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Effects of Signaled and Unsignaled Brain Stimulation, Water, and Sucrose Reinforcement on Running Behavior in Rats

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Four groups of rats, reinforced with signaled electrical stimulation of the brain (S-ESB), immediate ESB (I-ESB), water, or sucrose, were run for 20 ten-trial sessions in a runway. For 10 sessions the intertrial interval (ITI) was 60 sec and for 10 sessions the ITI was 5 sec. Both ESB groups ran faster with the shorter ITI, but the ITI effect was significantly smaller for the S-ESB group. The water group showed no significant ITI effect, and the sucrose group ran faster with the longer ITI. All groups showed overnight decrements in running speed. It may be that all reinforcing stimuli have a response-facilitating effect on behavior and that this effect alone can account for both the overnight decrements and the differences in the ITI effect from group to group.

When electrical stimulation of the brain (ESB) was made contingent on some behaviors, the frequency of those behaviors was observed to increase (Olds & Milner, 1954), thereby making ESB a positive reinforcing stimulus (Skinner, 1938). Closer examination of the effects of ESB reinforcement on behavior revealed, however, that ESB-reinforced behaviors differed from conventionally (e.g., food) reinforced behaviors; for example, performance for scheduled ESB reinforcement was said to be poor (Sidman, Brady, Boren, Conrad, & Schulman, 1955), and extinction was reported to be unusually rapid (Culbertson, Kling, & Berkley, 1966). These differences have come to be widely accepted as part of the data base of physiological psychology (e.g., see Deutsch & Deutsch, 1973; Milner, 1970).

In spite of this tradition, several experiments have shown that some of the observed differences between ESB- and conventionally reinforced behaviors can be attributed to the methods of presenting the reinforcing stimuli rather than to the reinforcers per se (Cantor, 1971; Gibson, Reid, Sakai, & Porter, 1965; Pliskoff, Wright, & Hawkins, 1965). Cantor, for example, showed that ESB-reinforced behaviors could be controlled by schedules of reinforcement when a brief signal preceded the onset of each ESB by .5 sec. This signal may be analogous to the feeder click for food, for example, thereby making the presentation of ESB more similar to the presentation of conventional reinforcers.

One experimental setting in which ESB-reinforced behavior has consistently been reported to differ from conventionally reinforced behavior is the runway. Rats reinforced by ESB run faster with short intertrial intervals (ITIs) than with long ITIs (Gallistel, 1966, 1967; Panksepp, Gandelman, & Trowill, 1968; Reid, Hunsicker, Kent, Lindsay, & Gallistel, 1973; Seward, Uyeda, & Olds, 1960; Spear, 1962),

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whereas rats reinforced by water have been reported to show no similar ITI effect (Gallistel, 1967), although this report has been challenged by Hunsicker and Reid (1974). Can the ITI effect on ESB-reinforced running be attributed to the method of presenting the ESB? In all but one of the above cited experiments, ESB was presented contiguously with a lever press or lick response in the goal box; in Spear's experiment, ESB was contingent only on entering the goal box. To answer the above question, we undertook the present research to compare the ITI effect demonstrated by a group of rats receiving signaled ESB reinforcement with that of a group receiving immediate ESB reinforcement. If the ITI effect occurs because ESB presentation is contiguous with the response in the goal box, then the signaled ESB group should not show an ITI effect.

A second phenomenon that we investigated was overnight decrement. This is the tendency for rats reinforced by ESB to run slower on the first trial of a session than on the last trial of the preceding day's session (Olds, 1956; Panksepp et al., 1968; Wasden, Reid, & Porter, 1965). Although Gallistel (1973) suggested that "the overnight decrement phenomenon appears to be peculiar to self-stimulation" (p. 192), no adequate test of this hypothesis has been made. The only experiment that employed a food and ESB group (Olds, 1956) had only *one* session in a runway and three sessions in a maze, and it was reported that the ESB group showed a decrement whereas the food group did not. Furthermore, Wasden et al. observed an overnight decrement in only half of their ESB-reinforced rats, and neither they nor Panksepp et al. ran animals in a similar setting for a conventional reinforcer. Logan (1960) did train rats in a runway for food reinforcement and reported a small overnight decrement. The procedure, however, was unlike that typically employed in an ESB experiment, with long and somewhat variable ITIs, which makes comparison difficult. In the present experiment, a water-reinforced group was run in order to compare its overnight decrement with that of the ESB groups (as well as to compare ITI effects).

A fourth group of rats reinforced by a 50% sucrose solution was added. Some theoretical positions have held that the performance of ESB-reinforced rats is not attributable to ESB per se but to the conditions of ESB reinforcement, which happen to maximize incentive motivation (Trowill, Panksepp, & Gandelman, 1969, p. 271). According to this view, the signaled ESB group should run slower than the immediate-ESB group because of incentive differences caused by differences in immediacy of reinforcement (Logan, 1960, pp. 44–52). Similarly, the water group should run slower than the sucrose group because of differences in quality of reinforcement (Goodrich, 1960; Kraeling, 1961; Rosen, 1966). It was hoped that the sucrose–water comparison would provide information helpful in comparing the ESB groups.

