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Effects of the anterior temporal lobe lesions, separate or combined with hippocampal damage, on spatial delayed responses guided by auditory stimulus

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Abstract. Seventeen dogs were trained in a three-choice auditory spatial delayed response task, guided by auditory stimulus, at a 10 s delay to a criterion of 90% correct responses in 90 consecutive trials. Four dogs then received bilateral anterior temporal lobe lesions (AT), 6 dogs received hippocampal lesions (H), and 7 dogs served as controls (C). Group C reached postoperative criterion immediately while groups AT and H needed additional training. When subsequently tested at longer delays and with distractions, the group H animals performed more poorly than either the AT or C animals. Further, the group H dogs were again impaired when they retrained at a 10 s delay. In the second phase, the group H and AT animals received a second lesion forming a group (HAT) with bilateral lesions to both the hippocampus and the anterior temporal lobe. Unexpectedly, dogs from group HAT were unimpaired in either postoperative retraining or during performance task and distractions. The results emphasize the importance of the hippocampus in spatial delayed response with an acoustic cue. Effect of combined lesions after extensive training is discussed. Data might support the view, that the hippocampus plays time limited role in memory storage.

Key words: anterior temporal lobe, hippocampus, lesion, memory of place, dogs

INTRODUCTION

Numerous studies have shown that large bilateral lesions of the medial temporal lobe cause memory loss. In monkeys recognition memory deficits produced by combined anterior (including amygdaloid complex, periamygdaloid cortex and the anterior part of entorhinal cortex) and posterior (including the hippocampal formation, the parahippocampal cortex and the posterior part of the entorhinal cortex) medial temporal lesions, made by aspiration, produce much more severe deficits than lesions to either region alone (Mishkin 1978, Murray and Mishkin 1984). However, the anterior and posterior medial temporal regions are not functional equivalent. Lesions to the anterior parts of the temporal lobe are critical for stimulus-reward association (Mishkin et al. 1982) as well as stimulus-stimulus association, i.e. retention of both cross-modal (Murray and Mishkin 1985) and intramodal stimuli associations (Murray and Gaffan 1994). On the other hand, acquisition of intramodal associations was unaffected by such lesions (Murray et al. 1993). Ablations of the posterior part of the temporal lobe have found to be critical for memory of place (Parkinson et al. 1988, Angeli et al. 1989).

Although, more detailed examination of the structures within the temporal lobe structures is limited by surgical techniques, that might be provided, at these regions of the brain, recently, a considerable number of publications is concern about the separate role of the amygdala, the hippocampus and underlying cortical areas on different kinds of memory. Murray et al. (1996) have shown dissociation of contributions of the anterior rhinal cortex and the amygdala to the visual recognition memory and food preferences in monkey. More recent data indicates that the amygdala plays a critical role in associating environmental stimuli with the value of particular reinforcers (Malkova et al. 1997, Thornton et al. 1998). The rhinal cortex appeared to play crucial role for visual and tactual recognition memory (for the review see Murray 1996). There is a number of evidence on rats indicating the role of the hippocampus in spatial learning and memory (for review see O'Keefe and Nadel 1979, Barnes 1988, Nadel 1995). On dogs it has been shown that bilateral hippocampal aspiration, which spare underlying cortical areas (Kowalska and Kosmal 1992), cause an impairment of spatial delayed responses directed by acoustic stimulus, trained in three choice Nencki Testing Apparatus (Kowalska 1995). However, in monkeys excitotoxic lesions limited to the hippocampus and amygdala did not impair both object and location recognition (Murray and Mishkin 1998). On the other hand, it has been shown that neurotoxic hippocampal lesion impaired spatial scene learning, and that impairment is equal to that observed after the rhinal cortex lesion in the monkey (Murray et al. 1998). Studies on rats indicate that lesions to the perirhinal cortex can disrupt performance of spatial task in the water maze (Liu and Bilkey 1998a) and radial arm maze (Liu and Bilkey 1998b), as well as delayed non-matching-to-position task (Wiig and Burwell 1998). Moreover, Suzuki et al. (1997) had documented that monkeys entorhinal cells receive sensory information about both object and spatial locations and their activity carries information about objects and locations held in short-term memory. This might suggest that within the temporal lobe structures, spatial domain might not be limited only to the hippocampus.

