# THE EFFECTS OF INTERPEDUNCULAR NUCLEUS LESIONS ON ACTIVE AND PASSIVE AVOIDANCE RESPONDING IN THE RAT

by 1264

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B. A., Northern Illinois University, 1966

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A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Psychology

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Manhattan, Kansas

1969

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**THIS BOOK CONTAINS NUMEROUS PAGES WITH DIAGRAMS THAT ARE CROOKED COMPARED TO THE REST OF THE INFORMATION ON THE PAGE.**

**THIS IS AS RECEIVED FROM CUSTOMER.**

Several studies have recently suggested that the behavioral functions associated with total septal lesions are dissociable in terms of specific septal projections. The septum is reciprocally connected to the hippocampus via the fimbria and the dorsal fornix (Powell & Cowan, 1955; Raisman, Cowan, & Powell, 1966). In addition, it provides diencephalic connections ventrally along the medial forebrain bundle *en route* to the hypothalamus (Raisman, 1966) and dorsally to the habenular nuclei via the stria medullaris or to bypass the habenular group and terminate in the interpeduncular nucleus (IPN). Fischman and McCleary (1966) found that fornicotomy in cats failed to produce a passive avoidance decrement similar to that following septal lesions. More recently, Van Hoesen, MacDougall, and Mitchell (1969) found that active and passive avoidance responding were differentially disrupted by lesions to septal tracts. In their study, various septal fibers were selectively tractomized in order to evaluate their separate contributions to the avoidance responding typically observed with septally lesioned rats. They found that fornix lesions produced no disruption of passive avoidance responding, whereas an interruption of the stria medullaris--habenular complex led to both an enhancement in acquisition of active avoidance and a decrement of passive avoidance behavior.

Although some of the stria medullaris fibers circumvent the habenular nuclei and join the secondary habenulo-interpeduncular fibers to form an anatomical link *between the septum and* the habenular nuclei and the interpeduncular nucleus, there are few, if any, behavioral studies examining the functional significance of this connection. Anatomically, most studies suggest that the IPN is a principal recipient of diencephalic efferent fibers of the habenulo-interpeduncular tract (fasciculus retroflexus of

Meynert). Based on the available evidence from cats, rabbits, and opossums, the habenulo-interpeduncular tract originates in the lateral and medial habenular nuclei and courses postero-ventrally with residual stria medullaris fibers to terminate in the IPN (Akagi, 1967; Cragg, 1961; Mitchell, 1963). Secondary collaterals branch dorso-caudally then rise at right angles to either join the efferent pedunculo-tegmental tract en route to the midbrain tegmental nuclei (Nauta, 1958) or pass postero-ventrally into the raphe nuclei near the region of the superior central nucleus (Way & Kaelber, 1969). The reciprocally connected interpeduncular efferents project anterodorsally as a diffuse bundle and join the descending habenulo-interpeduncular tract at the level of the mammillary bodies. Also, at this level a few collaterals are given off to the lateral and dorsolateral hypothalamic areas and to the dorsolateral and dorsomedial nuclei of the thalamus. Those fibers not leaving the habenulo-interpeduncular tract terminate in the lateral habenular nucleus or continue along its lateral border to enter the stria medullaris and terminate in the anterior hypothalamus (Massopust & Thompson, 1962).

Studies of lesion induced effects involving the IPN have generally shown significant disruption of the retention of simultaneous or successive brightness discriminations using either shock avoidance (Thompson, Baumeister & Rich, 1962; Thompson & Massopust, 1960; and Thompson & Rich, 1961) or food motivation (Thompson, Duke, Malin, & Hawkins, 1961). Thompson and his collaborators also found that IPN lesions produced marked postoperative retention deficits of a kinesthetic discrimination (Thompson, et al., 1961) and of an auditory (Thompson, et al., 1961) or visual (Thompson, 1960) signalled active avoidance task. In contrast, Hatton (1965) found that IPN lesions produced little or no impairments in retention of a simultaneous visual

discrimination unless accompanied by incidental damage to the decussation of the ventral tegmental tract. However, Hatton's finding supported the earlier observations of a decremental effect of IPN lesions on the retention of an active avoidance task.