Method

Subjects

Thirty-four experimentally naive male rats of the hooded Long-Evans strain were housed individually. Twenty rats had food and water continuously available; the remaining 14 had food continuously available but received water for only 30 min each day at approximately the same time each day.

Surgery and Histology

Each of the 20 rats that had food and water continuously available was to be trained to electrically stimulate its own brain. Thus, after anesthetization with an ip injection of 50 mg/kg Nembutal, each was stereotaxically implanted with a Plastic Products bipolar electrode (MS 303-0.010) made of intertwisted stainless steel wires .25 mm in diameter. With the incisor bar set at 3.2 mm above the interaural line, coordinates of .8 mm posterior to bregma, 1.5 mm lateral to the midline, and 8.6–8.8 mm below the surface of the skull were used, with the lateral hypothalamus as the target. (Although it was never used in this experiment, 9 of the 20 rats had an additional implanted electrode aimed at the lateral septum. Coordinates from bregma for this placement were anterior 1.4 mm, lateral .7 mm, and ventral 5.3 mm.) At least 7 days of recovery from surgery preceded the initiation of testing.

Following completion of the experiment, the implanted rats were killed and perfused through the

heart with saline followed by 10% formalin. The brains were extracted, fixed in 10% formalin, sliced at 40 μm, and stained with thionine to verify electrode loci.

Apparatus

The experimental environment consisted of a runway which was 183.0 × 15.0 × 15.5 cm. The first 22 cm of the runway were partitioned from the remainder of the runway by a removable door and constituted the start box. Immediately outside the start box was a photocell that operated a timer when broken. A similar photocell, which terminated the timer when broken, was located 13.5 cm from the opposite end of the runway. The area between this second photocell and the end wall was the goal box. In the middle of the end wall, at a height of 9.0 cm was a lever (Gerbrands Model G6312). The lever projected 1.7 cm into the goal box, was 5.2 cm wide and 1.3 cm thick, and had a force requirement of about 35.0 g. A liquid feeder cup (BRS/LVE Model 114-02) was located to the right of the lever on the side wall, 2.5 cm from the end wall, at a height of 4.0 cm. The entire runway was located on a table top in a windowless room which was lit by a shaded 60-W bulb hung above and to the side of the runway. Also located on the table top was a pellet feeder which made a click when operated; this was used as the source of a signal for one of the experimental groups.

Electrical stimulation was provided through a lead suspended from the ceiling and equipped with a commutator, which allowed the rats freedom of movement. The ESB was provided by a Grass S-6 stimulator; each stimulation consisted of a .5-sec train of rectangular, biphasic pulses, .2 msec in duration, presented at 100 Hz. The current intensity varied from rat to rat and ranged from 250 to 560 μA.

Procedure

The 20 rats with electrodes were divided into two groups of 10; the 14 remaining rats were divided into additional groups of 8 and 6 rats. Each of these four groups was trained in a similar fashion in the runway, but reinforcement was different for each group.

For the first group of 10 (Group S-ESB), the lever-press response was shaped with reinforcement consisting of a train of ESB that was signaled by a click .5 sec before its onset; i.e., each lever press was followed immediately by a click, followed by ESB .5 sec afterward. When the response was shaped, each rat received at least 100 reinforcements on a continuous reinforcement schedule before the session was terminated. In the course of several subsequent sessions, each rat was trained to traverse the runway; each run was reinforced by five response-contingent trains of signaled ESB. All rats learned to initiate lever pressing upon arrival in the goal box during these preliminary sessions, and priming, the presentation of free ESB, was never necessary to initiate lever pressing in subsequent sessions.

The experiment consisted of 20 ten-trial sessions, 10 sessions with a 60-sec ITI and 10 with a 5-sec ITI. Five of the S-ESB rats received the ten 60-sec ITI sessions followed by the 5-sec ITI condition; this order was reversed for the other five rats. During the 5-sec ITI condition, each rat was picked up from the goal box after its fifth lever press, placed in the start box, and left for 5 sec; then the door was removed to begin the next trial. During the 60-sec ITI condition, each rat, after its fifth lever press, was placed in its home cage, which was located beside the runway on the table top, and left for 55 sec. The rat was then placed in the start box for an additional 5 sec after which the door was removed to begin the next trial. This procedure was also followed at the beginning of a 60-sec ITI session after the rat was connected to the lead. If a rat either failed to leave the start box for 120 sec or failed to enter the goal box for 120 sec after breaking the first photocell, it was gently pushed into the goal box and a running time of 120 sec was recorded. Otherwise, the dependent variable was running time as indicated on the timer after each trial.