On the other hand, the results obtained on monkeys were related to the spatial responses cued by visual stimuli, thus, it was interesting to investigate the role of the temporal lobe structures in spatial delayed responses guided by acoustic stimulus, the task which was extensively used on dogs (Ławicka 1969, 1972). However, it is important to stress that so far, in dogs, there is no developed a good technique for surgical lesion of the posterior part of the rhinal cortex and the parahippocampal cortex which underlies the hippocampus. Therefore, in the present experiment the effects of lesions limited to the anterior temporal lobe structures on the retention and performance spatial delayed responses signalized by acoustic cue were investigated. The results has been compared with the effects of the lesions limited to the hippocampus on dogs trained in the same task described earlier (Kowalska 1995). Additionally, the effect separate *versus* combined lesions to both regions of the temporal lobe brain was examined.

METHODS

Subjects

Seventeen experimentally naive mongrel dogs, weighing 8-15 kg., were used in the study. Each animal was housed individually in a cage (2.7 x 1.2 x 3.0 m.) with free access to water. Food was given once a day, 15-20 h. before testing. Dogs were randomly assigned to 4 experimental groups. Four dogs (group AT) received bilateral removal of structures within the anterior temporal lobe (including the amygdala and anterior part of the rhi-

nal cortex). Six dogs received bilateral hippocampal lesions (Group H). Three dogs received a control lesion in which only brain tissue overlying the hippocampus was removed and four dogs were intact controls. Since there were no significant difference between both controls in each stage of training, they were combined in one control group (C). The pre- and postoperative training data for the Group H and control groups were earlier presented (Kowalska 1995). Group HAT *(n = 6)* consisted of dogs with bilateral combined hippocampal and anterior temporal lesions. These dogs all received two stage lesions. Three dogs were first given bilateral hippocampal lesions and then anterior lobe lesions. The other three received anterior lesions first and then hippocampal lesions.

Apparatus

The dogs were trained in a three-choice Nencki Testing Apparatus, which consists a room, 4.25 x 8.5 m. with three food dispensers located on the floor (Fig. 1). Each food dispenser contained 16 foodcups mounted on a rotating disc. The dogs had access to one food cup at a time through the well on the top of the box provides. Rewards consisted of approximately 5 g of cooked meat. A photocell is mounted in front of each food box. Speakers located above each feeder emitted a 100 Hz, 65 dB tone/pulse when activated by the experimenter. A 83 cm high wooden screen was interposed between the starting platform and the experimenter. During the delay period the animal was held at the starting position by a 82 cm long leash attached manually to a hook on the wooden screen. An electronic timer is activated when the animal is leashed to the hook. Releasing the dog activates another timer, which stops when the animal, approaching the foodwell, interrupts the photocell beam.

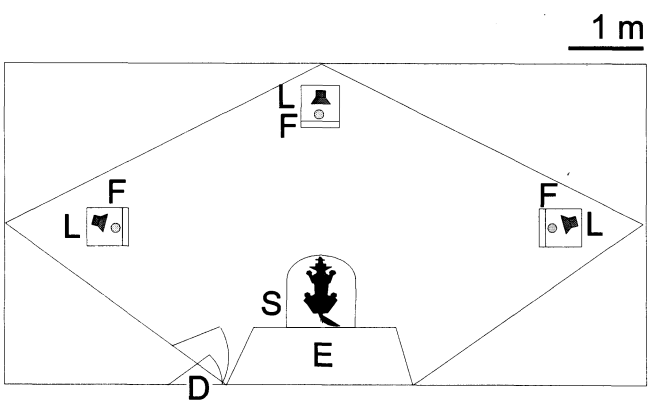

Fig. 1. Scheme of the Nencki testing apparatus. L, loudspeaker; F, feeder; S, starting platform where subject is waiting; E, location of experimenter; D, entrance door.

Testing procedure

PRELIMINARY TRAINING

The training started with familiarization of the dog to the experimental room. On the following day the animals were provided experience with eating from the feeders. The dogs were then shaped to approach the feeder at the sound of the rotating disc and to return to the starting platform after receiving food. Training continued until the criterion of 100% correct approaches in one session (15 trials) was reached.

ACQUISITION TRAINING

An auditory cue was introduced after the shaping procedure has been completed. At the beginning of each trial, the animal was leashed to the starting platform until a 3 s tone was emitted from one of the three speakers. If the dog approached the feeder associated with the activated speaker, it was reinforced by food.

