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# The role of the hippocampus in REM sleep and short-term memory in rats

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# THE ROLE OF THE HIPPOCAMPUS IN REM SLEEP AND SHORT-TERM MEMORY IN RATS

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for the Degree of Master of Arts

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I hereby declare that this dissertation is my own work and that I have not submitted it for a Master's Degree to any other university.

B.M. Chalmers

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ABSTRACT

In a series of three experiments the effects of hippocampal lesions on quantity of REM sleep and a number of related measures, as well as memory consolidation in rats, was investigated. In the first experiment dorsal hippocampal lesions, which extended to include the septal area, significantly reduced REM sleep after REM deprivation. Ventral hippocampal lesions had very little effect. However, in the second experiment when only the dorsal hippocampus was damaged, no disruption of REM sleep was found. Further investigation in the second and third experiments, which examined the relationship between the hippocampus, memory consolidation and REM sleep, found that neither the hippocampus nor REM sleep was essential for consolidation from short-term to long-term memory. These results gave no support to current theories of hippocampal involvement in REM sleep or to theories which suggest that the hippocampus or REM sleep is necessary for memory consolidation.

In the past the hippocampus has been assigned a number of functions in emotional control, reproductive drives, cortical activation, memory and learning (Read, 1969). In more recent years research findings have been interpreted to implicate the hippocampus in motivational processes (Grastyan, Karmos, Vereczkey and Kellenyi, 1966; Kamback, 1967; Rabe, 1963; Rabe and Haddad, 1968), goal directed behaviour (Adey, 1966; Brammer, 1964), inhibition of responses (Douglas, 1967), information processing (Bogshaw, Kimble and Pribram, 1965; Douglas and Pribram, 1966) and attentional processes (Grastyan, 1959; 1961; Kimble, 1968). In addition, a number of workers (Greenberg, Pearlman, Brooks, Mayer and Hartmann, 1968; Hernandez-Peon, 1966; Jouvet, 1963; Meissner, 1966; Parmeggiani, 1967; Torda, 1967; 1968a; 1968b; 1968c; 1969b; 1969c) have recently presented indirect, but nevertheless suggestive evidence that the hippocampus is involved in paradoxical (PS) or rapid eye movement (REM) sleep.

Hippocampal involvement in PS is indicated because the hippocampus is anatomically linked with the pontine nuclei which Jouvet (1962) has shown to be involved in the regulation of PS. During PS the hippocampus is activated by ascending influences from the caudal pontine nuclei over midbrain limbic circuits and septo-hippocampal paths (Meissner, 1966). Meissner has stated that it could be through the connections of the pontine nuclei with the hippocampus and its related structures that the neurophysiological basis of dreaming is established.

Also, activation of the hippocampus by electrical stimulation or by means of drugs has resulted in experiences similar to dreaming. Stimulation of the temporal cortex by Penfield (1958) and of the hippocampus by Pampiglioni and Falconer (1960) elicited dream-like experiences or vivid memories. Lysergic acid diethalamide (LSD) has been shown to activate the hippocampus (Chapman, Walter, Adey, Crandal, Rand, Brazier and Markhan, 1965; Torda, 1968b) either by acting directly on it (Baldwin, Lewis and Bach, 1959; Stumph, Petsche and Gogolak, 1962) or by exciting the temporoammonic circuits (Adey, Bell and Dennis, 1962). Furthermore, LSD has been shown to increase dream time (Green, 1965; Muzio, Roffwarg and Kaufman, 1966) and to reduce REM latency (Torda, 1968b).

Other theories suggesting hippocampal involvement in PS have speculated on the function of hippocampal theta in REM sleep. Many studies have shown that hippocampal theta is associated, among other things, with PS (Brugge, 1965; Jouvet, 1967; Lena and Parmeggiani, 1964; Okuma, Hayashi and Fujimori, 1964). However, the precise role that hippocampal theta plays in REM sleep is not clear. Parmeggiani (1967) presented evidence which suggested that hippocampal theta served to stabilize PS. He suppressed hippocampal theta by means of discrete septal lesions and found that the average number and mean duration of PS episodes fell by about 40%, but only when the animals were not in a soundproof room. He interpreted the loss of PS under noisy conditions as support for the idea that hippocampal theta stabilizes PS. Parmeggiani also pointed out that, in his septal animals, slow wave sleep was

less frequently followed by activated sleep. The animals woke up suddenly during the flattening of the electromyogram (EMG) instead of entering PS. The stabilization theory is also supported by evidence from Lena and Parmeggiani (1964) which suggested that the destruction of limbic efferent pathways, resulting in a dissociation of hippocampal theta from cortical desynchronisation, led to a disruption of PS.

In contrast to Parmeggiani, Hernandez-Peon (1966) and Torda (1967; 1968a; 1968b; 1968c; 1969b; 1969c) have assigned a somewhat different function to hippocampal theta in PS. Torda (1967; 1968a; 1968b; 1968c; 1969b; 1969c) has proposed that hippocampal theta acted as an inhibitory mechanism preventing the visual concomitants of processes related to memory retrieval, entering conscious awareness. Hernandez-Peon (1966) has formulated a neurophysiological model of dreams and hallucinations in which hippocampal theta activity plays an important role. This model is based on the commonly held assumption that dreams and hallucinations involve some common brain mechanisms (Green, 1964; Hernandez-Peon, 1966; Torda, 1968b). The model consists of a galaxy of systems. According to Hernandez-Peon the "Dream System" is tonically inhibited by the "Vigilance System" which is in turn tonically inhibited by the "Sleep System". The "Vigilance System" is reminiscent of the function of hippocampal theta as postulated by Torda (1967; 1968a; 1968b; 1968c; 1969b; 1969c). It would be expected then that agents which reduce the frequency of hippocampal theta thereby breaking down the "Vigilance System" or attenuating its inhibitory function should result in PS or hallucinations. This idea has been supported by a considerable amount of

evidence. When the frequency of hippocampal activity was from 2-4 cycles/second hallucinations occurred during wakefulness (Torda, 1968b; 1969a) and dreaming occurred during sleep (Torda, 1968a; 1969b). Furthermore, increasing hippocampal activity to 6 cycles/second by D amphetamine consumption (Stumph, 1965) abolished or significantly reduced dreaming (Rechtschaffen and Maron, 1965).

Evidence derived from the content analysis of the dreams of humans also implicates the hippocampus in REM sleep. It is well documented that dreams are often loaded with emotionally laden material from recent experiences (Berger, Oliey and Oswald, 1962; Cartwright, Bernick, Borowitz and Kling, 1969; Collins, Davison and Breger, 1967; Foulkes, 1964; Foulkes and Rechtschaffen, 1964; Greenberg and Liederman, 1966; Hunter and Breger, 1967; Lane and Breger, 1967) suggesting that there may be some common neural mechanism underlying PS and recent memory. Furthermore, the emotional nature of dream content indicates limbic involvement in REM sleep. Evidence reported by Torda (1969a; 1969c) and Greenberg et al. (1968) that the dreams of subjects with lesions in the hippocampus or related structures lacked both emotional depth and daily residue when compared to the dreams of normal subjects, further supports this point of view.

In addition to contributing to the emotional content of dreams the hippocampus could possibly be further implicated in REM sleep since both the hippocampus (Hagshaw et al., 1966; Pribram, 1967; Torda, 1967b) and REM sleep

(Breger, 1967; Evans and Newman, 1964; Feldman and Dement, 1968; Fishbein, 1969a; 1969b; 1969c; Fishbein, Swarz and McGaugh; Gaarder, 1966; Greenberg, 1970; Greenberg and Dewan, 1968; 1969; Greenberg, Pearlman, Fingar, Kantrowitz and Kawliche, 1970; Greenberg, Pillard and Pearlman, 1968; Hawkins, 1966; Jouvet, 1963; Lucaro, 1970; Newman and Evans, 1965; Pearlman and Greenberg, 1968; Swarz and McGaugh, 1970) are thought to be involved in information processing.

While the data from human subjects suggested that common neural mechanisms, including the hippocampus, underlie PS and recent memory, no comparable data is available from animals. The tendency in recent years has been to reject the idea of hippocampal involvement in the memory processes of animals (Douglas, 1967) but there are still a number of unanswered questions. Douglas (1967) and Drachman and Ommaya (1964) pointed out that hippocampal animals may have a memory deficit but because of the relatively simple nature of the tasks they perform in experiments in comparison with human verbal learning, the animal deficit has not been demonstrated.

Further weight is added to the argument that the nature of the task is important in demonstrating a memory deficit in hippocampal animals when it is considered that human Korsakoff patients only show a deficit in the recall of complex material while more or less automatic skills are relatively well preserved (Talland, 1960).

The evidence presented so far indicates that the hippocampus is involved in PS and in memory functions. In addition there are indications of a connection between PS and memory. The present series of experiments was designed to investigate the role of the hippocampus in PS and short-term memory (STM) as well as examining the connection between PS and memory.

## GENERAL PROCEDURE

### Subjects

In all the experiments the subjects were male hooded rats, weighing between 300 gm and 400 gm at the time of operation. All animals were experimentally naive. During the experiment the rats were housed singly and had free access to food and water. All animals were kept under conditions of constant light except those used in Experiment 1 where a normal day-night cycle of lighting existed in the animal room.