The purpose of this study was to investigate the role of the IPN as a midbrain contributor to avoidance behavior. Since the habenulo-interpeduncular tract provides a pathway connecting the septal-habenular complex to the midbrain, the second purpose of this study was to determine whether the behavioral changes following lesions in the interpeduncular region reflect the behavior occurring with damage further upstream in the habenular nucleus. Accordingly, the effects of IPN lesions and stria medullaris--habenular lesions were investigated in an appetitively motivated passive avoidance task and during the acquisition, retention, and extinction of a two-way shuttlebox avoidance task.

## Method

### Subjects

Sixty-five male, Long-Evans rats weighing 300-350 grams were purchased from Rockland Farms, Gilbertsville, Pa. and were housed in individual cages. Ad lib. food and water were available except for a food deprivation period during the passive avoidance phase of the experiment.

### Apparatus

The chamber for active avoidance testing consisted of an automated shuttlebox with 1.0 cm. Plexiglas walls. The 60 X 45 X 15 cm. shuttlebox was partitioned into two compartments of equal size by a Plexiglas barrier 6 cm. in height. Each compartment floor, constructed of 6.3 mm. stainless steel bars mounted 1.9 cm. apart, could be independently electrified. The shuttlebox was enclosed within a sound-attentuating chamber that was ventilated by a blower which provided a masking noise of 74 db (re: .0002 dynes/$cm^2$). The CS was provided by a 79 db buzzer mounted centrally 25 cm. above the shuttlebox floor.

The same shuttlebox was used for passive avoidance testing except for the following modifications. A food cup, which could be electrified, was placed 7.6 cm. above the grid floor at the end of one of the compartments. When the food cup was electrified, the animal received a shock to the mouth when the food cup was touched. In addition, a 30 cm. high guillotine door was installed over the barrier. Appropriately located pulleys allowed manual operation of this door. A one-way window in the sound-attenuating chamber permitted the experimenter to observe the subject. The chamber was illuminated by a pair of 5-w. bulbs located behind the shuttlebox. Latency

measures were taken with a stop watch.

Surgery and Histology

There were two experimental and two control groups. Subjects in the experimental groups were given either stria medullaris--habenular lesions or IPN lesions. The control subjects were either permitted to remain normal or given a sham lesion in which the electrode was lowered only to the dorsal aspect of the IPN then removed without producing a lesion. All operated subjects were anesthetized during surgery with an intraperitoneal administration of Equi-Thesin and given 45,000 units of Bicillin post-operatively. Lesions were produced with anodal dc at the uninsulated tip of a stainless steel electrode inserted stereotactically into the brain. The circuit was completed with an anal cathode. The stria medullaris--habenular lesions were bilateral, while the IPN lesions were produced with only one mid-line placement. Measurements were made from the bregma, midline, and dural surface with the incisor bar positioned horizontally along the interaural plane. Lesion coordinates in millimeters and currents were as follows: stria medullaris--habenular, 2.3 P, 0.6 L, 4.3 D with 1.0 ma for 10 secs.; IPN, 6.2 P, 0.0 L, 7.6 D with 1.0 ma for 10 secs.

Following testing, the rats were sacrificed with an overdose of Equi-Thesin and perfused successively with 0.9% saline and 10% formalin. Following extraction and fixation, the brains were embedded in celloidin and sectioned coronally at 20 μ. The sections were stained with cresyl violet and then counterstained with Luxol fast blue MBS.

Procedure

Passive avoidance. All subjects were tested on passive avoidance before

the active avoidance phase of the experiment. Four days after surgery the subjects were placed on a 23 hour food deprivation schedule. Four days later, passive avoidance training began. During the first two days of training, the subjects were placed in the shuttlebox for 20 min. to permit adjustment to the grid floor, to raising and lowering of the guillotine door, and to eating from the food cup. On the third, fourth, and fifth days, training consisted of raising the guillotine door for each trial to allow the subjects to cross the barrier to eat a 45 mg. Noyes food pellet in the food cup. If the subject did not return to the original compartment after eating, the experimenter gently nudged it back with a small plywood paddle. Twenty trials were given daily. After 35 trials, a 15 sec. intertrial interval was introduced and the subject was then permitted only 60 sec. to cross the barrier after the guillotine door had been raised. Response latencies, approaches, partial responses, and eating occurrences were recorded. The response latencies were the intervals between the raising the guillotine door and the subject's crossing the barrier into the food compartment with all four feet. An approach response was recorded if the subject fully crossed the barrier, then either returned to the original compartment without eating or did not eat within the allotted 60 sec. A partial response was recorded if the subject touched the grid floor of the food compartment then withdrew from the compartment without completely crossing the barrier. On the sixth day, shock trials were introduced. After allowing 5 regular trials, the food cup was electrified for 5 additional trials and delivered a shock of .25 ma ac to the subject when he touched it. Sixty sec. were again allowed for responding. After receiving a shock, subjects were nudged back into the original compartment. If the subject failed to respond, the guillotine door

was closed starting the next intertrial interval (ITI) and a 60 sec. response latency was recorded. Thirty post-shock test trials were then conducted immediately after the shock trials. The procedure during the post-shock trials were the same as during the training sessions.