The second group of 10 rats is referred to as Group I-ESB. For this group, reinforcement for the running response consisted of five trains of lever-response-contingent ESB, but the onset of each train was contiguous with lever depression. Otherwise, training procedures were identical to those described for the S-ESB group.

The third group (Group HOH), consisting of eight of the water-deprived rats, was trained with reinforcement consisting of a .15-ml cup of water. Shaping of the lever-press response was difficult and time consuming; therefore, after the response was shaped for four rats, runway training was begun. Each trial was reinforced by two presentations of the dipper; for four rats these were contingent on the (shaped) lever-press response, and for the other four, the two dipper presentations occurred automatically when they entered the goal box and immediately after they drank. The training procedure was otherwise identical to that described for Group S-ESB above. Four rats received the 5-sec ITI condition followed by the 60-sec ITI condition, and the other four received ITI conditions in the opposite order. Two of each order subgroup of four were lever pressers and two were not.

The remaining six water-deprived rats constituted Group 50%S. The lever-press response was shaped with reinforcement consisting of .15 ml of a 50% sucrose solution (i.e., 50 g of sugar per 100 ml of water). Runway training followed, with each trial being reinforced by two lever-response-contingent presentations of the dipper. Training followed the procedure outlined for group S-ESB. Four rats received the 5-sec ITI condition first; two rats received the 60-sec ITI condition first.

Results

Histology

Histological examination of electrode loci (Figure 1) for the 10 rats of Group S-

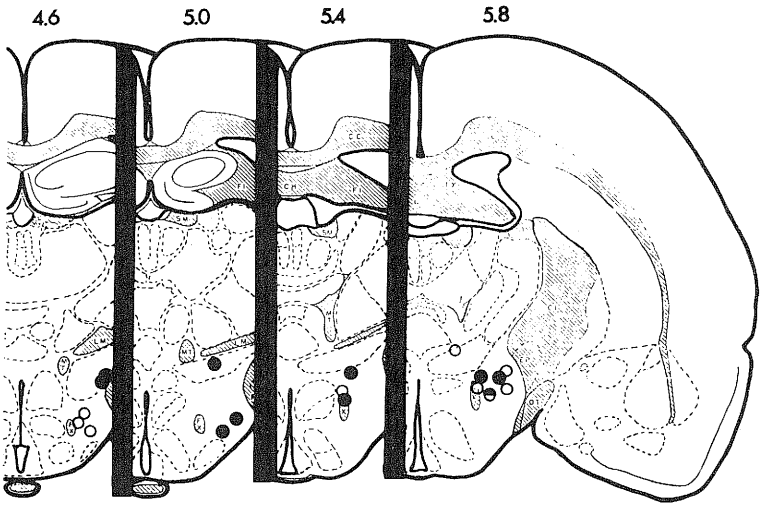

Figure 1. Locations of the electrode tips for the signaled-ESB group (filled circles) and for the immediate-ESB group (open circles) as plotted on drawings from de Groot (1959). (Numbers above each section indicate the distance anterior to the interaural line when the incisor bar is set at 5.0 mm above the interaural line.)

ESB revealed that 6 of the placements were in the lateral hypothalamus (LH), lateral or dorsolateral to the fornix in the area of the medial forebrain bundle (MFB), 2 were in the dopaminergic–ventral noradrenergic (DA-VNA) bundle (according to the atlas of Jacobowitz and Palkovits, 1974), 1 was directly dorsal to the fornix, and 1 was in the zona incerta (ZI). Nine of the 10 electrodes were therefore in areas containing catecholaminergic (CA) fibers, and the ZI electrode was probably stimulating a cholinergic substrate.

For Group I-ESB, one brain was lost; electrode placements of the other nine are shown in Figure 1. Seven electrodes were in the LH, lateral or dorsolateral to the fornix in the area of the MFB, one was dorsal to the fornix, and one was dorsomedial to the fornix in ZI. Thus, eight electrodes were in areas containing CA fibers, and the ZI placement was probably cholinergic.

During the various analyses that follow, an attempt was made to determine whether there was any consistent difference in the performance of the rats with ZI electrodes or of the two S-ESB rats with DA-VNA bundle placements as compared with their groups. No consistent differences were found, however. There were two rats in the S-ESB group and one rat in the I-ESB group that never ran on the first trial of a session. Two of these rats had electrodes in CA areas, and one was the ZI rat in the S-ESB group; the ZI rat in the I-ESB group did run on the first trial of every session, however, so it does not appear to be possible to relate the anomalous first-trial performance of these three rats to their electrode loci.

Runway Performance

The running time for each trial for each rat was converted to a speed by dividing that number into 100. These data were then subjected to two major analyses. The first was a four-way analysis of variance which looked at overall performance with groups, order of presentation of ITI conditions, ITI conditions, and sessions as the variables analyzed. The second looked within sessions at changes in running speed from trial to trial.