### Electrodes

The electrodes used in the first two experiments were constructed from unipolar Amphenol type 27-9 connectors which were modified for use as bipolar electrodes.

One pole of the electroencephelogram (EEG) electrode, (Fig. 1) was made by filing down the centre pin of the Amphenol connector until it protruded between 2,0 mm and 3,0 mm from the base. The other pole was constructed by soldering a piece of 0,01 inch diamel coated nickel chromium wire 10,0 mm long to the base of the connector. The wire was then insulated with polythene tubing and a small loop was made approximately 7,0 mm from the centre pin. The assembly was then insulated with Glyptal Electrical Varnish.

To construct the EMG electrode (Fig. 2) the centre pin of the Amphenol connector was filed down until it protruded about 0,5 mm from the base. Wires were then soldered to the base and the centre pin to form the two poles of the electrode. The wires were bent and insulated with polythene

tubing as indicated in Fig. 2. The electrode assembly was then insulated with Glyptal Electrical Varnish.

Insulation was scraped off the electrodes at the points where they were to make contact with the cortex or neck muscles.

Surgery

The surgical procedures differed from experiment to experiment but, in general, surgery was carried out under sodium pentothal anaesthesia. Atropine was administered five minutes before the pentothal to ensure ease of respiration during the operation. Brain damage was made by passing a current through the 0,5 mm uninsulated tip of a needle which was positioned stereotaxically. The coordinates at which the needle was inserted will be mentioned specifically when each experiment is described. Generally the needle served as a cathode and the circuit was completed by a rectal anode.

EEG and EMG electrodes were implanted either at the time the lesion was made or a few days later. One pole of the EEG electrode was implanted 2,0 mm anterior and the other 5,0 mm posterior to the bregma. Both poles were 1,5 mm lateral to the midline. The wires of the EMG electrode were looped through the neck muscles and the Amphenol connector rested on the skull. Both electrodes were secured to the skull by means of dental cement and anchoring screws.

Histology

At the completion of all experiments the subjects were perfused with an isotonic saline solution followed by 10% formalin. The brains were removed and fixed in 10%

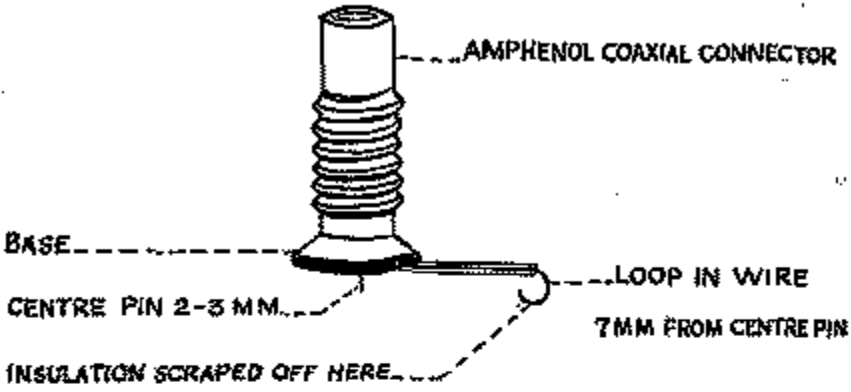



Fig. 1. EEG electrode

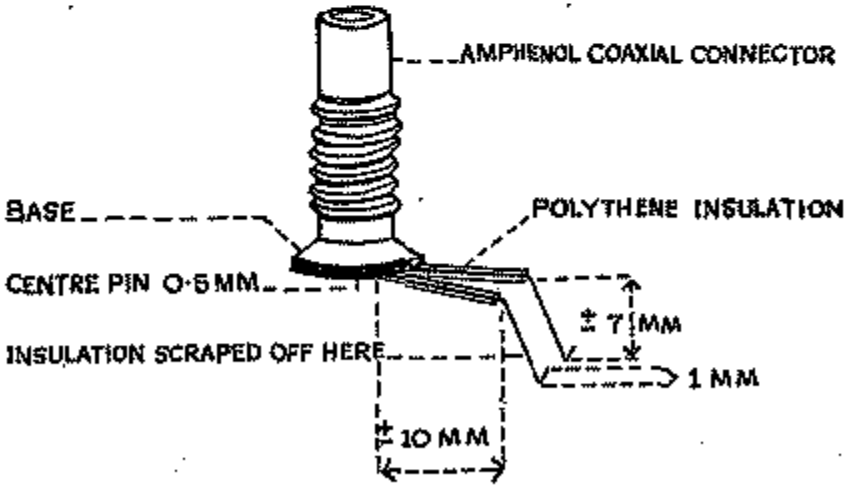



Fig. 2. EMG electrode

formalin. The frozen tissue technique was used to prepare sections at 50µ.

Apparatus

In all experiments PS deprivation was carried out by using the method described by Morden, Mitchell and Dement (1967). The subjects were placed on an inverted flower pot in a tank of water. The diameter of the base of the pot was 6,8 cm and it was 10,0 cm high. The tank was 33,0 cm wide, 46,0 cm long and 32,0 cm high. The bottom of the tank was covered with 5,0 cm water, whereas Morden et al. (1967) filled the tank until the water level was just below the lip of the flower pot. However, in this study, it was found that having less water in the tank had the advantage that animals could keep themselves clear of the water more easily while still being deprived of PS. Thus the problems which arise from being housed in a constantly damp environment were somewhat reduced. The tank was covered with wire mesh which was weighted down to prevent the rat from escaping. A food dish and water bottle were attached to the mesh within easy reach of the animal.

In the second and third experiments the tank was replaced by a bucket. The diameter of the base of the buckets was 19,0 cm and the height was 25,0 cm.

During adaptation and recording sessions of the first and second experiments the rats were kept in a semi-soundproof room in cages 26,0 cm long, 25,0 cm wide and 28,0 cm high. One side of each cage was made of transparent perspex so that the animals could be observed. The animals

were separated from the experimenter and recording equipment by a one-way screen.

Recordings of EEG and EMG activity were made on a Beckman Type R dynograph with paper speed set at 1.0 cm per second.

## EXPERIMENT 1

Experiment 1 investigated the effects of hippocampal lesions on PS. Since a growing body of evidence indicates a functional dissociation between the dorsal and ventral hippocampus (Adey, 1964; Elul, 1964; Goodfellow and Niemer, 1961; Gross, Chorover and Cohen, 1965; Hughes, 1965) it was decided that the effects of lesions in both these areas of the hippocampus on PS should be examined.

## PROCEDURE

### Surgery

The 18 animals used in this experiment were divided into four groups. Group DH with six rats, received bilateral lesions in the dorsal hippocampus. The lesions were made by delivering a 4,0 mA direct current to the brain for 30 sec at the co-ordinates given in Table 1.

Group VH also with six animals, received bilateral lesions in the ventral hippocampus. The lesions were made firstly, by delivering a 4,0 mA current to the brain through a needle at the co-ordinates given in Table 1. Secondly, the greater part of the ventral hippocampus was destroyed by delivering a current from a Grass Model LM3 Lesion Maker through the 2,0 mm uninsulated tip of a blade 1,0 mm wide. The intensity regulator on the lesion maker was set at 80 and the current was delivered for 40,0 sec. An alligator clip attached to the reflected skin on the animal's head completed the circuit.

Group C also with six rats served as operated controls. Three animals were used as a control for dorsal, and three animals as a control for ventral hippocampal lesions. The control animals were subjected to sham operations and the needle or blade was inserted at the co-ordinates given in Table 1. No current was delivered to the brains of animals in the control group.

The experimental animals were given from three to five days to recover from the operation and then the EEG and EMG electrodes were implanted under ether anaesthesia. Electrodes were not implanted at the same time as the lesion was made because of the high mortality rate (77%) caused by the ventral lesions in a pilot study. To save time and equipment it was decided to ensure that the animals would survive the effects of the lesion before implantation of the electrodes. On the control animals, however, the implants were done at the same time as the sham operation.

TABLE 1

Co-ordinates in mm that were used for lesion and control operations

| Group | Number of Subjects | Posterior to bregma | Lateral to midline | Ventral to surface of brain | Type of electrode |
|---|---|---|---|---|---|
| Dorsal Lesion | 6 | 3,25<br>4,25 | 1,00 2,50<br>1,25 2,75 | 3,25<br>3,25 | Needle |
| Ventral Lesion | 6 | 5,25<br>6,25 | 4,00<br>5,25 | 3,75<br>5,50 | Needle<br>Blade |
| Dorsal Control | 3 | 3,25<br>4,25 | 1,00 2,50<br>1,25 2,75 | 2,75<br>2,75 | Needle |
| Ventral Control | 3 | 5,25<br>6,25 | 4,00<br>5,25 | 3,00<br>3,50 | Needle<br>Blade |

Experimental Procedure

The animals were given from six to twelve days to recover from the first operation.

Following the recovery period all the animals received the same experimental treatment for the next eleven days. Table 1 summarizes the procedure followed for each animal. On each of the two adaptation days the rats were placed in cages in the room where they were to be tested with recording wires attached to their EEG and EMG electrodes, but no recordings were taken. Each adaptation period lasted for six hours, upon which the subjects were returned to their home cages. Following adaptation the subjects were left in their home cages for the third and fourth days while the apparatus in the recording room was used to test other animals.