Active avoidance. After completing the passive avoidance trials the subjects were returned to an ad lib. food schedule. Three days later active avoidance began. A standard two-way shuttlebox procedure was used. Trials were separated by a 30 sec. ITI and consisted of a 10 sec. CS presentation followed by the introduction of a 0.5 ma ac response-terminated shock. Each session was started with a 10 min. adaptation period. If the subjects crossed to the opposite compartment during the CS presentation, an avoidance response was recorded; otherwise, an escape response was counted. The CS, in both instances, was response-terminated. In addition, intertrial responses (ITRs) were counted. The subjects were given 25 trials daily until a criterion of 22 out of 25 avoidance responses was reached across sessions. If the subjects failed to reach criterion within 8 sessions, the training was terminated, and 200 avoidance responses were recorded as the acquisition score. After meeting the criterion, all subjects were given an eight day pause in training and then were retested until they again reached the same avoidance criterion. The procedure for these retention trials was the same as that used during the original active avoidance training.

Extinction trials began on the day after reaching criterion during the retention test. The ITI was reduced to 12 sec. If the subject failed to respond within the 10 sec. CS-UCS interval, an escape response was recorded; the CS was terminated; and the next ITI was initiated. If they responded during the interval, an avoidance response was recorded. Five daily sessions

of 25 trials were conducted with the shock omitted. Following the extinction period, all subjects were sacrificed.

# Results

## Histology

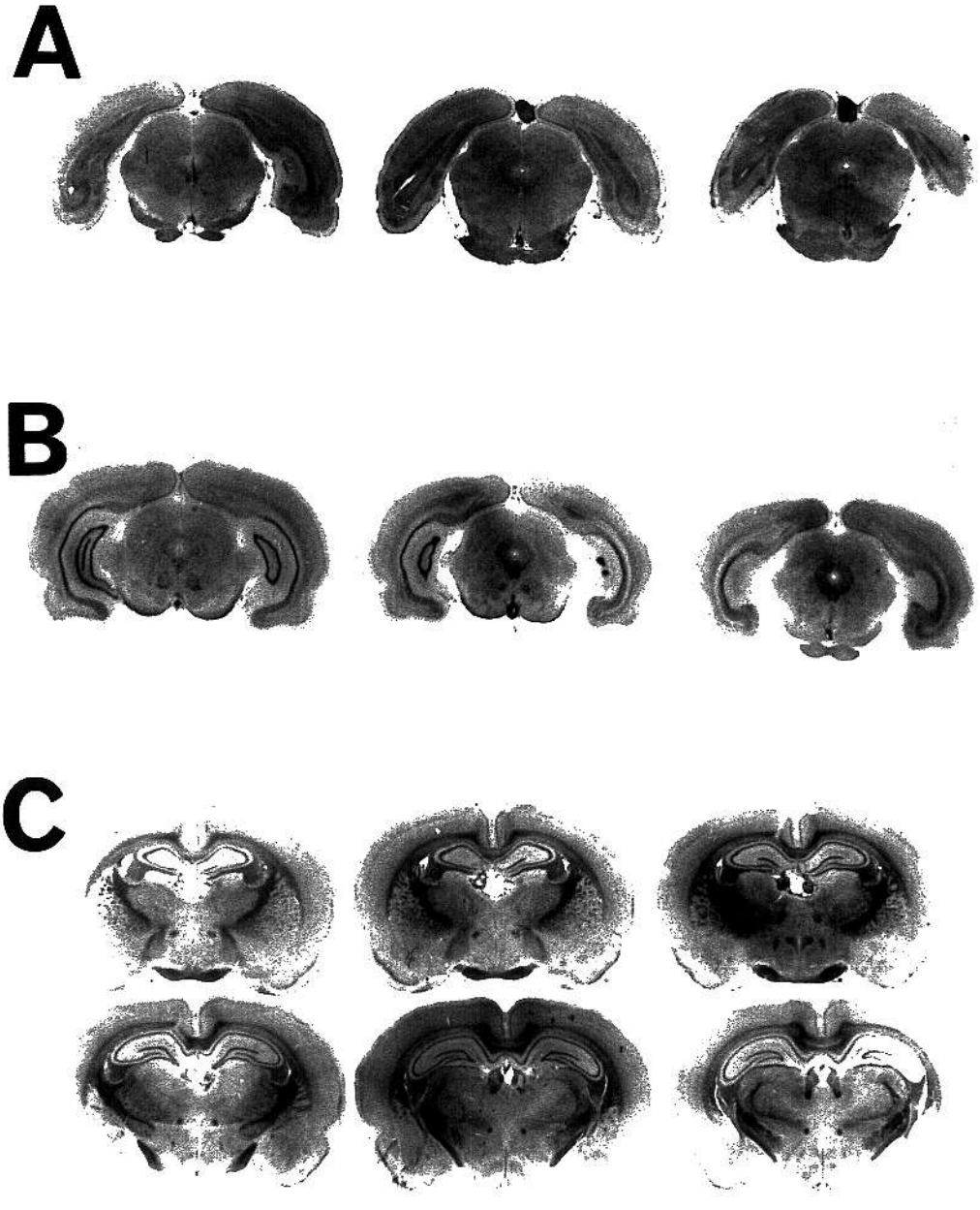
Photomicrographs of representative lesions are shown in Figure 1. Since discrete interpeduncular nucleus lesions were difficult to obtain, the subjects were classified according to the presence or absence of additional damage to the overlying ventral tegmental area. Histological analyses were conducted by independent experimenters without previous knowledge of the behavioral results.