Acquisition training was divided into two stages. In the first stage, the dog was released from the leash immediately after the cessation of the tone (0 s delay). Training continued until the dog met a criterion of 90% correct responses in 90 consecutive trials (6 experimental sessions). In the second stage the procedure was identical except that a 10 s delay was introduced between the presentation of the tone and the release of the animal. There were 15 trials in daily session (5 runs to each of feeder). The trials across feeders 1-3 were distributed pseudorandomly.

Correction trials were used when dog's performance dropped below 11 correct responses within the session. In that case whole erroneous trial was repeated.

FIRST SURGERY

Following acquisition training the experimental dogs were sedated by Combelen (Propriomylopromazine) 0.2 ml/kg combined with Atropine (0.05%) 0.3 mg/kg (i.m.), and then anesthetized with Nembutal (25 mg/kg i.p.). Bilateral lesions were made by aspiration with the aid of an operating microscope. Four dogs from Group

AT had removed anterior part of the temporal lobe including a part of the amygdala and anterior portion of the rhinal cortex. The hippocampus was removed through an opening in the visual association area of the cortex (about 7 mm in diameter) in an area belonging to the visual association area (Adrianov and Mering 1956); the white matter under it, which was localized on the suprasylvian gyrus, between the anterior end of fissura ectolateralis and fissura ectosylvia (Kreiner 1966) was also removed. More details of the method of the hippocampal lesion are provided elsewhere (Kowalska and Kosmal 1992). The seven control animals (Group C) consisted of three dogs (C1-C3), who had bilaterally removed only the cortex of the suprasylvian gyrus plus white matter overlying the hippocampus, and four intact animals (C4-C7).

POSTOPERATIVE RETRAINING

Two weeks after surgery or after a 2-weeks rest for the unoperated controls, the dogs were retrained to a criterion on the delayed response task with the 10 s delay. Then, their memory was assessed further with an additional task in which the delays were increased in stages from 10 to 30, 60, and finally to 120 s. Dogs were tested for 90 trials at each of the delays (15 trials per session for 6 sessions). Trials across feeder positions were distributed randomly. Following this, all of the dogs, except for one dog in Group H, received an additional test which included distractions. This test consisted of three blocks of 6 sessions (each of 90 trials) with the 60 s delay. In the first block dogs were tested with the 60 s delay, under the same conditions as before. In the second block a short, 5 s distraction was introduced after 30 s of the delay. There were three kinds of distractions: calling, stroking of the dog by the experimenter or giving it a very small amount of food on the platform. The three distractions were given in random order from trial to trial. During the distraction the experimenter encouraged the dog to change its position, in order to be sure that the dog could not rely upon position cues to remember the location of the tone. The third block was a repetition of the first block, with no distractions.

SECOND SURGERY

At the completion of the training with extended delays and distractions after the first surgery, three dogs from Group H (H-1, H-5 and H-6) and three dogs from Group AT (AT-2, AT-3 and AT-4) were retrained in the basic task with original delay of 10 s to a same criterion as in previous stages. Then, dogs received the second surgery. Dogs from Group H were given bilateral removal of the anterior temporal lobe, whereas dogs from Group AT received bilateral hippocampal lesions. Thus, all six dogs received bilateral combined lesions to both of the hippocampus and anterior temporal lobe. They constituted Group HAT.

RETRAINING AFTER SECOND SURGERY

Dogs from Group HAT were retrained postoperatively to a criterion of 90% correct responses in 90 consecu-