On the fifth and sixth days the first and second baseline recordings were made. Three or four animals were tested at a time. All but one group of three or four animals consisted of at least one control animal, the remainder of the group being made up of animals with dorsal or ventral hippocampal lesions. The last group consisted of two animals with ventral hippocampal lesions and they were tested with subjects from another sleep study which was being carried out at the time. All recording sessions lasted for six hours starting at about 9.30 a.m. EEG and EMG activity was continuously monitored. The animals were observed constantly and frequent notations were made on the recording regarding their behavioural state. The rats were returned to their home cages between the first and second baseline recording sessions.

After the second baseline recording had been obtained the animals were returned to their home cages until the following morning when a 72-hour period of PS deprivation commenced. During the period of deprivation the animals remained in the water tanks all the time except for about 10 minutes each day when the water was changed. Care was taken during the 10 minutes to ensure that the subjects did not fall asleep.

The period of PS deprivation lasted until the morning of the tenth day when the first of two post-deprivation recordings was taken. During this time the animals could sleep freely. After recording the animals were returned to the PS deprivation situation where they remained till the morning of the eleventh day when the second and final post-deprivation recording was taken in the same way as the first.

All recordings were scored visually for wakefulness, slow-wave sleep (SS), and PS. Note was taken of the length of each period of sleep and wakefulness, as well as the order in which the periods occurred.

TABLE 2

Treatment of animals on each of the eleven experimental days

| Day Number | Experimental Treatment |
|---|---|
| 1 | Adaptation |
| 2 | Adaptation |
| 3 | Home Cage |
| 4 | Home Cage |
| 5 | *1st Baseline recording* |
| 6 | 2nd Baseline recording |
| 7 | PS deprivation |
| 8 | PS deprivation |
| 9 | PS deprivation |
| 10 | 1st Post-deprivation recording |
| 11 | 2nd Post-deprivation recording |

## RESULTS

### Histology

Histological analysis revealed that both the dorsal and ventral lesions extended through the hippocampus and into its surrounding structures. The dorsal lesions varied from animal to animal. In four animals the entire dorsal hippocampus was destroyed and in the other two only the very posterior part was left undamaged. Substantial parts of the fornix were damaged in all of the animals and in addition five of the lesions spread to the posterior septal area. In all of the lesions the damage spread ventrally to the thalamus. Dorsally the lesions extended through the corpus callosum into the cortex. Figs. 3a, b and c show diagrams of a typical dorsal lesion; (a) is typical of the most anterior part of the lesion, (b) is the area where damage was most extensive, and (c) shows the posterior part of the lesion.

The ventral hippocampal lesions were more constant. The damage extended throughout the lateral and ventral hippocampus. The geniculate body, optic tract, corpus callosum, cortex and thalamus were also slightly damaged as a result of the lesion. The diagrams in Figs. 4a, b and c, are representative of five animals with ventral lesions. The first diagram shows the most anterior part of the lesion, the second the middle of the lesion and the third the most posterior part. The brain of one ventral lesion animal was so extensively damaged that it was decided to exclude its results from the analysis.

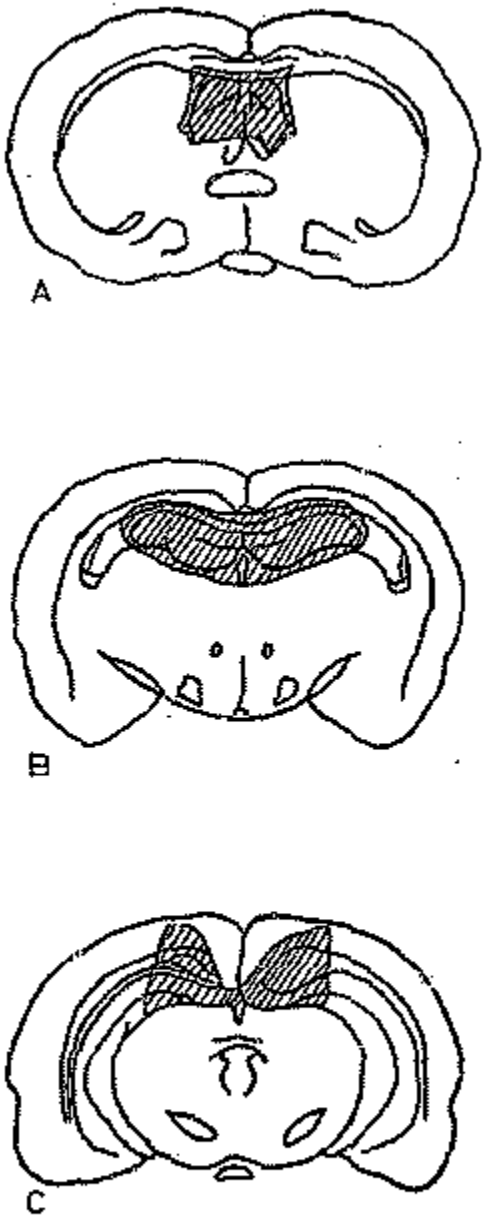



Fig. 3. The shaded area in the diagram shows the extent of a typical dorsal hippocampal lesion. A shows the *anterior* limit of the lesion, B the part of the lesion where damage was most extensive and C depicts the posterior part of a typical lesion.

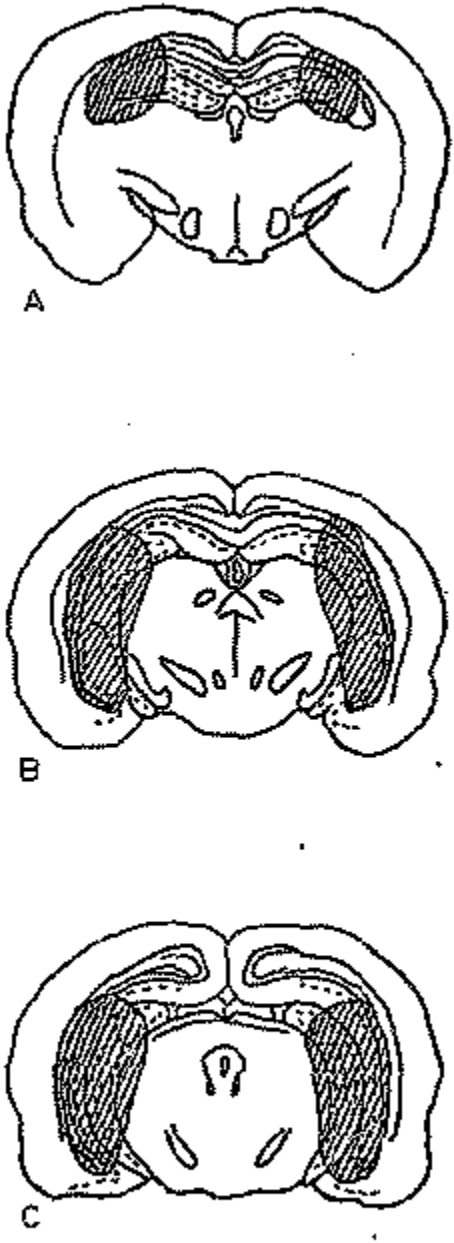



Fig. 4. A. The shaded area in the diagram shows the anterior limit of a typical ventral hippocampal lesion, B shows the part of the lesion where damage was most extensive and C depicts the posterior part of a typical lesion.

Data Analysis

The analysis of the data from this experiment was based on: the control group (C) with six rats, the dorsal hippocampal lesion group (DH) with six rats and the ventral hippocampal lesion group (VH) with five rats.

The following variables were analysed in Experiment 1: (a) the percentage of total sleep time spent having PS; (b) the percentage of sleep time during each quarter of recording time spent having PS; (c) latency to PS; the latency was measured from the onset of sleep to the first period of PS; (d) the duration of each PS period; (e) the mean number of PS periods; (f) the percentage of total sleep time spent having SS; (g) the percentage of total recording time spent asleep; (h) the latency to sleep measured from the start of recording until the animal showed SS for a period of at least 20 sec.

The lesions had very little effect on most of the variables that were measured in this experiment. However, some differences between the groups did appear. The most noticeable difference was that although the groups had almost the same percentage PS before deprivation, considerable differences appeared between the groups after deprivation (See Fig. 5). Group DH had the least PS and VH slightly more, but both groups had considerably less PS than Group C. This observation was confirmed by an analysis of variance (Table 3) which revealed a significant Groups X Sessions interaction ($F=8,91$, $df=2/14$, $p<.01$). Another major difference which can be seen in Fig. 5 is that the controls spent significantly more of the recording