Six subjects received IPN lesions which were restricted to the IPN with less than 10% of the damaged tissue extending dorsally into the ventral tegmentum region. These lesions typically started near the anterior margin of the pons and extended caudally to the anterior aspect of the raphe nuclei. Two subjects received damage that originated at the anterior border of the IPN, severing the fasiculus retroflexus, and continued posteriorally in the nucleus to the midpontine region. In two instances the lesions extended ventrally to inflict minor damage to the transverse fibers of the pons. No relationship was observed between performance and the locus of IPN damage.

Nine subjects designated as a combined IPN-tegmentum group possessed IPN damage comparable to the IPN group. In such instances, however, 10% or more of the destroyed tissue extended dorsally into the medial portion of the ventral tegmentum. Combined lesions placed more posteriorally typically invaded the ventral aspect of the brachium conjunctivum. In four subjects minor secondary damage was observed unilaterally in the red nucleus and within the region of crossed fibers of the dorsal tegmentum.

Five subjects received essentially tegmental lesions. This group was characterized by less than 10% of the destroyed area encroaching upon the

Fig. 1. Photomicrographs showing representative interpeduncular nucleus lesions (a & b) and a stria medullaris--habenular lesion (c).


A
B
C

IPN region and accompanied by tegmental damage equivalent to the combined IPN-tegmental group. In one subject the anterior portion of the lesion involved minor damage to the supramammillary decussation. However, no secondary damage was observed to the adjacent mammillary body or hypothalamic nuclei. The eleven operated control subjects sustained damage only from the insertion of the electrode.

Eleven subjects received lesions typically destroying 70% or more of both stria medullaris and habenular nuclei. These lesions were often accompanied by incidental damage to the adjacent anterodorsal hippocampus (N=5), anterior (N=2), dorsomedial thalamic (N=6), and anterior median cingulate gyrus (N=5). Certain subjects were rejected because they were difficult to classify. In such cases, 20% or more of the damaged tissue exceeded the confines of the stria medullaris--habenular complex. Accordingly, 10 subjects with damage invading principally upon the septum, fornix, and sizable portions of the anterodorsal hippocampus were discarded.

Performance

The variables with repeated measures were analyzed with an unweighted means analysis of variance (ANOVA) while a simple ANOVA was used on variables with a single measure. Neuman-Keuls test were conducted for comparing treatment means using a .05 level for significance (cf., Winer, 1962).