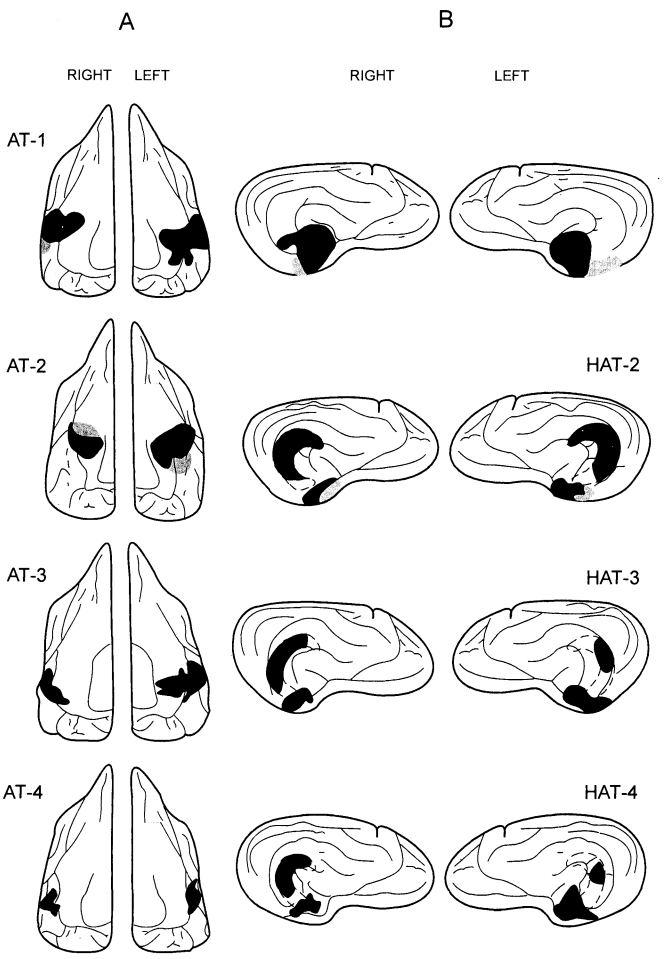

Fig. 2. Ventral (A) and lateral (B) aspects of the dogs brain for the dogs from group AT, showing the reconstructions of the first, anterior temporal lobe lesions, and the second, hippocampal lesions. Black area represents cortical injury, gray area represents injury to the subcortical brain tissue.

tive trails with a delay of 10 s and then, similarly to the training after a first surgery, they were given tasks with extended delays 30, 60 and 120 s, and distractions in blocks of 90 trials for each condition.

HISTOLOGY

At the completion of the experiment, the animals received a lethal dose of Nembutal and were perfused intracardially with saline followed by formalin. The brains were then removed, embedded in paraffin for frozen sectioning, cut into 40 μm thick section and alternately according to the Nissl's and Clüver-Barrera's techniques.

Reconstructions of the lesions of groups AT and HAT are shown of Figs. 2-4. Histological verification revealed some differences in extensions of the lesions among the dogs from the Group AT (Fig. 2). Anterior part of the perirhinal and entorhinal cortices were removed bilaterally in all dogs in exception of dog AT-4, who had spared the entorhinal cortex. Lateral nucleus of the amygdala was removed unilaterally in the right hemisphere in AT-2, whereas in AT-1, the only ventral parts of this lateral nucleus and basolateral nucleus were aspirated in the left hemisphere. In dogs AT-3, and AT-4 amygdalar nuclei were not removed. Dogs AT-1 and AT-4, had additional bilateral incisions of the lesions to the ventral parts of the posterior composite gyrus.

Reconstructions of the hippocampal lesions followed the anterior temporal lobe surgery are presented in Fig. 2, part B, (HAT-2, HAT-3, HAT-4). Coronal sections for the HAT-2 are presented in Fig. 4. Dogs had removed bilaterally the dorsal, and most of the ventral

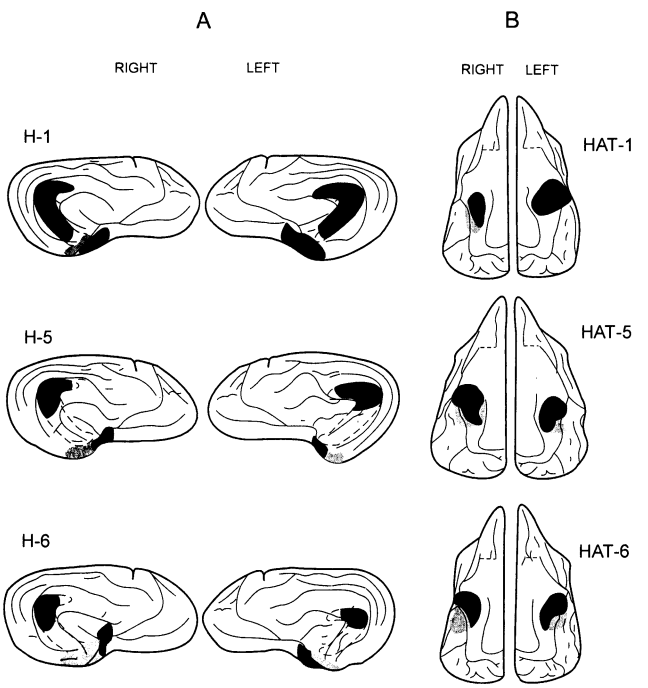

Fig. 3. Lateral (A) and ventral (B) aspect of the dogs brain for the dogs from group H, showing the reconstructions of the first, hippocampal lesions, and second anterior temporal lobe lesions. Black area represents cortical injury, gray area represents injury to the subcortical brain tissue.