Overall performance. For this analysis, only the last 9 of the 10 sessions under each ITI condition were used in order to allow 1 session as a transition day from one ITI condition to another or as an additional training day. A mean speed for each rat for each session was obtained by averaging the speeds from the seven fastest trials; this was done to eliminate

warm-up effects (see within-sessions analysis below). Thus, the data used consisted of 18 numbers for each rat in each experimental group.

Prior to running the main analysis, a separate four-way analysis of variance was performed on Group HOH, with lever pressers and nonpressers as two levels of the group variable. This analysis revealed that the lever pressers ran faster than the nonpressers, $F(1, 4) = 10.04$, $p < .05$. This group difference did not, however, interact with order, $F(1, 4) = 2.71$, $p > .05$, ITI, $F(1, 4) = .07$, $p > .05$, or sessions, $F(8, 32) = 1.05$, $p > .05$. The failure of any interaction to be significant means that the effects of these independent variables on the lever pressers and nonpressers were similar, and the two subgroups of Group HOH were therefore combined for the purposes of further analysis.

The main analysis of variance revealed that the groups differed, $F(3, 26) = 4.04$, $p < .017$; the order of presentation of ITI conditions was insignificant, $F(1, 26) = 0.79$, $p > .05$; there was an effect of ITI, $F(1, 26) = 17.59$, $p < .001$; and there was an effect of sessions, $F(8, 208) = 5.20$, $p < .001$. Since there was little effect of order, the data were combined over this variable, and the group means are shown in Figure 2 with sessions combined into three three-session blocks. There were only two significant interactions in the entire analysis: The effect of ITI on running speed was different among groups (i.e., ITI and groups interacted), $F(3, 26) = 25.64$, $p < .001$, and there was an ITI × Sessions × Order interaction, $F(8, 208) = 2.10$, $p < .038$. This latter interaction occurs when groups are combined. Since an ITI × Order interaction is equivalent to a difference between first and second blocks of nine sessions, the triple interaction can be taken to mean that this difference itself differed over sessions. This interaction has little meaning and is not considered further.

The sessions effect, although significant, failed to interact with either groups or ITI, $F(24, 208) = .97$, $p > .05$ and $F(8, 208) = 1.78$, $p > .05$, respectively. This means that the sessions effect was similar

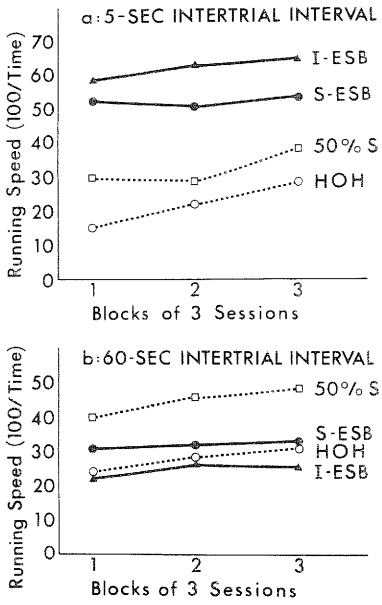

Figure 2. Mean running speed for each group in blocks of three sessions during the last nine sessions of the 5-sec intertrial interval (ITI) condition (a) and of the 60-sec ITI condition (b). (Abbreviations: I-ESB = immediate electrical stimulation of the brain; S-ESB = signaled ESB; 50%S = water deprived, with sucrose reinforcement; HOH = water deprived, with water reinforcement.)

for all groups under both ITI conditions, and Figure 2 certainly suggests this. All groups showed a general increase in running speed over sessions in both ITI conditions. The remaining group effect, the ITI effect, and their interaction are the results of principal interest, and these effects are more intensively examined below with post hoc tests.

Tests of simple main effects (Winer, 1962, pp. 311ff) comparing running speed during the 5-sec ITI condition with running speed during the 60-sec ITI condition *within each group* revealed that the ESB groups ran faster during 5-sec ITI: $F(1, 26) = 24.35$, $p < .001$ for S-ESB and $F(1, 26) = 84.17$, $p < .001$ for I-ESB. On the other hand, there was no significant effect of ITI on running speed for the water group, $F(1, 26) = 1.65$, $p > .05$, and the sucrose group actually ran faster during sessions with a 60-sec ITI than during 5-sec ITI sessions, $F(1, 26) = 7.33$, $p < .025$. These comparisons, to some extent, clarify

the source of the interaction observed in the overall analysis of variance. It is clear that the effect of ITI on running speed depended on the group being tested.

Further tests were done to examine more closely the interaction of groups with ITI for pairs of experimental groups.[1] The first test revealed that there was an interaction between groups and ITI for the two ESB groups, $F(1, 26) = 8.99$, $p < .01$. For each of the ESB groups, there was a Groups × ITI interaction in comparisons with the water group: $F(3, 26) = 18.14$, $p < .005$ for S-ESB and $F(3, 26) = 50.69$, $p < .001$ for I-ESB. Similarly, there was a Groups × ITI interaction in comparisons of each of the ESB groups with the sucrose group: $F(3, 26) = 28.43$, $p < .001$ for S-ESB and $F(3, 26) = 63.97$, $p < .001$ for I-ESB. Finally, there was no significant Groups × ITI interaction in the comparison of the water and sucrose groups, $F(3, 26) = 1.45$, $p > .05$.