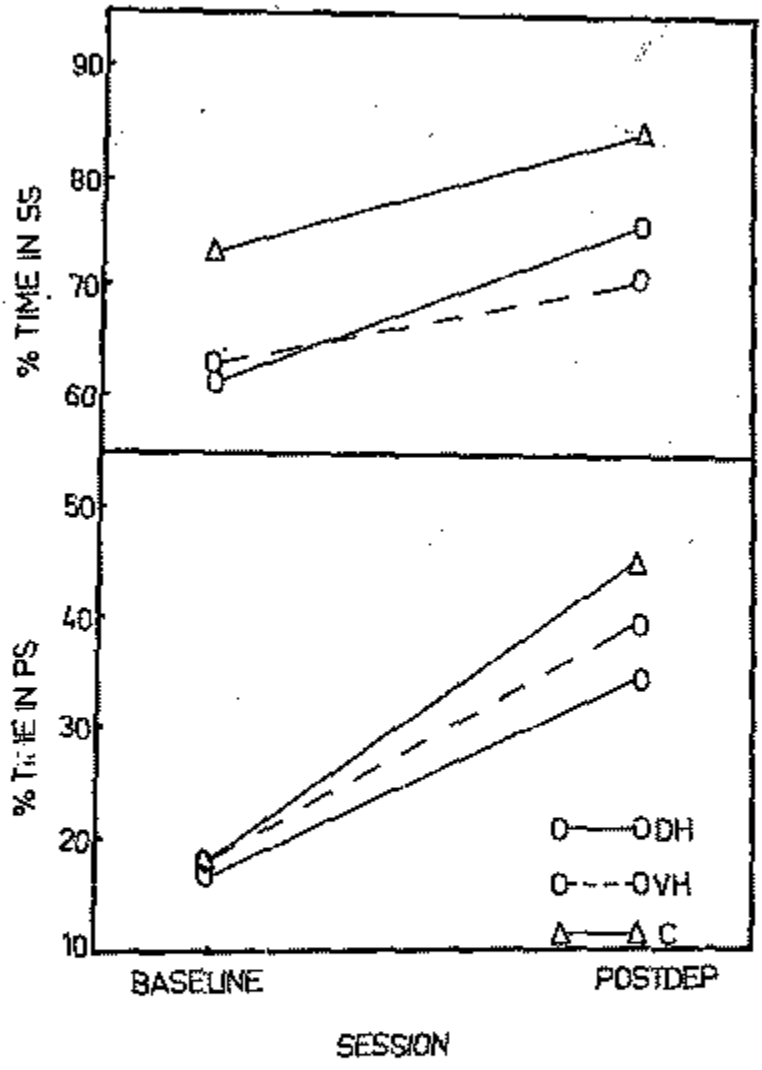



Fig. 5. Mean percentage of sleep time spent in PS and mean percentage of recording time spent in SS for *Groups C*, DH and VH before and after deprivation.

TABLE 3

Source table for the analysis of variance on percentage time in PS data

| Source of Variance | df | SS | MS | F | P |
|---|---|---|---|---|---|
| Total | 33 | 4896,20 | | | |
| *Between Subjects* | 16 | 454,97 | | | |
| A (Groups) | 2 | 224,28 | 112,14 | 6,80 | p<,01 |
| S/A | 14 | 230,69 | 16,48 | | |
| Within Subjects | 17 | 4441,23 | | | |
| B (Sessions) | 1 | 4206,03 | 4206,03 | 569,15 | p<,01 |
| AB | 2 | 131,69 | 65,85 | 8,91 | p<,01 |
| SB/A | 14 | 103,51 | 7,39 | | |

TABLE 4

Source table for the analysis of the percentage of recording time spent in sleep

| Source of Variance | df | SS | MS | F | P |
|---|---|---|---|---|---|
| Total | 32 | 5464,01 | | | |
| Between Subjects | 16 | 2196,23 | | | |
| A (Groups) | 2 | 1033,86 | 516,93 | 6,23 | p<,05 |
| S/A | 14 | 1162,37 | 83,03 | | |
| Within Subjects | 16 | 3267,78 | | | |
| B (Sessions) | 1 | 1198,57 | 1198,57 | 7,73 | p<,05 |
| AB | 2 | 54,13 | 27,07 | 0,17 | |
| SB/A | 13 | 2015,08 | 155,01 | | |

time sleeping than the other two groups ($F=6,23$, $df=2/14$, $p<.05$) (See Table 4).

The differences between the groups after deprivation seemed to be due to the brain damaged animals having fewer rather than shorter periods of PS. Fig. 6 indicates that there was practically no difference between the length of PS periods that each group had. However, Fig. 6 also indicates that Group DH, in particular, had considerably fewer PS periods than the other groups, although the group differences did not reach statistical significance ($F=1,59$, $df=2$).

From Fig. 7 which depicts the percentage PS in each quarter of the recording session, it can be seen that the differences between the groups did not appear at any specific stage during the recording session. However, Fig. 7 does show that the animals had much more PS in the earlier stages of the session after deprivation than they had before deprivation. This difference in distribution of PS is also evident from the analysis of variance (Table 5) which shows a significant Quarters X Sessions interaction ($F=28; 75$, $df=3/42$, $p<.001$).

The latency to SS and PS data revealed that all groups had similar latencies after deprivation. However, before deprivation there were wide variations in both the latency to SS and PS (See Fig. 8). A statistical analysis of latency to PS findings (Table 6) revealed a significant Groups X Sessions interaction ($F=4,94$, $df=2/14$, $p<.05$) but there were no statistically significant group differences in the latency to SS data.

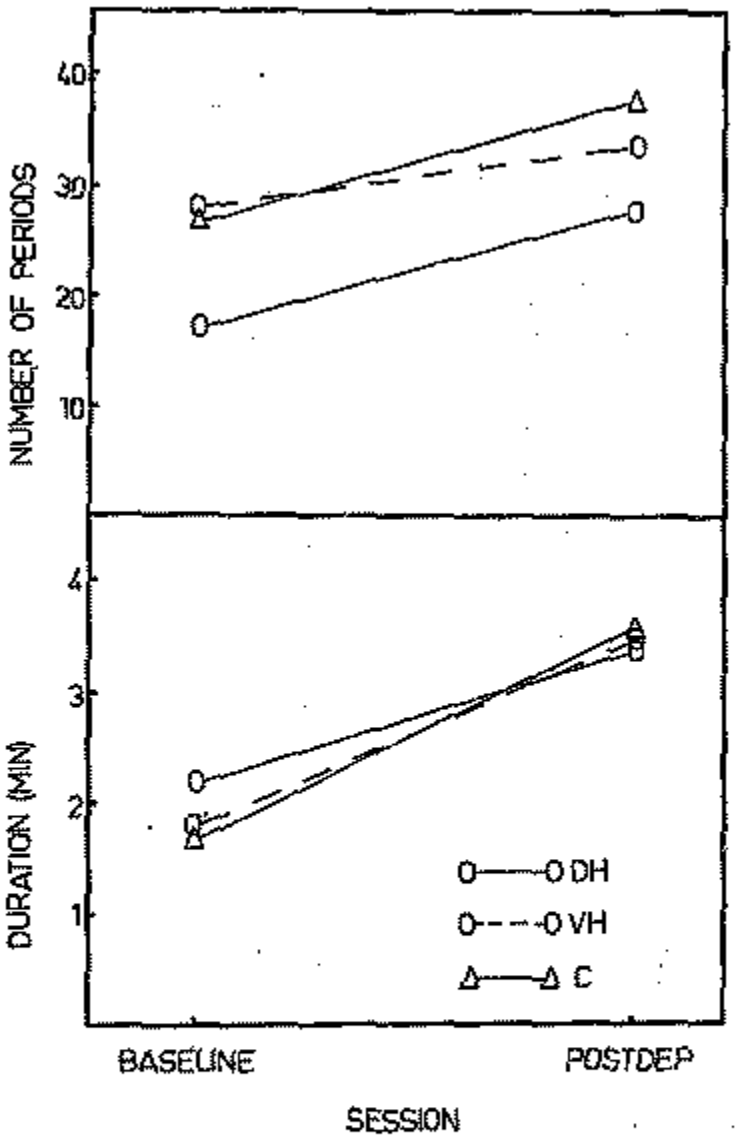



Fig. 6. Mean duration in minutes and mean number of PS periods for Groups C, DH and VH before and after deprivation.

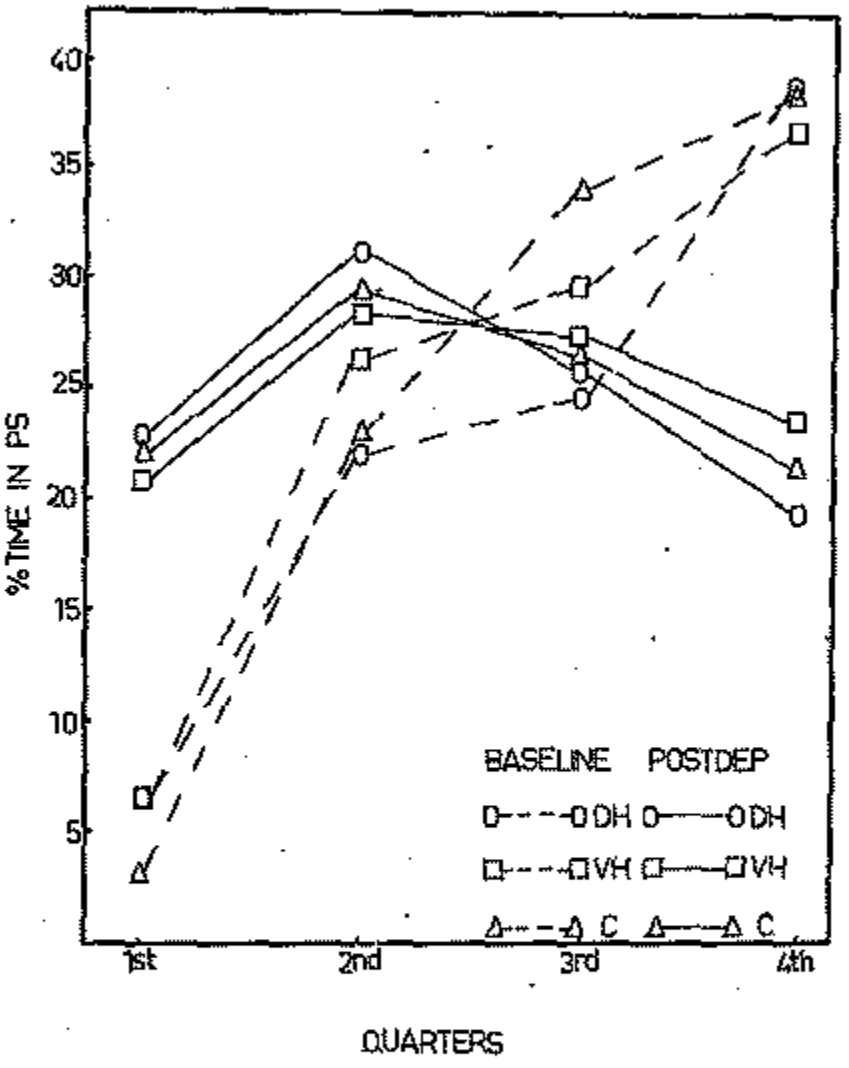



Fig. 7. Mean percentage of sleep time spent in PS during each quarter of recording time for Groups C, DH and VH before and after deprivation.