Passive avoidance. The ANOVA of post-shock eating occurrences across five-trial blocks showed a reliable treatment effect ($F=18.02$, $df=5/48$, $p<.001$) and trials effect ($F=6.80$, $df=5/241$, $p<.001$). Comparison of the group main effects, illustrated in Figure 2, revealed that the percent eating responses for all four lesion groups was reliably higher than for the two control groups. No reliable differences were found between the control

groups or between the lesioned groups. The percent eating responses for all groups increased over post-shock trials.

The analysis of variance of post-shock latencies yielded similar group effects. A reliable treatment effect ($F$=13.23, $df$=5/48, $p$<.001) and trial effect ($F$=8.21, $df$=5/241, $p$<.001) was obtained, but no interaction was found. The post-shock latency measure closely reflected the percent eating responses measure. No reliable effect was observed for the number of shocks received, or for the number of approach or partial responses emitted.

Active avoidance. All subjects reached the criterion within the allotted trials. The means and group size are provided in Table 1. ANOVAs yielded a reliable treatment effect for trials to criterion ($F$=6.85, $df$=5/49, $p$<.001) and percent avoidance during acquisition ($F$=4.23, $df$=5/49, $p$<.001), but no reliable treatment effect occurred for ITRs. Comparison of the treatment means for trials to criterion showed that the stria medullaris--habenular and IPN lesioned groups differed from the operated control, normal control, and tegmental lesioned groups. There was no reliable difference between the former two and the latter three groups nor did any of these groups differ from the combined IPN-tegmental lesioned group.

Mean comparison of percent avoidance closely reflected the effects obtained with trials to criterion. The normal, and operated control groups and the tegmental lesioned group did not differ reliably, nor did the combined IPN--tegmental lesioned group and the two control groups. The stria medullaris--habenular, IPN, and the combined IPN--tegmental lesioned groups did not differ reliably, although a reliable difference existed between the tegmental lesioned group and the combined IPN--tegmental, IPN, and stria medullaris--habenular lesioned groups. Additionally, mean comparison showed

Fig. 2. Mean percentage of eating occurrences per group across five-trial blocks.