These main effects and interactions can be summarized as follows: (a) Both ESB groups ran faster with the shorter ITI, but the difference between 5-sec ITI and 60-sec ITI running speed for the signaled ESB group was smaller than that difference for the immediate-ESB group, i.e., the signal attenuated the ITI effect in the S-ESB group; (b) the HOH and 50%S groups tended to run or ran faster with the longer ITI, and thus ITI had a different effect on the running speed of the ESB groups than it had on the running speed of the sucrose and water groups; and (c) the difference between 5-sec ITI and 60-sec ITI running speed for the water group was not significantly different from that difference for the sucrose group (i.e., the effect of ITI on running speed was similar for the sucrose and water groups).

All post hoc analyses up to this point have dealt with the differential effect of ITI within groups or with a comparison of the ITI effect from group to group (interactions). Additional post hoc examinations of the data which were of some interest were tests of simple main effects of groups within each ITI condition. Some of these comparisons were not particularly meaningful since, for example, no attempt was made to equate reinforcer magnitude for Group S-ESB to that for Group HOH under fixed ITI conditions. However, some differences were meaningful; the sucrose group, for example, might have run faster than the water group. These tests looked for these differences.

A test of simple main effects of groups during the 5-sec ITI condition revealed that the running speeds of the groups differed, $F(3, 26) = 14.99$, $p < .001$. Subsequent individual comparisons (Scheffé tests) showed that the two ESB groups did not differ significantly from each other, $F(3, 26) = 2.75$, $p > .05$, but both differed from the water group: $F(3, 26) = 20.31$, $p < .005$ for S-ESB and $F(3, 26) = 36.82$, $p < .001$ for I-ESB. Group I-ESB differed from the sucrose group, $F(3, 26) = 18.43$, $p < .005$, but Group S-ESB did not, $F(3, 26) = 8.17$, $p > .05$. The water and sucrose groups did not differ significantly in running speed during the 5-sec ITI condition, $F(3, 26) = 1.51$, $p > .05$.

The test of simple main effects of groups during the 60-sec ITI condition approached but failed to attain significance, $F(3, 26) = 3.26$, $.05 < p < .10$. Although it is necessary to be extremely cautious about drawing any conclusions from a failure to reject the null hypothesis (which was, in this case, that groups do not differ during the 60-sec ITI condition), this finding, when considered in conjunction with the previously observed large main effect of groups during 5-sec ITI, is particularly interesting because it suggests that the major condition that contributed to the observed differences between groups was the 5-sec ITI condition.

[1] A test of the interaction of ITI with the ESB groups was one of the original tests planned in the preparation of this experiment; for this reason the F ratio for this interaction was tested against an unadjusted critical F value based on degrees of freedom of 1 and 26 rather than against the Scheffé adjusted critical F which, in this case, would be three times the unadjusted value for degrees of freedom of 3 and 26. This latter critical value of F should be (and was) used in testing the significance of F ratios for interactions in all other individual comparisons (Scheffé, 1960). Incidentally, the ESB Groups × ITI interaction is significant even when the Scheffé adjusted critical F is used.

Effects of trials within sessions. Intrasession data consisted of 10 running speeds, 1 for each trial. For each rat, the running speeds for each trial during the last three sessions in each ITI condition were averaged. The group means of these data are presented in Figure 3. These scores were combined into five trial-blocks of two trials each for the purpose of statistical analysis.

A separate analysis of variance was done for each ITI condition. During the 5-sec ITI condition, the groups differed, $F(3, 30) = 8.58$, $p < .001$; running speed changed over trials, $F(4, 120) = 68.27$, $p < .001$; and groups and trials interacted, $F(12, 120) = 4.26$, $p < .005$. Tests of simple main effects, done to clarify the source of the interaction, showed that all four groups showed a trials effect: $F(3, 144) = 26.50$, $p < .001$ for S-ESB; $F(3, 144) = 44.11$, $p < .001$ for I-ESB; $F(3, 112) = 3.44$, $p < .025$ for HOH; and $F(3, 80) = 6.90$, $p < .001$ for 50%S. Since the shape of the trials effect was similar for all groups, the interaction must mean that the group differences differed from trial to trial.

