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PROCEEDINGS OF THE
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1969
Volume 4

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FOREWORD

This edition, the fifth of APA's *Convention Proceedings*, comes close to representing the full range of offerings by psychologists participating in the annual convention. The 1965 volume represented participation by five divisions; the 1969 edition represents participation by 25 divisions and the Board of Professional Affairs. Thus it has become apparent that the proceedings experiment, started in 1965 by the Project on Scientific Information Exchange in Psychology and supported by the National Science Foundation, has become an established improvement in the dissemination of convention information.

The rapid growth of participation by APA divisions presented a problem in the production of the 1969 edition. The 1968 volume weighed over three pounds and measured one and a half inches thick. It quickly became evident that the papers anticipated from 10 additional divisions in 1969 would necessitate two volumes and an increase in price which might place the *Proceedings* beyond the reach of many interested persons. The solution adopted was to package the material by division and offer these packages (paper summaries) at a reasonable price. Members now have the options of ordering one division package, or several, or the entire collection bound in two volumes. The Convention Committee, the Publications Board, and the Board of Directors concurred in this approach in the hope that such a method of distribution would make the *Proceedings* available to the widest possible audience at a reasonable price and increase the selectivity of material in accordance with individual needs and interests.

Perhaps the greatest advantage in the *Proceedings* is its availability in advance of the meeting. This year's volumes are scheduled to be available late in July. Thus it is now possible for the annual convention to permit more extensive and informative interactions, both in paper-reading sessions and in symposia, by authors and audiences.

It is hoped that in the future the *Proceedings* will become even more fully integrated with APA's total system for communicating information responsive to the diverse needs of persons interested in psychological information.

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Heart Rate Conditioning

Table 1 shows that HR acceleration produced by subcortical ESB was conditioned to a previously neutral tone, after tone-ESB pairings. In addition, HR acceleration to the tone extinguished rapidly when ESB was no longer paired with the tone. Again, neither ICSS nor escape responses could be obtained from any *S* with ESB at the location that produced the UR.

TABLE 1
Mean Change in Heart Rate at Different Stages of Conditioning

S	US alone	CS alone (habituation)	CS after pairing (conditioning)	Trial	CS alone (extinction)
L	25.2*	1.2	19.6*	55-60	3.6
P	24.4*	0.7	15.4*	75-80	1.8
M	28.0*	1.6	16.8*	35-40	3.8
J	15.6*	1.8	11.0*	35-40	2.0

* $p < .01$.

Skeletal-Motor Conditioning

There was no indication of any conditioning of the ESB-elicited skeletal-motor response after 300 CS-US pairings (Condition 1). However, when ESB at a rewarding location followed CS-US pairing (Condition 2), signs of skeletal-motor conditioning began to appear after approximately 50 trials. The CR differed slightly for each *S* in its early stages, but consisted basically of mouth-opening without vocalization, along with head movements towards the side of the US. As training with rewarding brain stimulation continued, the tone eventually elicited a muffled type of vocalization (different from the barking type elicited by the US), as well as occasional ear and eyebrow movements. Head movement in the direction of the US diminished with continued training, but was still observable when Condition 2 was terminated. Therefore, although the tone-elicited CR contained components of the UR at the end of Condition 2, the two responses were still distinguishable. When Condition 3 was presented, the CR completely disappeared after approximately 40 trials.

Histology

Histology has been completed on the brains of two *Ss*. In both *Ss* the tip of the electrode used for eliciting the UR was located in the region between the inferior and superior colliculi at the level AP O. In one *S*, the tip of the rewarding electrode was located in the lateral septal region, and in the other *S* it was located in the region of the anterior medial forebrain bundle.

The results of this experiment suggest that (a) external reward is unnecessary for conditioning HR changes elicited by subcortical brain stimulation, and (b) an additional form of reinforcement, e.g., ESB at a rewarding locus, is necessary for conditioning skeletal-motor responses elicited by subcortical brain stimulation. These results are also in line with those of other conditioning experiments where autonomic CRs have been found to develop earlier than motor CRs (Banks, Miller, & Ogawa, 1966; Graham, Cohen, & Shmavonian, 1964), and suggest that conditioning is based on a hierarchical system of neural events.