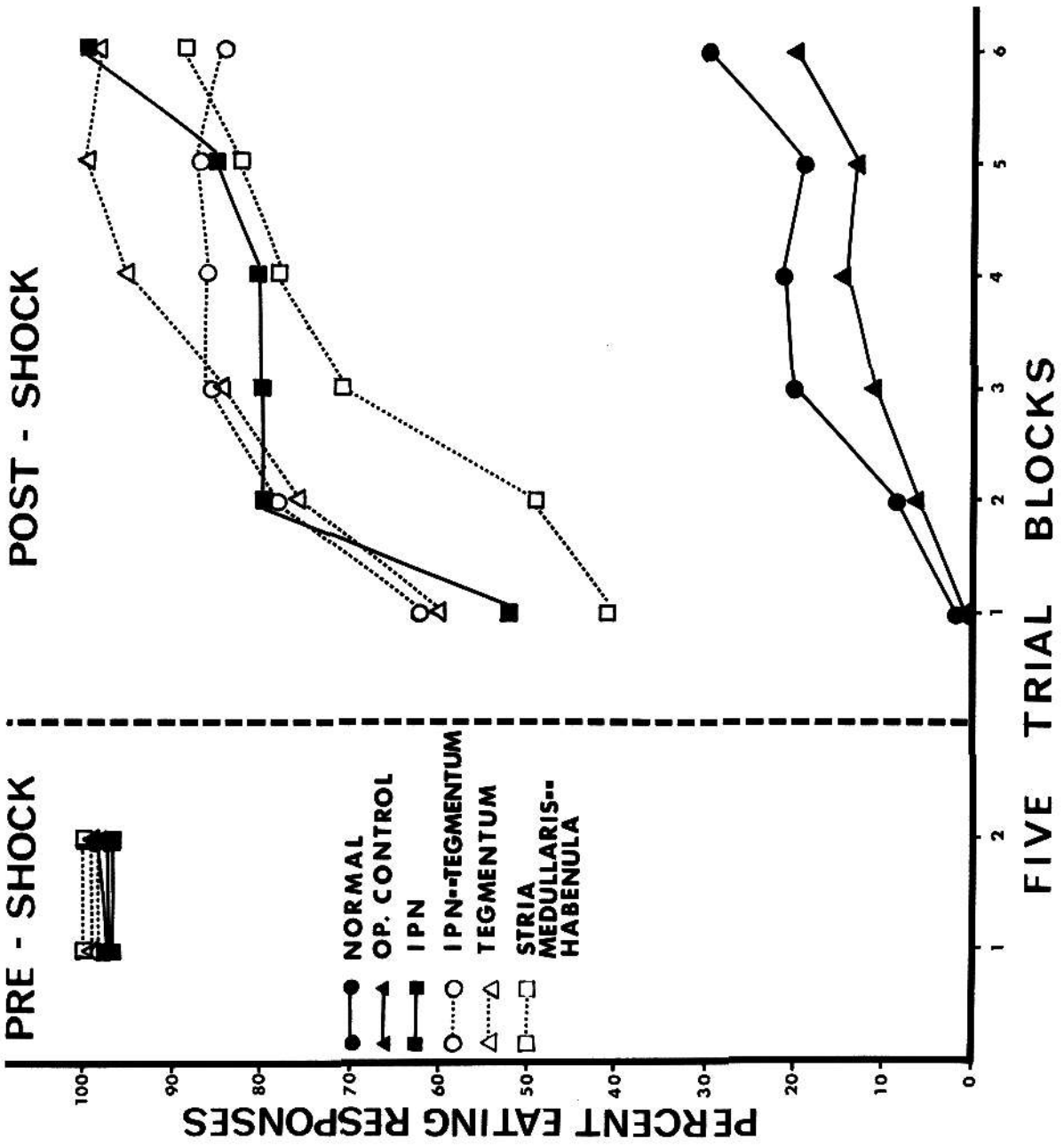

PRE - SHOCK
POST - SHOCK
NORMAL
OP. CONTROL
IPN
IPN--TEGMENTUM
TEGMENTUM
STRIA MEDULLARIS--HABENULA
PERCENT EATING RESPONSES
FIVE TRIAL BLOCKS

Table 1

Mean Performance and Group n for Active Avoidance

| | GROUP | | | | | |
|---|---|---|---|---|---|---|
| | IPN | Stria Medullaris--Habenula | IPN--Tegmentum | Operated Control | Normal | Tegmentum |
| Trials to Crit. | 49.2 | 42.3 | 63.3 | 76.2 | 86.2 | 89.0 |
| Percentage Avoid. | 72.8 | 74.1 | 62.8 | 56.9 | 51.5 | 48.0 |
| ITRs per Five Trials | 1.2 | 1.7 | 3.6 | 1.5 | 0.8 | 1.6 |
| n | 6 | 11 | 9 | 12 | 12 | 5 |

a reliable difference between the control groups and either the IPN or stria medullaris--habenular lesioned group.

Even though there was no overall treatment effect for the average number of ITRs, comparison of means per 5-trial block of acquisition revealed that a significant difference did exist between the normal group and the combined IPN-tegmental lesioned group. None of the remaining comparisons were significant.

ANOVAS of trials to criterion and total percent avoidance during retention yielded no significant differences between groups.

Statistical analysis of percent avoidance during extinction revealed no reliable group effect, days effect, or treatment X days interaction. Since the ITI was shortened during the extinction trials, the ITRs were not analyzed.

## Discussion

The major findings of this study suggest that the interpeduncular nucleus plays a significant role in active and passive avoidance conditioning which, during passive avoidance, can be differentiated from that of the overlying tegmentum. Additionally, the behavioral effects seen following interpeduncular lesions resembled those seen following damage to the stria medullaris--habenular complex.

The observation that stria medullaris lesions lead to a facilitation of active avoidance behavior replicated the earlier findings of Van Hoesen, MacDougall, and Mitchell (1969). However, this study failed to replicate the finding of Van Hoesen et al. that rats with stria medullaris--habenular lesions show a greater resistance to massed extinction. The reasons for this discrepancy are not clear.

It seems unlikely that the behavioral effects observed following stria medullaris--habenular lesions in this study could be attributed to incidental damage to the anterodorsal hippocampus, dorsomedial thalamus, or anterior median cingulate gyrus. Kimura (1958) and Kaada, Rasmussen, & Kviem (1962) showed that anterodorsal hippocampal lesions produced no deficit in passive avoidance responding. Later, Gerbrandt (1965) reported similar findings for dorsomedial thalamic lesions and Vanderwolf (1964) and Bohus and DeWied (1967) also reported similar observations. In addition, Lubar (1962) showed that anterior median cortical lesions did not interfere with appropriate passive avoidance responding, although Trafton (1967) and Albert and Bignami (1968) observed that similar lesions led to a decrement in a two-way active avoidance task. Thus, to attribute these findings to damage to any structure outside the stria medullaris--habenular complex seems

unfounded.