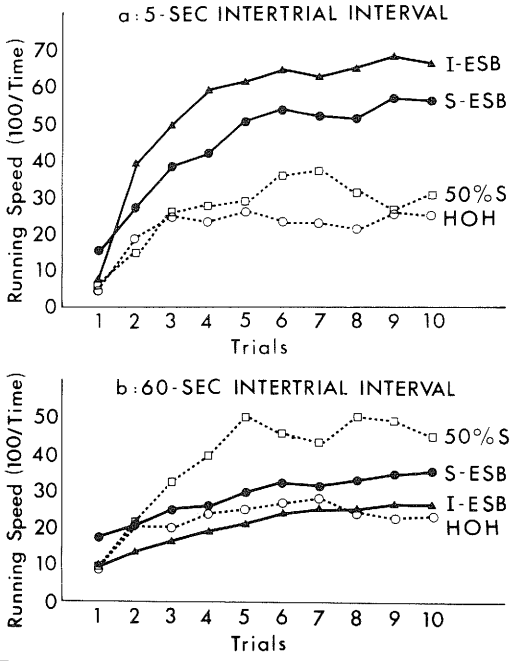

Figure 3. Group means of running speeds on each trial averaged over individual trials in the last three sessions in the 5-sec intertrial interval (ITI) condition (a) and in the 60-sec ITI condition (b). (Abbreviations: I-ESB = immediate electrical stimulation of the brain; S-ESB = signaled ESB; 50%S = water deprived, with sucrose reinforcement; HOH = water deprived, with water reinforcement.)

During the 60-sec ITI condition, the groups differed, $F(3, 30) = 13.06$, $p < .001$; there was a trials effect, $F(4, 120) = 36.80$, $p < .001$, and an interaction, $F(12, 120) = 3.14$, $p < .001$. Tests of simple main effects showed that there was a trials effect for three groups: $F(3, 144) = 5.74$, $p < .001$ for S-ESB; $F(3, 144) = 5.03$, $p < .001$ for I-ESB; and $F(3, 80) = 15.25$, $p < .001$ for 50%S, but for the HOH group the trials effect failed to reach significance, $F(3, 112) = 1.98$, $p > .05$. The HOH group appeared, from Figure 3, however, to be showing a trend similar to the other three groups for the first seven trials but, unlike these groups, seemed to slow down for the last three. As in the 5-sec ITI condition, the shape of the trials effect was similar for all four groups, and the interaction must therefore be attributable to differences in group differences from trial to trial.

A summary of the analyses of trial effects is basically that within both ITI conditions, all groups (with the possible exception of Group HOH in the 60-sec ITI condition) ran at an initially and similarly slower speed on the early trials of a session than on the later trials. Although the groups reached different asymptotes of performance, the data showed clearly that this overnight decrement in runway behavior was a general phenomenon and occurred in every group.

Figure 3 shows that running speed on the first trial was slow for all groups but gives no information about the number of times rats ran or refused to run. Table 1 shows the percentage of rats from each group that never ran on any of the 20 first-trials and the percentages that ran 1–5 times, 6–10 times, etc. The table shows that 20% (two rats) of Group S-ESB and 10% (one rat) of Group I-ESB never ran but 37.5%–50% of the rats in every group ran 16–20 times on the 20 first-trials.

One final observation should be stressed: No rat in either of the ESB groups ever had to be primed. On some occasions some rats failed to run, and as already men-

Table 1
Percentage of Rats That Ran on the First Trial of the 20 Sessions

No. of sessions out of 20	Group			
	S-ESB (10)	I-ESB (10)	HOH (8)	50%S (6)
0	20	10	–	–
1–5	–	30	–	–
6–10	20	10	25	33.3
11–15	10	10	37.5	16.7
16–20	50	40	37.5	50

Note. Each group *n* is given in parenthesis. Abbreviations: S-ESB = signaled electrical stimulation of the brain; I-ESB = immediate ESB; HOH = water deprived, with water reinforcement; 50%S = water deprived, with sucrose reinforcement.

tioned, three rats never ran on the first trial; in no case, however, was it ever necessary to prime a rat once it had been pushed into the goal box. Every rat initiated lever pressing on its own once it reached the goal box area.

Discussion

The results can be summarized as follows: (a) Nondeprived rats reinforced with ESB ran faster with 5-sec ITIs than with 60-sec ITIs; (b) water-deprived rats reinforced with sucrose or water tended to run faster with 60-sec ITIs; (c) the ESB, sucrose, and water groups all showed overnight decrements in running speed; and (d) the ITI effect for the signaled-ESB group was significantly smaller than for the immediate-ESB group.

The observed ITI effect for the ESB groups replicated earlier reports by Gallistel (1966, 1967), Panksepp et al. (1968), Reid et al. (1973), Seward et al. (1960), and Spear (1962). It might be argued that amount of handling may be confounded with the ITI effect since this factor varied with variations in ITI. Specifically, during 5-sec ITI trials the rat is handled only once (picked up from the goal box and placed in the start box), whereas during 60-sec ITI trials the rat is handled twice (picked up and placed in the home cage and picked up and placed in the start box). This same criticism applies to Gallistel (1967), Panksepp et al. (1968), Seward et al. (1960), and Spear (1962) all of whom used this procedure; Gallistel (1966) and Reid et al. (1973) did not report the details of handling. On the surface it appears that a less confounded procedure would be to briefly place the rat in its home cage during a 5-sec ITI trial and thereby equate amount of handling for 5-sec and 60-sec ITI trials. We contend that this procedure would be similarly, and possibly more, subject to criticism on the basis of the same handling variable. With the procedures employed here and in earlier experiments, every trial is immediately preceded by about the same amount of handling, whereas with the suggested procedure, short-ITI trials would be explicitly immediately preceded by more handling than long-ITI trials. If whatever change produced by handling decays over time since handling, then clearly the procedure usually used in experiments of this kind is the better of the two discussed. It is clear, however, that an empirical investigation of the handling question is needed.