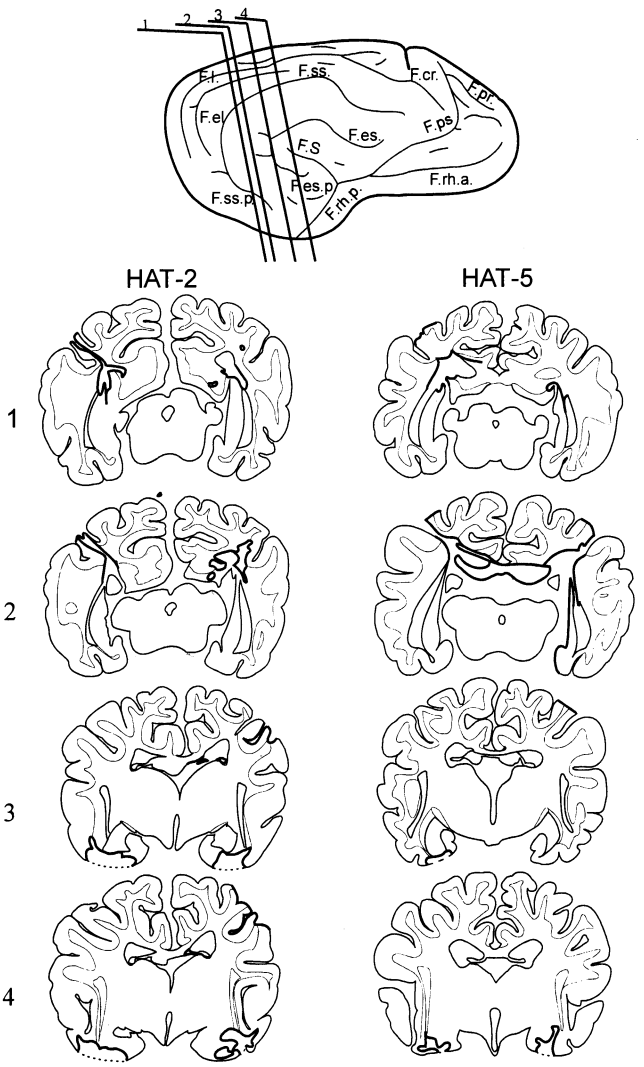

Fig. 4. Coronal sections through the anterior temporal lobe and the hippocampus in two cases, AT-2 (HAT-2) and H-5 (HAT-5). Abbreviations: F.S., fissura Sylvia; F. es., f. ectosylvia; F. es. p., f. ectosylvia posterior; F. ss., f. suprasylvia; F. ss. p., f. suprasylvia posterior; F. el., f. ectolateralis; F.l., f. lateralis; F. cor., f. coronalis; F. cr., f. cruciata; F. ps., f. presylvia; F. pr., f. prorea; F. rh.a., f. rhinalis anterior; F. rh.p., f. rhinalis posterior.

parts of the hippocampus. The CA1- CA4 fields and the dentate gyrus of the hippocampus were damaged. Subiculum was removed mostly on the dorsal part of the hippocampal formation. Most extensive bilateral lesion in HAT-2 was observed, whereas two other dogs (HAT-3 and HAT-4) had more extensive damage to the right hippocampus in comparison to lesions to the left hippocampus. Further examination revealed also substantial enlarge of the ventricles in HAT-3. In this dog remaining part of the hippocampus, was very narrow.

All of the dogs that were submitted anterior temporal lesion after the hippocampal removal (HAT-1, HAT-5, HAT-6) had bilaterally removed perirhinal and entorhinal cortices and had the injury to the amygdala complex (see Figs. 3 and 4). However in HAT-1, on the right side only the lateral part of the parvocellular and magnocellular basal nuclei were removed. In HAT-6 lesion of the amygdala was bilaterally limited to the ventral part of the lateral nucleus, and lateral part of the basal magnocellular nucleus. Additionally, in HAT-6 unilateral incisions to the right Sylvian gyrus was observed. Coronal sections for HAT-5 are presented in Fig. 4.

Reconstruction of the lesions each of the dog from Group H were presented in the previous paper (Kowalska 1995).