Such an interpretation of the data from the present experiment, however, is limited by the possibility that (a) tone-elicited HR acceleration was secondary to undetectable skeletal-motor changes and/or respiratory changes, and (b) tone-elicited skeletal-motor responses might have been obtained without the addition of rewarding ESB if the *Ss* had been given more CS-US pairings without reward. Although these possibilities cannot be completely ruled out, there is some evidence to suggest that they are unlikely. First, with regard to *a*, the *Ss* were lightly tranquilized during HR conditioning procedures and, consequently, movements were minimal. Furthermore, when any movement did occur during tone presentation, it usually began prior to the onset of the tone. Second, with regard to *b*, the CR elicited by the tone in Condition 2 disappeared when CS and US were again paired without the additional rewarding ESB (Condition 3).

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EFFECT OF FOREBRAIN LESIONS ON ACQUISITION AND RETENTION OF ONE-TRIAL LEARNING IN CHICKS¹

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Precocial newly hatched chicks are well suited for the study of neurophysiological bases of behavior because of their availability and ease of maintenance, learning ability (Zolman, 1968), immature blood-brain barrier (Hehman, Vonderahe, & Peters, 1961), etc. In earlier experiments we have studied their memory processes by administering transcranial electroshocks of various intensities before or after a learning trial, followed by retention tests (e.g., Lee-Teng, 1968, 1969; Lee-Teng & Sherman, 1966). The present experiment was conducted as a preliminary step to localize the parts of the brain that were critically involved in the learning and memory processes. Different surface areas of the forebrain were aspirated, either before or after the administration of one-trial learning, in independent groups of chicks. Retention tests were given later to assess the effects of the lesions.

METHOD

Day-old White Leghorn cockerels obtained in the mornings from a nearby hatchery were used. They were housed individually in small cartons in a warm experimental room during the 3-day experiment. No food or water was provided.

In Exp. I, surgery was performed on Day 1, between 9 a.m. and 3 p.m.; learning trial was administered on Day 2, around 3 p.m.; and retention was tested on Day 3, around 10 a.m. In Exp. II, learning trial was given on Day 1, around 3 p.m., and a retention trial was given on Day 2, around 9 a.m. About 20% of the chicks did not show retention and were discarded. For *Ss* that had shown retention, surgical or control treatments were performed on Day 2, and retention was further tested on Day 3, around 10 a.m.

In each experiment, there were 5 surgical or control conditions: anesthesia-only, sham operation, medial lesion, side lesions, and total lesion. Each *S* in the Anesthesia-only group received .09 ml. of Equithesin injected intraperitoneally, but had no surgery. For all the other *Ss*, the head was held stable on a stereotaxic device while under anesthesia, and the dorsal surface of the forebrain was exposed. The *Ss* in the Total group had the entire dorsal surface removed to the depth of about 1.5 mm. by aspiration. The Medial group had lesion of the medial strip, about 1/3 of the surface. The Side group had lesions of the complementary 2/3 of the surface. For *Ss* in the Sham group, the sinus was nipped to induce similar extent of bleeding as happened in the lesioned groups, but no brain tissue was removed. Three percent of the *Ss* died of anesthesia without surgery. Among the *Ss* that had surgery, the death rate was 5%, most of them during operation. Four *Ss* randomly selected from each group were perfused after the retention tests, and their brains were removed and grossly examined. No brain lesion was observed in the Anesthesia-only and Sham groups, and the wounds in the other groups were as expected.

The one-trial learning involved suppression of the chick's predilection to peck at a small shiny ball by coating it with a bad-tasting chemical. Typically, after one peck at the lure during the learning trial, the *S* would not peck again. Detailed procedure has been described (Lee-Teng, 1969). During the test trial, the same lure was presented to each *S* for 5 sec. and whether it pecked or not was recorded. The *E* could tell if the *S* had operation by the presence of sutures. The *E* could not distinguish, however, among the Sham-operated and the three lesioned groups.