The explanation given by Gallistel (1973, pp. 208ff) for the ITI effect with ESB reinforcement was that the vigor of a response depends on a rapidly decaying "drive" induced by ESB. "Drive" refers to activity in cells normally activated by some homeostatic imbalance or organismic need. Thus, the rats are running under a stronger drive 5 sec after receiving ESB than they are 60 sec after ESB and therefore run faster with the shorter ITI. Data from many investigations have shown that brain stimulation, in sites identical to those that support ESB, has the ability to produce eating (Hoebel & Teitelbaum, 1962; Margules & Olds, 1962), drinking (Mogenson & Stevenson, 1966), and sexual behavior (Caggiula & Hoebel, 1966; Herberg, 1963), which suggests that ESB can activate a drive substrate. On the other hand, the electrical stimulation used to produce eating, drinking, and sexual behavior often consisted of much longer trains than those typically used for ESB reinforcement. Furthermore, not all ESB electrodes can be shown to produce stimulus-bound consummatory behaviors (Margules & Olds, 1962; Mogenson & Stevenson, 1966). These considerations sug-

gest that, at least in some cases, ESB does not activate a drive substrate at all.

Trowill et al. (1969) suggested that the differences between performance for ESB and that for conventional reinforcers can be resolved by regarding ESB as an incentive, but they do elaborate specifically on the way this might result in faster running with shorter ITIs. Incentive theories (Bindra, 1976; Bolles, 1972; Milner, 1970, 1976) are based on the assumption that an animal *expects* a reinforcing stimulus as a result of making a particular response. This expectation occurs when, in the brain of the animal, the neural activity representing the response acquires associations with neural activity representing the reinforcing stimulus. Milner (1976) suggested that this neural representation of the reinforcing stimulus is not itself reinforcing but that it becomes connected, because of contiguous firing, with a subcortical system that is. The subcortical reinforcement system consists of cells that, when fired, provide general facilitation of response activity.

In order for an expectancy to be effective in producing a response, response activity has to be linked to response facilitation through at least two sets of associative connections, one between the response cells and a neural representation of the reinforcing stimulus and one between that neural representation and a subcortical response-facilitating system. If either link or both links weaken, the vigor of the response is reduced.

Many data suggest that associative connections are strongest immediately after being activated and decay with time when they are not used (e.g., see Mackintosh, 1974, pp. 162 ff). Thus, any response resulting from an expectation should weaken over time, regardless of the reinforcer. The observation of a consistent overnight decrement in running for water and sucrose as well as for ESB confirms that such an effect exists. It should be possible to reduce the decrement by preexposing an animal to the reinforcing stimulus before the first trial; the findings by Bruce (1938) that pretrial presentations of food to food-deprived rats or of water to water-deprived rats resulted in faster running and by Gray (1976), Hoffman, Flesher, and Chorney (1961), and Spear, Gordon, and Martin (1973) that pretrial shock increased the rate of avoidance responding support this view.

If ESB reinforcement immediately drove a response-facilitating system to a high level, which then began to decay, whereas water and sucrose reinforcement had a more slowly rising response-facilitating effect followed by a decay, then both the observed differences in ITI effects and the similarities in overnight decrements would be expected. These differences in response-facilitating effects may be due to the nature of the ESB stimulation. It is well known that tetanic electrical stimulation has a potentiating effect on synaptic strength. According to Lloyd (1949), a 1-sec burst of stimulation potentiates the monosynaptic spinal reflex by as much as three times, and the reflex returns to normal in about 1 min. This time course is similar to that of ESB reinforcement and encourages speculation that posttetanic potentiation may be a factor in exaggerating the ITI effect when the reinforcement consists of tetanic bursts of electrical stimulation.

There is another possible interpretation of the cause of the ITI effect in the 50%S group: That is that a sucrose solution at a concentration as high as that used here is less preferred when presented in discrete amounts at short intervals than when presented at longer intervals. This possibility cannot be refuted from the present data and should be tested empirically by observing the ITI effect demonstrated by groups of rats run at different values along a range of sucrose concentrations. Nevertheless, the water group in this and in Gallistel's (1967) experiment showed a tendency toward faster running with the longer ITI, and the above speculation regarding the cause of this tendency and of a reverse ITI effect in the ESB groups is not subject to the same criticism as the sucrose group.