RESULTS

The preoperative and postoperative training scores are provided in Table I, and Fig. 5. Before surgery all of the dogs reached the criterion of 90% correct responses in the minimal amount of 90 trials, both for 0 s and 10 s delays. The error scores also did not differ significantly between groups. After surgery, one dog from the AT Group and three dogs from the hippocampal group needed additional trials in order to reach the criterion with 10 s delay and all of the dogs had elevated number of errors. These results were confirmed by two-factorial

TABLE I

Number of trials (T) and errors (E) preceding criterion on delayed responses before and after first surgery. AT, anterior temporal lesion; H, hippocampal lesion; C, control

Subjects	Preoperative training				Postoperative training	
	0 s delay		10 s delay		10 s delay	
	T	E	T	E	T	E
AT-1	90	8	90	5	90	9
AT-2	90	5	90	1	225	72
AT-3	90	0	90	1	90	2
AT-4	90	6	90	2	90	7
H-1	90	5	90	0	90	6
H-2	90	2	90	8	90	9
H-3	90	0	90	6	195	36
H-4	90	1	90	2	90	8
H-5	90	7	90	7	285	55
H-6	90	7	90	1	105	12
C-1	90	5	90	3	90	3
C-2	90	0	90	0	90	0
C-3	90	0	90	8	90	5
C-4	90	3	90	2	90	3
C-5	90	8	90	3	90	3
C-6	90	1	90	1	90	4
C-7	90	1	90	9	90	4

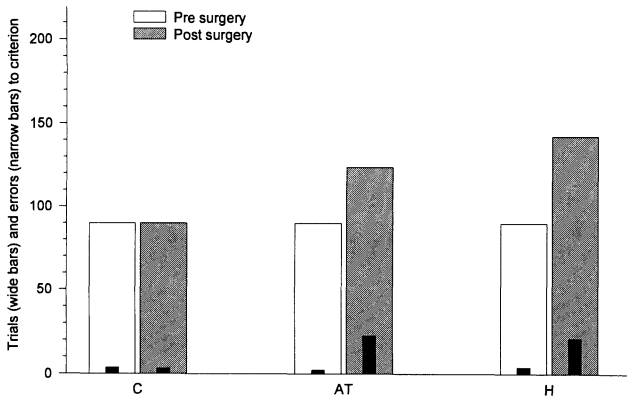

Fig. 5. Preoperative learning and postoperative relearning of spatial delayed responses. The wide bars indicate the mean number of trials for each group to learn (white bar) or relearn (shaded bars). The narrow black bars represent the mean number of errors. C, control group (n = 7); AT, group with anterior temporal lesions (n = 4); H, group with hippocampal lesions (n = 6).

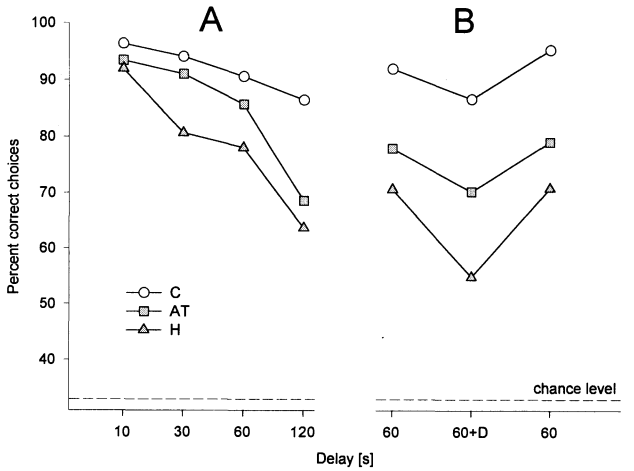

Fig. 6. Mean postoperative performance in delayed responses across delays (A) and training with distractions (B) following lesions of the anterior temporal lobe (group AT, n = 4), the hippocampus (group H, n = 6), and in controls (group C, n = 7).

TABLE II

Performance on delayed responses, and on additional training with distractions after first surgery: the scores are number correct responses in 90 trials at each delay interval. AVE indicates the average scores across the four delay conditions. AT, anterior temporal lesion; H, hippocampal lesion; C, control