In order to check specifically lesions' effect on acquisition, *Ss* that had pecked at the lure during the first Day 3 test trial were given a second test trial about 10 min. later.

RESULTS AND DISCUSSION

Base-line Pecking and Head-Shaking Responses

Presentation of the lure during the learning trial generally elicited high incidences of pecking followed by vigorous shaking of the head. The percentage of chicks that failed to peck within 5 sec. and shake their head within 10 sec. after pecking (the "no-peck" percentage) was 14% on Day 1 (Exp. II) and 4% on Day 2 (Exp. I), and these chicks were discarded. It is important to note that the lower no-peck percentage on Day 2 was not caused by Day 1 surgical or control treatments. The same low Day 2 percentage has been obtained repeatedly from samples of large numbers of normal chicks, and the percentage is further lowered to 2% on Day 3 among normal chicks. In addition, despite the different Day 1 surgical or control procedures, there was no intergroup difference in either pecking or shaking latency during the Day 2 learning trial among the five groups in Exp. I.

Retention in the Control Groups

Without forebrain lesions, the majority of the *Ss* showed retention of the learning when tested on Day 3. The no-peck percentage rose sharply from the Day 3 base-line value of 2% to about 80%. The χ^2 test was used in the following data analysis to evaluate the significance of group differences. In Exp. I, the no-peck percentages were 83% ($N = 249$) in Anesthesia-only and 68% ($N = 31$) in Sham-operated. The difference between the two groups was not significant at the .05 level, and the pooled results gave a combined control value of 81%. In Exp. II, the no-peck percentages were 86% ($N = 123$) for Anesthesia-only and 88% ($N = 33$) for Sham-operated. Difference between these two groups was also not significant, and the pooled results gave a combined control value of 87%.

The lack of significant difference between the Anesthesia-only and Sham-operated groups in both experiments indicated that exposing the forebrain, plus nipping the sinus

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to induce bleeding, did not affect S's response during the Day 3 retention test.

In Exp. I, all Ss received the learning trial on Day 2 and the retention test on Day 3. In Exp. II, the learning trial was given on Day 1, followed by a retention test on Day 2, and only those Ss that showed retention were kept. The preselection of Ss in Exp. II, plus the different timing of the learning trial between the 2 experiments, nevertheless did not vary the results of the Day 3 retention test. For both the Anesthesia-only and the Sham-operated groups, the differences between Exp. I and II were not significant at the .05 level.

Retention in the Experimental Groups

Results from the first Day 3 retention test are presented in Fig. 1. Clearly, both the type of lesions and the timing of surgery were important variables affecting Day 3 results. In both experiments, the no-peck percentage decreased as the area of lesion increased. Further, for each type of lesion, the no-peck percentage was lower in Exp. I, where surgery was performed on Day 1, before the learning trial, than in Exp. II, where surgery was performed on Day 2, after the learning trial, among Ss that had demonstrated retention. For both Exp. I and II, the differences among the control and the experimental groups were significant at the .001 level. The differences between Exp. I and II were significant at the .01 level for Total lesion, and at .05 level for both the Medial and the Side lesions.

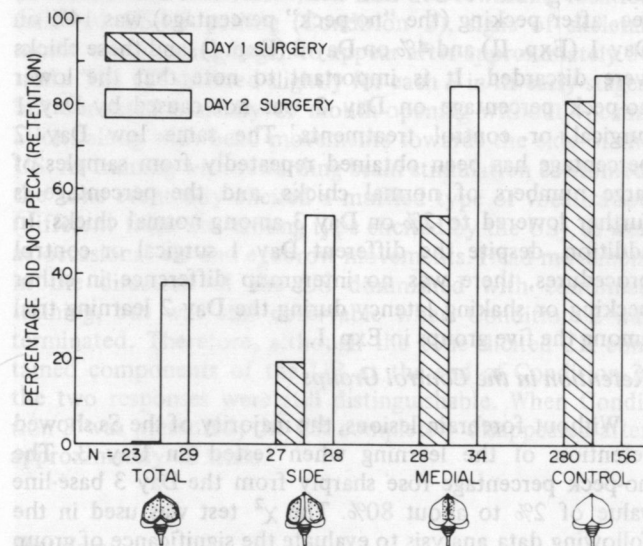


Fig. 1. Percentage of Ss that did not peck during the first Day 3 test trial.