TABLE 5

Source table for the analysis of the distribution of PS through the four quarters of recording time

| Source of Variance | df | SS | MS | F | P |
|---|---|---|---|---|---|
| Total | 135 | 16787,15 | | | |
| Between Subjects | 16 | 196,76 | | | |
| A (Groups) | 2 | 22,05 | 11,02 | 0,88 | |
| S/A | 14 | 174,71 | 12,48 | | |
| Within Subjects | 119 | 16590,39 | | | |
| B (Sessions) | 1 | 15,83 | 15,83 | 1,27 | |
| AB | 2 | 19,33 | 9,67 | 0,77 | |
| SB/A | 14 | 174,82 | 12,49 | | |
| C (Quarters) | 3 | 5483,26 | 1827,75 | 26,59 | p<,001 |
| AC | 6 | 176,74 | 29,46 | 0,43 | |
| SC/A | 42 | 2887,46 | 68,75 | | |
| BC | 3 | 5098,16 | 1699,39 | 28,75 | p<,001 |
| ABC | 6 | 252,57 | 42,10 | 0,71 | |
| SBC/A | 42 | 2482,22 | 59,10 | | |

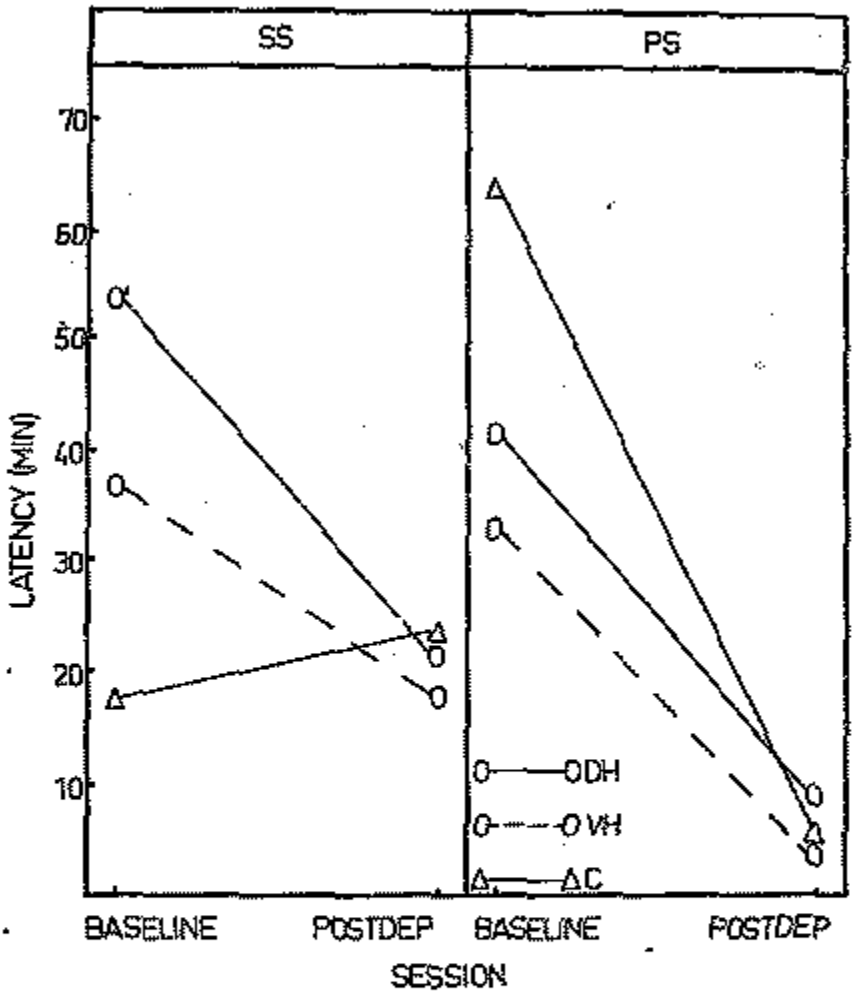



Fig. 8. Latency to SS and PS for Groups C, DH and VH under baseline and postdeprivation conditions.

TABLE 6

Source table for the analysis of latency to PE data

| Source of Variance | df | SS | MS | F | P |
|---|---|---|---|---|---|
| Total | 33 | 23424,67 | | | |
| Between Subjects | 16 | 4390,63 | | | |
| A (Groups) | 2 | 894,59 | 447,30 | 1,79 | |
| S/A | 14 | 3496,04 | 249,72 | | |
| Within Subjects | 17 | 19034,04 | | | |
| B (Sessions) | 1 | 14263,91 | 14263,91 | 71,40 | p<,01 |
| AB | 2 | 1973,17 | 986,59 | 4,94 | p<,05 |
| SB/A | 14 | 2796,96 | 199,78 | | |

## DISCUSSION

The results of this experiment gave no clear indication of the role of the hippocampus in PS even though the dorsal lesions had a disruptive effect on PS. Since the dorsal lesions included the caudal septal area and since Parmeggiani (1967) has reported that septal lesions disrupt PS under certain conditions, no firm conclusions can be drawn from this study about the role of the dorsal hippocampus. However, it is clear that the disruption by the dorsal lesions was due to the animals having fewer rather than shorter periods of PS.

The ventral hippocampal lesions, however were somewhat more accurate than the dorsal ones and were largely confined to the hippocampus. These lesions also disrupted PS slightly but the differences between Group C and Group VH were small and not significant.

While this experiment does not clearly indicate a disruption of PS by the hippocampal lesions, it is clear that SS was disrupted by both lesions. This finding would be predicted by Parmeggiani (1967) who reported that the hippocampus acts antagonistically to the reticular activating system (RAS). In the present experiment the removal of the hippocampus clearly resulted in a reduction in sleep indicating that the hippocampus could serve to counteract the desynchronizing effects of the RAS.

From the evidence provided by this experiment no conclusions about functional differences between the dorsal and ventral hippocampus can be drawn because of the inaccuracy of the dorsal lesions.

## EXPERIMENT 2

In this experiment only the effect of lesions in the dorsal hippocampus was examined. This approach was used because the results of the first experiment indicated that if the hippocampus is of importance in PS, it is the dorsal hippocampus which plays the most significant role. But the dorsal lesions encroached radically on extra-hippocampal structures whereas the ventral lesions were largely confined to the hippocampus. Therefore, Experiment 1 was replicated with more localised lesions in the dorsal hippocampus. In addition, this experiment was designed to examine the connection between PS and memory. Cohen and Dement (1966) and Cohen, Duncan and Dement (1967) have shown that electro-convulsive shock (ECS) delivered after PS deprivation significantly reduced pressure for PS. Dewan (1969) used this finding to support his hypothesis that PS is involved in memory consolidation. According to Dewan (1969) the ECS reduced PS pressure by disrupting memory and consequently reducing the amount of material requiring consolidation. Therefore, quantity of PS could be regarded as a measure of the amount of material requiring consolidation from STM to long-term memory (LTM). If this is so then animals could be placed in learning situations where the correct response is not readily observable and an indication of the amount of learning could be obtained by measuring PS without being hindered by the procedural and measuring difficulties which normally arise in animal learning experiments. Thus, far less easily defined learning situations could be used with animals if PS can be regarded as a measure of learning.

In the experiments reported here, the PS deprivation situation was regarded as a novel environment to which the animals had to learn to adapt themselves. However, it would be very difficult to accurately measure the learning that takes place in such a situation by observing the animal's behaviour. But if Dewan's (1969) hypothesis that quantity of PS can be regarded as a measure of amount of learning is correct, then some indication of learning could be obtained. This evidence could give some clue as to the reason for the deficit shown by animals with hippocampal lesions in adapting to novel stimuli (Leaton, 1965) and new environments (Meissner, Ervin and Stevens, 1966). If the adaptation deficit is due to some memory defect this would probably be reflected in the quantity of PS and would probably not be reflected in PS if it is due to some other cause.