The observation that delaying the onset of ESB by .5 sec and preceding it with a signal was a sufficient condition to signifi-

cantly attenuate the ITI effect for ESB-reinforced rats in the runway has not been reported previously. This influence on the ITI effect may be due to another specific consequence of ESB. We have to ask ourselves what it is that the animal expects as a result of the association of the neural activities representing the response and the reinforcing stimulus. The animal may expect, in the case of water, for example, the sight and sound of a water dipper, and later the taste of water, all of which expectancies are associated with the response-facilitatory effects of reinforcement. In the case of immediate ESB reinforcement, however, what the animal expects may be a highly abnormal form of brain activity consisting of sensory input distorted by the electrical stimulation. We suggest that the animal may have difficulty in forming an expectancy of this peculiar activity except immediately after it has occurred, much as we have difficulty remembering a word in a strange language for longer than a few seconds after it has been heard. If this is the case for immediate ESB, response cells may not be able to elicit the complete neural representation of the reinforcing stimulus associated with the response, and this results in less activation of the response-facilitation cells that are required to produce a vigorous response.

If instead of immediate ESB the response is followed by a click and other normal forms of sensory stimulation, the animal is able to establish expectancies in the usual way with these stimuli. When the ESB is presented .5 sec later and fires the reinforcement system, the neural representations of the click and other sensory stimuli become associated with the reinforcement cells (i.e., the response-facilitatory system). The .5-sec interval is close to the optimum for associating conditioned and unconditioned stimuli (cf. Mackintosh, 1974, pp. 62–66). To some extent, therefore, the combination of delaying and signaling the ESB allows the animal to form associations between the response and the reinforcing stimulus that bypass the abnormal brain activity produced by the ESB. Our data on the interaction between ITI effect and mode of delivery of ESB suggest that for a few seconds after the ESB it is advantageous to have associations form between the response and the (abnormal) neural activity produced by the ESB but that after longer intervals ESB-produced activity is poorly recalled by association and it is better to have formed the association to some familiar intermediary such as a click.

This interpretation ignores the possibility that the attenuation of the ITI effect in the S-ESB group was caused not by the signal but simply by the delay, which was confounded with the signal. If it was shown that a group of rats receiving delayed ESB demonstrated a smaller ITI effect than a group receiving immediate ESB, then this theory of the role of the signal would be less tenable. However, other data are consistent with this interpretation of the role of a signal for ESB reinforcement. Beninger, Bellisle, and Milner (1977) recently demonstrated that neither signal nor delay was necessary for maintaining lever-press responding on an interval schedule in a Skinner box. They did observe, however, that of three groups, one with signaled ESB, one with nonsignaled but delayed (.5 sec) ESB, and one with immediate ESB, only the signaled-ESB group showed a significant session to session improvement in latency to the first response at the beginning of a session. This observation might mean that the expectancy for ESB reinforcement was better established in the signaled-ESB group.

What we are suggesting here is that there are two distinct systems that are involved in producing behavior. One of these, traditional "drive," arises from a homeostatic imbalance or organismic need and has a tonic activating effect on behavior. The other is an acute (i.e., short-term) response facilitation which results from the presentation of reinforcing stimuli. Deutsch and Howarth (1963) and Gallistel (1973) never made this distinction and suggested that the ESB reinforcement produced vigorous responding because the ESB stimulus activated not only a reinforcement system in the brain but also a drive system. However, as mentioned ear-

lier, some data suggest that it is possible that ESB reinforcement does not always activate a drive system. We suggest that all reinforcing stimuli produce an acute response facilitation – and our data support this position – but that ESB reinforcement produces more rapidly a more vigorous response facilitation than that produced by conventional reinforcers.

To account for the fact that there was running on the first trial for ESB, there must also be a more enduring consequence of this type of reinforcement. The two ESB groups in this experiment were maintained on ad lib food and water, yet 17 of the 20 rats ran on the first trial of a session at least some of the time and 9 of them ran on the first trial at least 75% of the time. Beninger et al. (1977) observed that rats with a history of ESB reinforcement initiated responding more rapidly than control rats even after months without exposure to ESB. These findings suggest that the expectancy which results from a history of reinforcement is alone sufficient to produce at least a low level of responding, even in the absence of a known "drive." This is not a new idea (cf. Morgan, 1974). If reinforcement occurs artificially, for example by ESB, this low level of responding is facilitated even though no "drive" has been reduced. Some addictions may come about through this mechanism.

The findings from this study strongly suggest that caution should be used in theorizing about the function of the neural substrate stimulated by ESB electrodes. Since responding for ESB is now being extensively used for the evaluation of drug effects (Wauquier & Rolls, 1976) and the findings are being used as the basis for theories of reinforcement and motivation, it is important that the similarities of the behavioral effects of ESB reinforcement to those of conventional reinforcers be considered as fully as possible.

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