In Exp. II, since surgery was performed after the learning trial, retention deficit in the lesioned groups might have been explained in terms of dissociation. According to this interpretation, the lesioned areas were not centrally involved in retention. Rather, lesions changed the general background state of the Ss, which in turn affected their response during the retention test. This interpretation is rejected because clearly more retention deficit was observed in Exp. I where no change-of-state was involved between the learning and retention trials, as the learning trial was given after the surgery.

The lower no-peck percentages obtained when surgery was performed before the learning trial suggested that the lesioned areas were involved in not only retention, but also acquisition. This interpretation was supported when the investigators analyzed the response of the Ss that had pecked during the first Day 3 retention test and were given a second test trial about 10 min. later. Pecking during the first test trial gave the Ss a chance to learn; testing 10 min. later was more a measure of acquisition than a measure of long-term retention. The no-peck percentage during the second test was much higher in the Sham and Medial groups than in the Side and Total groups, but showed neither consistent nor significant differences between Day 1 and Day 2 surgery. Pooling Ss with Day 1 or Day 2 surgery, the no-peck percentage was 81% for the Sham and Medial groups combined, and 40% for the Side and Total groups combined, and the difference was significant at the .01 level.

To sum up, results from the present study indicated that the dorsal surface of the forebrain was critically involved in both acquisition and retention of the one-trial learning, and deficit in performance was proportionate to the size of the lesion. Another possible interpretation of the data was that the structures at the sides of the forebrain surface were more importantly involved than those medially situated.

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L-NOREPINEPHRINE: A POSSIBLE SYNAPTIC TRANSMITTER FOR THE SUPPRESSION OF FEEDING BEHAVIOR BY SATIETY

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RESULTS

Feeding behavior is regulated by a medial hypothalamic satiety system (VMH) that inhibits a feeding system in the perifornical medial forebrain bundle (pmfB) (Stellar, 1964). Anatomical connections from the satiety system to the feeding system have been demonstrated by gold thioglucose lesions of VMH. The overeating produced by these lesions is due to denervation of synaptic terminals in the pmfB (Arees & Mayer, 1967). The pmfB contains very high densities of l-norepinephrine (l-NE) synaptic terminals (Fuxe, 1964). These l-NE terminals may mediate the suppression of behavior associated with satiety (Margules, 1969).

METHOD

Interperitoneally (ip) l-NE or l epinephrine (l-E) suppresses feeding (Russek, Stevenson, & Mogenson, 1968) and l-E selectively suppresses self-stimulation at brain sites that show electrically elicited feeding but not at brain sites that do not (Mogenson, Russek, & Stevenson, 1969). IP doses of amphetamine (A), a drug that releases l-NE from the brain (Carr & Moore, 1969), induces satiety for food and raises thresholds for electrically elicited feeding (Coons, 1964). Similarly, direct application of (A) to the MFB suppresses food intake in food-deprived rats (Booth, 1968). Conversely, direct application to the MFB of chlorpromazine (CPZ), a drug that blocks central alpha l-NE receptors, increases food uptake (Leibowitz, as reported by Miller, 1969) as do ip injections of CPZ (Reynolds & Carlisle, 1961). These considerations led to a reexamination of l-NE induced feeding and l-NE induced suppression of behavior in the pmfB (Grossman, 1962). Variable depth bilateral cannulas aimed for the pmfB were implanted into 7 albino Charles River rats. Thirteen of the 14 cannulas were in the pmfB or within .5 mm. of it. Ten additional rats not implanted with cannulas were used in experiments on ip administration of drugs. All rats were maintained in their cages on ad lib Purina Laboratory Chow and water. Seventy-two minute licking tests were conducted Monday to Friday in a Plexiglas response chamber containing a burette of milk (1 part Borden's sweetened condensed milk to 2 parts of water) and a water burette (never licked in this study). Each lick on the milk burette activated a drinkometer, banks of counters, and a Gerbrands cumulative recorder. Several weeks of practice licking were required to achieve stable satiety curves. Once stability was established, an experimental manipulation was performed not more often than once a week. In rats with cannulas, the first manipulation consisted of insertion of 1 mm. longer empty inner cannulas. The second and third manipulations were identical insertions with drugs in inner cannulas. Cannulas were loaded by tapping powdered compounds (three taps on a glass plate) into the inner cannulas. High doses consisted of two treatments of 15 and 2 min. prior to the test, and low doses of one treatment at 5 min. prior to the test.