The results suggest that the IPN is as important as the ventral tegmentum in accounting for passive avoidance performance. It should be noted that a significant portion of the habenulo-interpeduncular tract travels along the dorsal surface of the IPN (Nauta, 1958), instead of terminating within the nucleus. Thus, the passive avoidance decrement could be attributable to either the IPN damage or to the damage to the more dorsally lying fibers that bypass the IPN.

The results obtained during active avoidance training suggest that enhancement obtained with IPN lesions is reduced when additional damage extends into the ventral tegmental area. The observation that subjects with lesions restricted to the tegmental area produce a small but unreliable decrement in active avoidance performance suggests that the tegmental damage cancels the facilitory effect of the IPN lesion. The finding that the combined IPN-tegmental lesioned group did not differ reliably from either the control groups or from the IPN and the stria medullaris--habenular lesioned groups supports this suggestion.

Subjects with tegmental damage often showed a variety of motor abnormalities, particularly when the tegmental damage was quite extensive. These abnormalities included postural asymmetries of the neck musculature, a tendency to circle in one direction or another. Subjects within the combined IPN-tegmental lesioned group demonstrated larger numbers of ITRs per five-trial block. This reflected their sporadic increases in motor activity. It is possible that these motor abnormalities interfered with the avoidance facilitation produced by IPN lesions.

Previous investigators have preoperatively trained subjects on active

avoidance tasks or on a discriminated avoidance task and then tested for an impairment in retention following IPN lesions. Thompson (1960), Thompson, Duke, Malin, and Hawkins (1961), and Thompson and Massopust (1960) trained rats to jump and grasp onto the edge of an avoidance chamber after the onset of a visual stimulus in order to avoid receiving a shock. These investigations demonstrated consistently that IPN lesions produced a marked decrement in retention. Hatton (1965) reported similar findings using a two-way shuttlebox avoidance task. The facilitation of active avoidance conditioning found in this study following IPN lesions appears surprising in view of these results. However, the IPN lesions described in these studies often spared nearly 50% of the IPN, while inflicting sizable damage to the ventral tegmentum and, occasionally, to the dorsal tegmental region. In contrast, this study regarded similar damage as unacceptable IPN lesions and designated them as combined IPN-tegmental lesions. The fact that notable motor impairment was observed in the combined IPN-tegmental and the tegmental lesioned groups suggests that the retention deficits observed by Thompson and by Hatton may be attributable to the motor abnormalities accompanying extensive ventral midbrain lesions.

The results of this study suggest that the behaviorally defined effects of IPN lesions reflect changes that occur with damage to various septal nuclei or to their projections. Van Hoesen et al. (1969) reported that severing the septal tracts coursing through the stria medullaris--habenular complex led to active and passive avoidance behavior resembling the avoidance behavior occurring after septal lesions. In view of the direct IPN-habenular connections, the observation of a behavioral similarity after discrete lesions to these nuclei suggests that the IPN is an integral midbrain component

of the limbic system.

Based on electrophysiological evidence, Nauta (1958; 1960; 1963) suggested that a neural circuit existed between the limbic forebrain structures and the paramedian midbrain region, including the IPN and ventral tegmental nuclei. Nauta interpreted this system as a major lower circuit of the limbic system consisting of multisynaptic connections originating in the septo-hippocampal area and coursing in the precommissural fornix, medial forebrain bundle, and mammillo-tegmental tract to terminate in the "limbic midbrain area". However, when the present study is examined in connection with the findings of Van Hoesen et al. (1969) it appears equally reasonable that the limbic system--midbrain circuit contributing to avoidance responding may be distributed over the more direct septo-habenular-IPN route.

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MASTER OF SCIENCE

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1969

Rats with lesions placed in the interpeduncular nucleus (IPN), tegmentum, combined IPN--ventral tegmentum, or stria medullaris--habenular complex were examined for deficits in active and passive avoidance conditioning. All lesioned groups were impaired on the food motivated passive avoidance task while only the IPN and stria medullaris--habenular lesions facilitated the acquisition of a two-way shuttlebox active avoidance task. The similarity of behavior following IPN and stria medullaris--habenular lesions suggests that the interpeduncular nucleus serves as a significant limbic-midbrain contributor to avoidance responding.