The hippocampus also becomes implicated in this aspect of the experiment since Hostetter (1968) has found that hippocampal lesions weaken the retrograde amnesic effect of ECS. If Dewan's hypothesis is correct then it would be expected that the ECS would have no effect on PS rebound shown by hippocampal animals deprived of PS.

Thus the first part of Experiment 2 was designed to examine the effects of dorsal hippocampal lesions on PS. The second part, however, examined the effects of ECS on PS rebound of dorsal hippocampal and control animals in order to evaluate the possible link between PS, hippocampal lesions and memory.

## PROCEDURE

### Surgery

The 12 animals used in this experiment were divided into two groups of six animals each. Group DH received bilateral lesions in the dorsal hippocampus and Group C served as operated controls. A Grass Model LM 3 Radio Frequency Lesion Maker was used to prepare the lesions. The current was delivered to the brain through the 0,75 mm uninsulated tip of a needle for 30 sec. The needle was inserted into the brain at the co-ordinates given in Table 7.

Control operations were carried out by inserting the needle into the brain at the co-ordinates given in Table 7. These co-ordinates were identical to those used for the experimental animals except that the ventral co-ordinate did not permit the needle to enter the hippocampus. No current was delivered to the brains of control rats. EEG and EMG electrodes were implanted at the time of the operation.

### Apparatus

A Lafayette shocker, Model A615, in series with a Hunter Timer was used to deliver an ECS (20 mA A.C. for 1,3 sec) to the rats. The shocker was connected to the animal's ears by means of crocodile clips which had been wrapped in cloth and dipped in 1% saline solution.

### Experimental Procedure

This experiment consisted of two parts. The procedure for part 1 was identical to that of the first

experiment except that recordings were taken for four, instead of six hours, starting at about 5.30 p.m.

At the completion of part 1, the rats were returned to their home cages for a rest period of five days. After this part 2 commenced and they were again deprived of PS for 72 hours. At the end of the 72-hour period all animals were anaesthetized with ether and given an ECS. Ether anaesthesia was used to eliminate the fatalities generally caused by ECS (Fishbein et al., 1970). About 15 minutes after the ECS had been given the rats' EEG and EMG were monitored for a period of four hours.

Records were scored as before.

TABLE 7

Co-ordinates in mm that were used for lesion and control operations

| Group | Number of subjects | Posterior to bregma | Lateral to midline | Ventral to surface of brain |
|---|---|---|---|---|
| Dorsal lesion | 6 | 4,00 | 1,25<br>2,75 | 2,5 |
| Control | 6 | 4,00 | 1,25<br>2,75 | 2,00 |

## RESULTS

### Histology

The histological analysis carried out on the brains of rats used in Experiment 2 showed that most of the damage was confined to the anterior-dorsal hippocampus. The most anterior damage was at the level depicted in Fig. 9a while the posterior margin is depicted in Fig. 9c. Fig. 9b shows the area where the damage was most extensive and it can be seen that little damage extended beyond the hippocampus except into the corpus callosum and cortex. Three of the lesions extended ventrally to cause very slight damage to the thalamus. Figs. 9a, b and c can be regarded as being representative of all the lesions.

In contrast to the results of Experiment 1, very few differences were found between the groups in this experiment. Table 8 summarizes the findings of this experiment and it can be seen that the groups did not differ significantly on various measures of PS before deprivation, after deprivation or after ECS. The lack of group differences was apparent with respect to percentage PS, latency to PS, latency to SS, distribution of PS through the recording session, mean duration of PS periods and number of PS periods. The only significant difference to appear in the results was related to the total amount of sleep (Fig. 10). Although the groups had similar quantities of sleep before and after PS deprivation, Group DH had very much less sleep than Group C after deprivation followed by ECS. This observation was confirmed by an analysis of variance (Table 9) which revealed a

significant Groups x Sessions interaction ($F=5,32$, $df=2/20$, $p<.05$).

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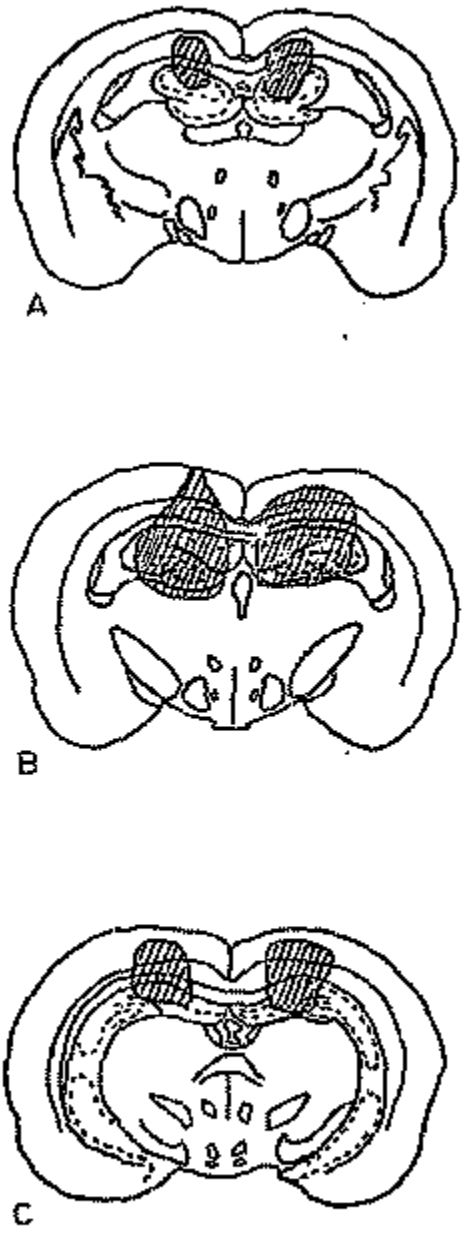



*Fig.* 9. A, B and C. Representative diagrams of the anterior (A), middle (B) and posterior (C) parts of a dorsal hippocampal lesion.

TABLE 8

Summary of the findings where no significant differences were found between the groups.

| Measure | Baseline | | After deprivation | | After deprivation and ECS | |
|---|---|---|---|---|---|---|
| | C | DH | C | DH | C | DH |
| Percentage PS | 13,30 | 16,30 | 40,30 | 41,10 | 37,10 | 36,40 |
| Latency to PS (min) | 26,20 | 40,30 | 4,70 | 5,60 | 8,90 | 7,50 |
| Latency to SS | 51,50 | 34,40 | 26,30 | 23,70 | 21,40 | 31,50 |
| Duration of PS periods (min) | 1,57 | 1,89 | 3,01 | 2,74 | 2,70 | 2,50 |
| Number of PS periods (min) | 9,60 | 10,40 | 22,60 | 24,80 | 24,30 | 20,30 |
| Mean percentage 1st | 1,90 | 5,00 | 21,10 | 17,30 | 14,80 | 19,C |
| of sleep time 2nd | 29,70 | 19,20 | 28,60 | 28,90 | 31,10 | 29,10 |
| having PS during 3rd | 25,50 | 33,50 | 27,50 | 25,70 | 26,60 | 21,10 |
| each hour of 4th | 35,50 | 41,20 | 22,90 | 26,90 | 27,70 | 30,40 |
| recording | | | | | | |

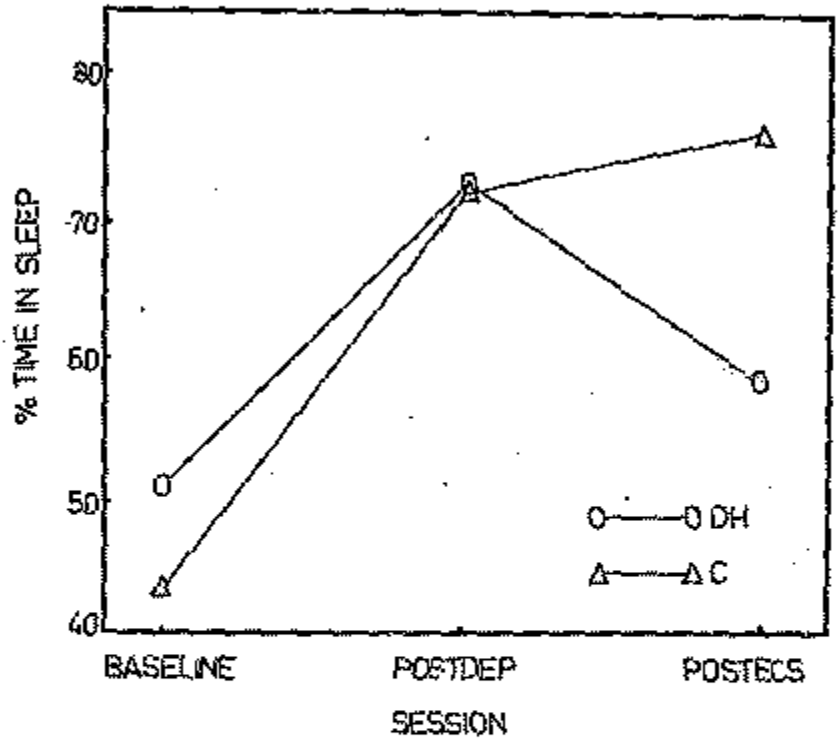



Fig. 10. Mean percentage of total recording time spent in sleep for Groups C and DH during baseline, postdeprivation and post ECS sessions.