Direct application of 19 μ g. of the alpha-noradrenergic blocker, phentolamine hydrochloride (PA), to the pmfB increased mean total milk licks from the pretreatment control of 2,690 to 4,426, and total milk consumption from 19.5 to 32.1 cc (Fig. 1). Both increases were statistically significant ($t = 3.87$, $df = 6$, $p < .01$ and $t = 5.28$, $df = 6$, $p < .01$). Such treatment also caused overeating of mash in one of one rat tested.

Nine μ g. of PA had similar statistically significant effects, which were smaller and shorter lasting (Fig. 1) ($t = 3.72$, $df = 3$, $p < .05$ and $t = 3.22$, $df = 3$, $p < .05$). Administration of this dose after satiation occurred (at the end of Period 5) reduced the PA induced increases to levels not significantly different from control ($t = .89$, $df = 3$ and $t = 1.22$, $df = 3$). On the day after application of PA, mean licks (2,167) (Fig. 1) and mean milk consumption (14.3 cc) were below pretreatment control. The decrease in mean licks was not statistically significant; however, mean milk consumption ($t = 4.74$, $df = 3$, $p < .05$) and rat weights on these days rebounded significantly below predrug control (554.7 vs. a mean control of 566.0 gm.) ($t = 3.37$, $df = 6$, $p < .05$).

Direct application of 28 μ g. of l-NE to the same sites decreased total milk licks from the mean pretreatment control of 2,540 to 1,146 (Fig. 1) and decreased total milk consumption from 17.7 to 7.7 cc. Both decreases were statistically significant ($t = 3.64$, $df = 6$, $p < .05$ and $t = 3.37$, $df = 6$, $p < .05$). Nine of 10 rats showed these decreases. Fourteen μ g. of l-NE had smaller effects (Fig. 1). Six micrograms of l-NE caused overeating in 1 of 3 rats tested. In this rat, PA (18 μ g.), increased milk consumption by 120% as compared to 40% for l-NE. Twenty-four hours of food deprivation attenuated the suppressant effect of direct application of l-NE on total milk licks ($t = 2.90$, $df = 6$, $p < .05$) and on total milk consumption ($t = 3.16$,

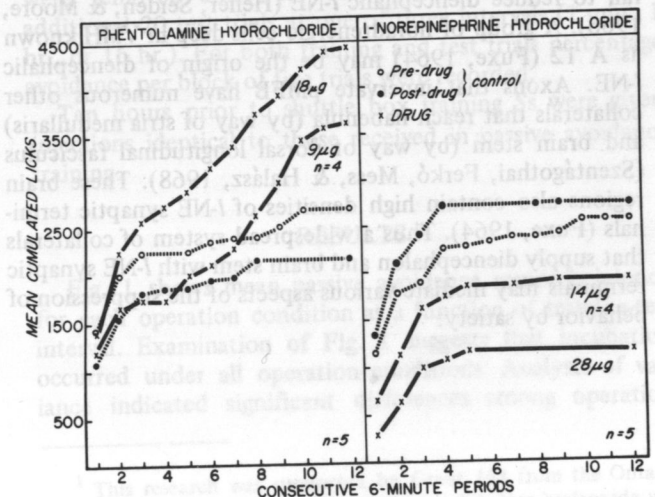


Fig. 1. Effects of direct application of drugs to the pmfB on milk licking behavior.