has been demonstrated in a number of studies that peripheral anticholinergic effects are similar for both scopolamine and methyl scopolamine, whereas methyl scopolamine has very weak central effect, if any (Goodman & Gillman, 1965). Janssen & Neimegeers (1967) have shown that the median effective dose (ED50) for production of miosis in rats with scopolamine is .004 mg/kg and .003 mg/kg for methyl scopolamine, but for central effects the ED50 for scopolamine is .016 mg/kg and for methyl scopolamine is greater than .3 mg/kg. Samuel, Kodamma, & Menear (1965), claim scopolamine to be from 10 to 316 times more effective than methyl scopolamine in producing central effects as measured by the ED50 for the disruption of avoidance behavior.

Using related anticholinergics, atropine and methyl atropine, Whitehouse, Lloyd, & Fifer (1964) showed equal decrements in eating behavior at equal doses of both drugs, yet demonstrated disruption of a discrimination task with atropine and not with equivalent doses of methyl atropine.

It has been assumed in this study as in many others (Bohdanecky, Jarvik, & Carley, 1967; Carlton, 1963) that at equal doses methyl scopolamine will have similar peripheral effects as scopolamine but will have minimal central effects and thus will serve as a control for the influence of peripheral anticholinergic blocking on behavior.

SUBJECTS

Ss were 35 male Long-Evans hooded rats between 100 and 150 days old. They were kept on a 23½-h food-deprivation schedule; they were permitted to eat ad lib for ½ h each day after each experimental session. Water was available at all times in the home cage and during experimental sessions.

DESIGN

A 7 by 7 Latin square design with five independent replications was utilized (Edwards, 1950). Each replication involved seven different rats and continued for 7 consecutive days during which time each of seven drug dosages were given. Three doses of scopolamine hydrobromide¹ (.1, .3, and 1.0 mg/kg), three doses of scopolamine methyl bromide (.1, .3, and 1.0 mg/kg), and a saline control dose were given in a random sequence to each animal with the restriction that, within each replication, all seven doses were given on each day of extinction.

APPARATUS

Seven Electrocraft Skinner boxes that delivered a 45-mg Noyes pellet each time the animal depressed a lever that protruded from one wall of the chamber were used. The manner of operation of the feeder permitted the animal to receive pellets at a maximum rate of one every 3 sec. The

Fig. 1. Dose effect curves for scopolamine and methyl scopolamine for total time and feeder operations to extinction.

pellet had to be eaten within 3 sec of presentation or else it would be removed from the apparatus. Operation of the boxes was controlled by solid-state programming equipment.

PROCEDURE

Ss were trained for ½ h each day on a continuously reinforced schedule (CRF) for 3 weeks until response rates had stabilized. They were then assigned randomly to treatment conditions and extinction was begun. During extinction training, the food hoppers were emptied, but the feeder mechanism continued to function.

Ss remained in the boxes each day until 5 min without the emission of a response

had elapsed. The number of feeder operations and the time taken to reach the extinction criterion were recorded.

Drugs were administered in a "blind" manner so that the E did not know which dose each animal had received. Intraperitoneal injections were given 15 min before the beginning of each session. Drugs were mixed in normal saline in different concentrations so that each animal received a constant volume per body weight, 2.5 cc/kg.

RESULTS

Figure 1 shows the dosage curves for both scopolamine and methyl scopolamine on both measures of extinction.

Dosage effects were significant both in time to extinction ($F = 2.956$, $df = 6/150$, $p < .05$) and in number of dipper operations ($F = 2.282$, $df = 6/150$, $p < .05$). Replication effects and interactions of replications with dosages and with days were not significant for either measure.

Orthogonal contrasts showed that the 1.0-mg/kg dosage of scopolamine were significantly different from the 1.0-mg/kg dosage of methyl scopolamine ($F = 10.61$, $df = 1/150$, $p < .005$), but not significantly different from the saline control ($F = 2.39$, $df = 1/150$, $p > .1$) on the time to extinction measure. These comparisons for feeder operation data were not significant ($F = 3.45$, $df = 1/150$, $p < .1$ and $F = 1.30$, $df = 1/150$, $p > .2$).

Increasing doses of scopolamine resulted in increased resistance to extinction. However, the highest doses did not show significantly slower extinction than the saline control. The increasing trend suggests that larger doses might have resulted in prolongation of extinction. Methyl scopolamine decreased extinction as well, but did not show the recovery seen at higher doses of scopolamine.

DISCUSSION

The scopolamine data are contrary to the previously described findings that showed resistance to extinction increased over saline controls at similar doses of scopolamine. The effects of methyl scopolamine, however, are not surprising in that attenuated parasympathetic functioning would be expected to interfere with responding by producing madiasis, dryness of mouth and nasal passages, attenuated heart rate and blood pressure, and interference with the functions of the gastro-intestinal tract (Goodman & Gillman, 1965).

Unlike the animals in the previous studies, these rats had never experienced either shock or nonreinforcement and thus their tendency to continue responding in the absence of reinforcement would not be as strong. Also, the change to

nonreinforcement from CRF is more drastic than from partial reinforcement schedules and such change could override the effects of the drug at the dosages given. It may be that under these circumstances the peripheral debilitating effects of scopolamine are maximal and the central facilitating effects are minimal so that higher doses would be required to demonstrate reliable prolongation of extinction.

The results of this study tend to support the finding that scopolamine can prolong extinction of a previously continuously reinforced response when peripheral effects are controlled. The dosage requirement for this effect is greater than that shown previously for extinction of partially reinforced and shock motivated behavior. Also, under these conditions the peripheral anticholinergic effects of scopolamine can disrupt behavior.

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NOTE

1. The scopolamine hydrobromide was generously donated by the Upjohn Co., Don Mills, Ontario, Canada.