TABLE 9

Source table for the analysis of the percentage of recording time spent in sleep

| Source of Variance | df | SS | MS | F | P |
|---|---|---|---|---|---|
| Total | 35 | 9159,07 | | | |
| Between Subjects | 11 | 2080,79 | | | |
| A (Groups) | 1 | 85,93 | 85,93 | 0,72 | |
| S/A | 10 | 1194,86 | 119,49 | | |
| Within Subjects | 24 | 7078,28 | | | |
| B (Session) | 2 | 4249,25 | 2124,63 | 23,01 | p<,001 |
| AB | 2 | 982,51 | 491,26 | 5,32 | p<,025 |
| SB/A | 20 | 1846,52 | 92,33 | | |

## DISCUSSION

Contrary to the findings of Experiment 1 the results of Experiment 2 indicated that the dorsal hippocampus did not play a direct role in the PS process. The most likely explanation of the different findings of Experiments 1 and 2 seems to be in terms of the extent and location of the lesions. The histological analysis has shown that the lesions of Experiment 1 were very much larger and included extra-hippocampal structures including the posterior septal area and the major hippocampal afferent and efferent pathways. Whereas, the brain damage in Experiment 2 was confined to the anterior dorsal hippocampus. It appears then, that the hippocampal pathways or the caudal septal area either alone or in combination with the hippocampus have to be damaged before PS is disrupted.

Apart from anatomical differences, there were many procedural differences between Experiment 1 and the first part of Experiment 2. In the first experiment recordings were taken in the morning and in the second in the evening. Furthermore, the animals used in Experiment 1 were housed in a normal light-dark cycle while those used in Experiment 2 were kept under conditions of constant light. It seems unlikely that these factors could account for the inconsistent findings unless the time of day or lighting conditions affected hippocampals and controls differentially. Finally the differences could have been caused by the shorter periods of testing used in the second study. However, an examination of the data from the first four hours of Experiment 1 indicates that this was not the cause of the discrepant findings.

The data from both these experiments gave little support to theories regarding hippocampal involvement in PS. Parmeggiani's (1967) theory that hippocampal theta activity served to stabilize PS was not directly investigated since the animals were only tested in a quiet environment. However, Parmeggiani's theory could not account for the decline in PS following deprivation which was observed in the dorsal hippocampal animals of Experiment 1. Furthermore, the recordings do not show any evidence of a tendency among the brain-damaged animals to wake up during the flattening of EMG instead of entering PS. Lastly Fig. 6 and Table 8 show that the length of PS periods of the lesioned animals in both experiments were much the same as the controls. Where a decrease in the amount of PS was evident in Experiment 1, Fig. 6 indicates that this was due to having fewer rather than shorter periods of PS.

Both Hernandez-Peon (1966) and Torda (1967; 1968a; 1968b; 1968c; 1969a; 1969b) proposed that removal of the inhibitory influence of the hippocampus would increase the amount of PS. These theories have clearly received no support from either experiment.

A likely explanation of results of Experiment 1 can be made in terms of information processing. A number of studies have suggested that PS is important for information processing (Breger, 1967; Evans and Newman, 1965; Fishbein, 1969a; 1969b; 1969c; Fishbein et al, 1970; Greenberg, 1970; Greenberg et al., 1970; Greenberg and Dewan, 1968; Hawkins, 1966; Jouvet, 1963; Lucero, 1970; Newman and Evans, 1965; Pearlman and Greenberg, 1968; Swarz and

McGaugh, 1970). Dewan (1969) proposed that the more information there is to process the more PS the animal will have. However, it is known that hippocampal animals have difficulty in habituating to novel stimuli (Leaton, 1965) and new environments (Meissner et al., 1966). Therefore, since the animals do not readily adapt to the PS deprivation environment it is possible that they have little information to process and consequently little PS. Clearly this explanation, too, does not receive any support from the second experiment.

The results from part 2 of the second experiment have shown that the ECS had very little effect on the quantity of PS shown by the animals in both groups. This finding is at variance with those of Cohen and Dement (1966) and Cohen et al. (1967) who reported that ECS reduced PS rebound. It is difficult to explain these discrepant findings because the studies are not directly comparable. Firstly Cohen and his co-workers used cats and in this experiment rats were used. Secondly the intensity of ECS varied in the two studies. In this experiment an ECS of 20 mA for 1,3 sec was delivered whereas Cohen et al. (1967) used 12 mA for 1,0 sec. However the variations in these studies do not alter the doubt that is cast on Dewan's (1969) explanation for the reduction of PS rebound which was in terms of the retrograde amnesia (RA) commonly caused by ECS (Brady, Stebbins and Hunt, 1953; Buresova, Bures and Gerbrandt, 1968; Chorover and Schiller, 1966; Duncan, 1949; Gerbrandt, Buresova and Bures, 1968; Glickman, 1961; Heriot and Coleman, 1962; Hughes, Barrett

and Ray, 1970; Hunt and Brady, 1951; Madsen and McGaugh, 1961; Pinel, 1969; Quartermain, Paolino and Miller, 1965). Dewan's hypothesis was that the RA resulting from ECS caused a decrease in the amount of material requiring consolidation and therefore a reduction in PS. While there is no direct evidence that the ECS administered in the present experiment caused RA it seems reasonable to assume that RA was present since the same level of ECS administered by Wolfowitz and Holdstock (1971) caused RA in rats. Consequently, it is reasonable to assume that the ECS did cause RA but did not reduce PS rebound.

## EXPERIMENT 3

The results of part 2 of the second experiment were contrary to what would be predicted by Dewan's (1969) hypothesis that ECS, which disrupts memory consolidation, reduces pressure for PS. Neither do the results of Experiment 2 give any clear evidence in support of Hostetter's (1968) finding that hippocampal lesions protect memory from the effects of ECS. However, Experiment 2 only examined both Dewan's and Hostetter's points of view in a very indirect way. In the third experiment the idea that PS is necessary for memory consolidation *and* the idea that hippocampal lesions protect memory from the effects of ECS was examined far more directly than in the second experiment. This was done by examining the effects of ECS on the performance of an avoidance response by hippocampal and control animals which had been deprived of PS.

## PROCEDURE

### Surgery

The 40 rats used in the third experiment were divided into four groups of 10 animals each. Groups DH-ECS and DH-No ECS (DH-NECS) received bilateral lesions of the dorsal *hippocampus* and Groups C-ECS and C-NECS served as operated controls. The co-ordinates for the third experiment were identical to those used in the second experiment.

### Apparatus

Apparatus consisted of a 72,0x30,0x28,0 cm shuttle box with a black and a white compartment separated by an 8,0 cm high barrier. A 1,0 mA A.C. current was delivered to alternate bars of the grid floor in the black compartment. ECS was delivered in the same way as in the previous experiment.

### Experimental Procedure

After surgery and the recovery period all animals were trained in a one-way shuttle box avoidance task. The one-way shuttle box avoidance task was used because it has been reported (Douglas, 1967) that this task does not favour learning in either control or hippocampal animals. Immediately before the first trial on the training session the animals had a one-minute adaptation period in the shuttle box. Each trial consisted of placing the rat in the black compartment and sounding a buzzer immediately. Shock followed 10 sec later. The buzzer and shock continued until the rat jumped into the white compartment. The shock could be avoided by jumping the barrier before the 10-sec. interval elapsed. Training was continued

until a criterion of three successive avoidance responses was reached. The number of trials to criterion was recorded.

Following training all animals were deprived of PS for 72 hours. Following deprivation all animals were given an ether anaesthetic and Groups DS-ECS and C-ECS received one ECS. The rats were returned to their home cages for a 48-hour recovery period and then tested for retention in the shuttle box. The procedure for the retention test was the same as that for the training session except that the one-minute adaptation period was omitted.

## RESULTS

### Histology

The lesions in this experiment were very much the same as those of Experiment 2.

The data obtained from this study revealed that neither the ECS nor the hippocampal lesions had any marked effect on performance during the retention sessions. However, inspection of Fig. 11 shows that the hippocampal groups had difficulty in learning the task. The relatively slow learning of the hippocampals as opposed to the controls made it necessary to analyse the results by means of an analysis of covariance. Even adjusting the retention scores in terms of the original learning failed to reveal any group differences (Table 10). However, interpretation of the results was complicated by the finding that an analysis of variance carried out on the training-retention difference scores (Table 11), followed by a Duncan's New Multiple Range Test, showed that the hippocampals did have a significant savings score ($F=10,5$, $df$ 3/36, $p<.01$). However, the significant savings score was obviously strongly influenced by the large number of trials taken to learn the task (See Fig. 11).

It is also apparent from Fig. 11 that Group C-ECS showed very little training-retention improvement in relation to the other groups and that both groups which received an ECS did not perform as well as the N-ECS groups in the retention trials. These differences, however, are small and do not approach significance.