Subjects	Delays					Tests		
	10 s	30 s	60 s	120 s	AVE	60 s	60 s + D	60 s
AT-1	81	82	71	40	68.5	40	35	30
AT-2	85	81	82	73	80.2	87	65	82
AT-3	88	87	83	78	84.0	80	82	84
AT-4	83	78	73	56	72.5	74	71	80
H-1	84	61	51	34	57.5	39	30	33
H-2	81	77	65	52	68.8	72	61	81
H-3	82	56	76	51	66.2	41	31	30
H-4	82	69	63	62	69.0	-	-	-
H-5	86	88	81	67	80.5	83	66	85
H-6	82	84	86	78	82.5	83	59	90
C-1	87	73	72	71	75.8	66	63	72
C-2	90	89	90	90	89.8	90	89	90
C-3	85	85	87	77	83.5	84	81	88
C-4	87	89	73	77	81.5	81	67	85
C-5	87	89	87	89	88.0	90	87	89
C-6	86	90	77	52	76.2	83	78	89
C-7	86	79	85	89	84.8	86	82	89

TABLE III

Number of trials (T) and errors (E) preceding criterion on delayed responses before and after second surgery. Before second surgery: AT, anterior temporal lesion or H, hippocampal lesion. After second surgery: HAT, combined hippocampal and anterior temporal lesion

Subjects	Before II surgery 10 s delay		After II surgery 10 s delay	
	T	E	T	E
AT-2→HAT-2	90	5	90	9
AT-3→HAT-3	90	1	90	0
AT4→HAT-4	90	1	90	2
H-1→HAT-1	330	93	90	4
H-5→HAT-5	135	17	90	7
H-6→HAT-6	180	29	90	1

ANOVAs, indicating a significant postoperative increase of trials ($F_{1,14} = 4.6$, $P=0.05$), and errors ($F_{1,14} = 6.53$, $P<0.03$].

The scores for the performance test with extended delays are shown in Table II, and in Fig. 6, part A. The level of dogs' performance declined gradually with longer delays. All groups showed progressive deterioration in performance with increasing delays. The two operated groups, however, performed more poorly than the controls. Across the four delays (10, 30, 60 and 120 s) dogs in group AT (X = 84.8%) performed more poorly than animals from group C (X = 92%) but better than dogs from group H (X = 78.6%). ANOVA confirmed both a significant group effect and a significant delay effect (for lesions, $F_{2,14} = 4.3$, $P<0.034$; for the delays, $F_{3,14} = 17.0$, $P<0.001$). There were no significant interactions. The Tukey pairwise multiple comparison test revealed that the average performance across the delay differed significantly only between the hippocampal and control dogs ($P=0.028$).

Training with distractions (Table II, and Fig. 6, part B) influenced the dogs' performance (effect of block: $F_{2,13} = 17.3$, $P<0.001$). However, neither the lesion factor nor the interaction between the groups and blocks reached significance.

Although the size of the anterior temporal lesions differed between dogs, it seemed not to correlate with their performance scores on the stages of postoperative training. Also no specific changes in dogs behavior after surgeries were observed.

After training with distractions, six dogs (3 from each of group: AT and H) were retrained with original delay of 10 s. As it is shown in Table III, the dogs from Group AT reached the criterion immediately while every dog from Group H needed significantly more trials, and made more errors, in order to reach the criterion (One-way Mann-Whitney test, $U = 0$, $P=0.037$, for trials, and $P=0.046$, for errors). All of the dogs after the second surgery, had combined lesion to the hippocampus and to the anterior temporal lobe (Group HAT). During postoperative training the dog HAT-2 died on the epileptic

TABLE IV

Performance on delayed responses, and on additional training with distractions for the dogs after combined hippocampal and anterior temporal lesions (HAT): the scores are numbers of correct responses in 90 trials at each delay interval. AVE indicates the average scores across the four delay conditions

Subjects	Delays					Tests		
	10 s	30 s	60 s	120 s	AVE	60 s	60 s + D	60 s
HAT-2	81	82	86	-	-	-	-	-
HAT3	90	88	86	88	88.0	89	86	89
HAT4	88	88	83	79	84.5	79	55	78
HAT-1	86	84	87	52	77.2	42	42	43
HAT-5	83	65	76	77	75.2	85	85	87
HAT-6	89	88	81	89	86.8	89	71	65

attack, on the stage of training with delay of 120 s, thus his data are incomplete. Surprisingly, the dogs with combined lesion to the hippocampus and anterior temporal lobe, independent of size of lesions, were not impaired both in retraining of the delayed responses with 10 s delay (see Table III), and in performance the task with extended delays and distractions (Table IV). The average scores for groups of the animals who were submitted the first separated, and then the second, combined lesions to the anterior temporal lobe and the hippocampus are presented in Fig. 7. After the second surgery, both animals from former group AT and H showed higher scores of performance with extended delays. This result is confirmed by individual data presented in Table IV, showing that all of the dogs, with exception of HAT-5, had an elevated mean number of correct responses with the longer delays in comparison of their performance after the first surgery (see Table II). Similarly, dogs submitted combined lesions, with exception of HAT-4, had shown less distractibility after the second than after the first surgery.