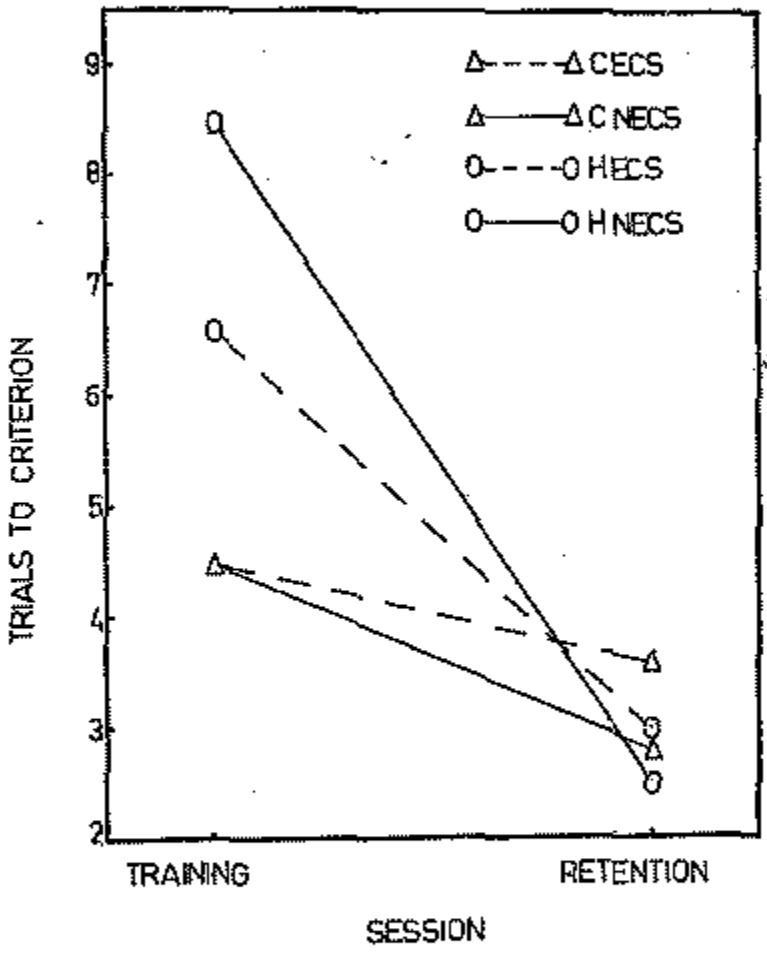



Fig. 11. Trials to criterion for Groups C-ECS, C-NECS, H-ECS and H-NECS during training and retention *sessions*.

TABLE 10

Source table for the analysis of covariance on the trials to criterion data

| Source of variance | df | SS | MS | F |
|---|---|---|---|---|
| Total | 38 | 151,57 | | |
| Between Groups | 3 | 5,8 | 1,93 | 0,46 |
| Within Groups | 35 | 145,77 | 4,16 | |

TABLE 11

*Source table for the analysis of covariance on the* training-retantion difference scores

| Source of Variance | df | SS | MS | F | P |
|---|---|---|---|---|---|
| Total | 39 | 1119 | | | |
| Between Groups | 3 | 523 | 174,00 | 10,[illegible] | p<.01 |
| Within Groups | 36 | 546 | 16,55 | | |

## DISCUSSION

The results of Experiment 3 gave no clear indication of the possible role of the hippocampus or PS in memory consolidation. As far as the hippocampus is concerned no conclusion can be drawn from the data which showed that the hippocampals had a significantly greater savings score than the controls, irrespective of whether or not an ECS was given. The reason for the significantly greater savings score was the large number of trials that the hippocampals took to learn the task. This learning problem was not expected because it has been reported that hippocampals do not have difficulty in mastering a one-way shuttle box avoidance task (Niki, 1962). Furthermore, this initial learning problem cannot be explained in terms of some recent theories on the function of the hippocampus. The "working memory" hypothesis or response inhibition theory proposed by Douglas (1967) do not seem to explain this finding. The information processing theory proposed by Bagshaw et al. (1965) would probably explain the results of this experiment best in that animals which have difficulty in processing the information received in the shuttle box would have difficulty in mastering the task. However, while this would explain the difficulty on the training trials, it does not explain the retention performance. It is, therefore, obvious that lesions such as those used in this study do not prohibit the formation of permanent memories but may hinder their early formation. Once these permanent memories are acquired they influence performance as effectively as the memories of control animals

In addition, there is little evidence supporting the idea that hippocampal lesions protected memory from the effects of ECS. Examination of Fig. 11 revealed that the C-ECS group took longer to show mastery on the retention trials than the DH-ECS group. However, because of the insignificant differences between the groups no conclusions supporting the idea that hippocampal lesions protect memory from ECS can be made.

Similarly, no conclusive evidence supporting the idea that PS is involved in memory consolidation was found. The analysis of covariance indicated that the ECS had no effect on the memories of any of the animals. This finding is in conflict with those of Fishbein (1969); Fishbein et al. (1970); Pearlman and Greenberg (1968) and Wolfowitz and Holdstock (1971), who have found that ECS disrupts memory after the animals have been deprived of PS for a number of hours, suggesting that PS is essential for consolidation of memory. However, the results of the present study, at least for the C-ECS group, would be predicted by the reports of Buresova et al. (1968); Chorover and Schiller (1966) and Quartermain et al. (1965) that consolidation from STM to LTM takes only a few seconds. Other authors have reported that consolidation takes much longer (Barrett and Ray, 1969; Duncan, 1949; Glickman, 1961; Heriot and Coleman, 1962; Hughes et al. 1970; McGaugh and Petrinovich, 1966; and Pfingst and King, 1969). The question of the length of time it takes for consolidation from STM to LTM to occur seems to have been ignored by authors proposing that PS is essential for memory consolidation. If, in fact, consolidation does

only take a few seconds, then obviously PS is not necessary. However, if the time period is much longer, it is quite possible that the animals may have had PS between training and the time ECS was administered. In studies where it has been shown that consolidation takes relatively long it should be ensured that the animals do not have PS during the interval between training and testing, otherwise no conclusive answers as to either the time required for consolidation to take place or the necessity of PS for consolidation can be made.

A further drawback of experiments investigating the need for PS in consolidation is that insufficient attention has been paid to the effects of environment on learning. Agranoff (1967) found that fish which had been taught an avoidance response failed to consolidate if they were left in the tank in which the learning took place. His interpretation of this data was that the stimulating environment prevented consolidation from occurring. The water tank deprivation technique also requires that the animal is placed in what could be a highly stimulating environment shortly after learning. These unusually stimulating surroundings could, if Agranoff is correct, prevent consolidation equally as well as the PS deprivation. Since the ECS had no significant effect on the performance of control animals, no real assessment can be made of the possible protection that the hippocampal lesion may have afforded memory.

## GENERAL DISCUSSION

The three experiments of this study have raised a number of questions. The results of Experiment 1 are the most suggestive since they indicate that both the dorsal and, to a lesser extent, the ventral hippocampus or structures closely related to them play a definite role in PS. However, from the results of Experiment 2 it appears that the anterior-dorsal hippocampus "per se" does not play a direct role in PS regulation. Therefore, while there is some suggestion of hippocampal involvement in PS the precise area of the hippocampus or its related structures which were responsible for the results of Experiment 1 have not been determined. In addition, the results of the second part of Experiment 2 and Experiment 3 have not revealed the role of the hippocampus in PS.

The results of Experiment 1 also indicate that the caudal septal area is very possibly involved in the PS process. The septal area was the only extra-hippocampal structure, other than the pathways, to be substantially damaged by the dorsal lesions indicating, in agreement with Parmeggiani's (1967) finding, that lesions in the septal area can disrupt PS. Further weight is added to the argument that the septal area is involved in PS when it is considered that the ventral hippocampus also has close anatomical links with the septal area (Green, 1964). But the association of the hippocampal lesion with the septal area is not sufficient to explain the results because the lesions of Experiment 2 which did not actually damage the septal area also probably disrupted the septo-hippocampal pathways.

It is apparent that a study of the effects of septal lesions could throw more light on the role of the septal area alone as opposed to the hippocampus alone. Even though Parmeggiani (1967) examined the effects of septal lesions on PS his main concern was to study the effects of disrupting hippocampal theta activity and the septal lesion was a means to achieve this end. From the results of the experiments reported here there is evidence suggesting that the septo-hippocampal complex may play an important role in PS even though the hippocampus alone does not seem to play an essential role in the PS process.

As has been indicated earlier, the second and third experiments attempted to investigate a number of theories linking the hippocampus, PS and memory. The results gave no conclusive support to any of the theories that were discussed. Nevertheless, many of the results were in the direction that would be predicted by the theories, but trends were small and could, at best, be regarded as be indicative. Only more detailed experiments can fully explain the differences between the findings here and those reported by other authors.

When interpreting the results of these experiments care must be exercised since they suffer from the weakness of all lesion studies that the differences may not be attributable to the damaged structure itself. Brain damage in one area may have a secondary effect on some other structure which is directly responsible for the function which is being considered. Also direct comparisons of results from different species may not be valid because phylogenetic

differences in the development of the hippocampus may have a bearing on the function of the hippocampus. Also it is clear from human studies that lesions in the hippocampus or its related structures cause qualitative differences in dreams which cannot be measured in animals (Greenberg et al., 1968; Torda, 1969a; 1969c). Finally, the evidence presented here and elsewhere also suggests that the hippocampus should not be regarded as a single structure with a unitary function (Adey, 1964; Elul, 1964; Goodfellow and Niemar, 1961; Gross et al., 1965; Hughes, 1965). Even a dorsal - ventral differentiation is not sufficient in the light of results of Experiments 1 and 2 and other findings which showed that anterior - dorsal and posterior - dorsal lesions can have different effects on behaviour (Kimura, 1958).

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