Across the experiment the mean latency of responses did not differ between groups. No correlation was observed between the speed of responding and the performance level on the task.

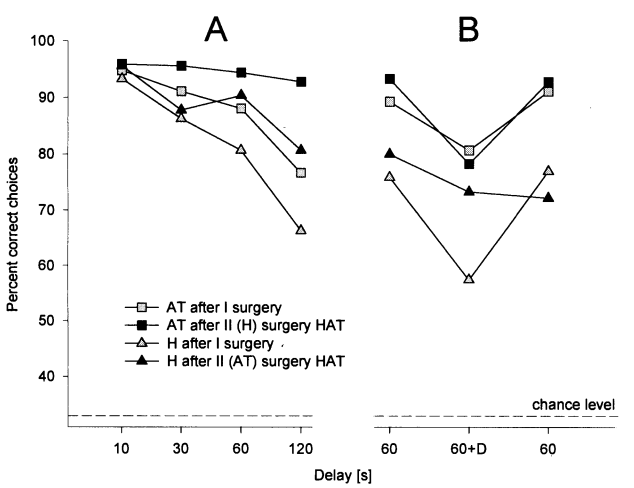

Fig. 7. Mean postoperative performance in delayed responses across delays (A) and training with distractions (B) following lesions of the anterior temporal lobe (group AT, $n = 3$), the hippocampus (group H, $n = 3$), after first surgery (shaded squares and triangles), and for the same groups after the second surgery (black squares and triangles) forming the group HAT.

DISCUSSION

The present data indicate that lesions to an anterior temporal lobe, region that includes the anterior portion of the rhinal cortex and part of the amygdala, produces no significant effect on retention of an auditory based delayed response task. In addition, the anterior lesions did not affect performance at long delays, or sensitivity to distraction.

This may suggest that within the temporal lobe structures only the hippocampus is important for memory of spatial location of auditory cue. In primates the amygdala does not seem to be involved specifically in spatial learning and memory. However, there are some indications on monkeys suggesting involvement of the rhinal cortical areas in spatial tasks (Suzuki et al. 1997, Murray et al. 1998). In the present study, only the anterior part of the rhinal cortex was consistently damaged. We wont be able to determine whether more extensive damage to the rhinal cortex is disruptive of this auditory-based spatial task until we have improved surgical techniques for removing the medio-caudal part of the dogs temporal lobe. Another issue is that the studies on monkeys involved a spatial task with visual cues, whereas this study looked only at the memory of the location of an auditory cue. So, it might be possible, that memory for location of auditory cues is processed in a different brain circuit than memory of visual cues. Alternatively, there might be notable species differences between mid-temporal structures in processing spatial tasks. Other possibility is that only some spatial tasks are sensitive to the lesions of this region.

Unexpectedly, after the second surgery, which resulted in combined lesions to the anterior temporal lobe and the hippocampus, performance on the spatial delayed response was generally higher than after separate lesion to these structures. This was true both for the animals who were lesioned first in the anterior temporal region and for those who received hippocampal first and anterior temporal lesions second. Since the hippocampal dogs needed extended retraining to the criterion after performance task with longer delays and distractions, they had longer time frame between the both surgeries, than animals with the anterior temporal lesions. Even though both groups of dogs with combined lesions were not impaired.

The results of the hippocampal lesions were surprising. It had been previously shown that hippocampal lesions affect acquisition of a spatial delayed response,

performance over extended delays (with and without distractions) and retraining (Kowalska 1995). The present experiment did no find any effect on performance over subsequent retraining after the addition of an anterior temporal lesion.

One explanation of this phenomenon is that, combined lesion to the two parts of the temporal lobe could abolish the effect of separate hippocampal injury. However, to prove this, it is necessary to look at the effects of simultaneously produced combined lesions on the early and also later stages of experiment. Another possibility is that the extensive training led to brain structures outside of the hippocampus assuming control over spatial delayed response performance. This hypothesis is consistent with the suggestion of Squire and Zola-Morgan that the hippocampus is important only for the initial phase of information storage since it serves maintenance of the information only in the limits of time (Zola-Morgan and Squire 1990, Squire 1992).

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