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COGNITIVE FUNCTIONS OF THE TEMPORAL LOBE IN THE DOG: A REVIEW

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Contents

Abstract

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1 The current paper reviews the role of temporal lobe structures in learning and different kinds of memory, with an emphasis on behavioral tasks that re auditory stimuli.
2 The effects of lesions to structures in the temporal lobe were examined in separate groups of dogs, which were trained on an auditory spatial delayed response, or in a trial-unique auditory delayed match to sample recognition task.
3 Spatial memory was impaired after bilateral hippocampal lesions. On the other hand, neither an anterior temporal lesion or rhinal cortical injury nor combined lesion to the hippocampus and the anterior temporal lobe, affected postoperative retraining and performance of the spatial task.
4 Auditory recognition memory task was not impaired after a hippocampal and/or rhinal cortex lesion. However, postoperative retraining of the task was impaired after a lesion to auditory association areas.
5 These results confirm the role of the hippocampus in spatial memory in the dog. On the other hand, the organization of auditory recognition functions within the temporal lobe appears to be different from those described for visual recognition functions.

Keywords: auditory cortex, auditory memory, hippocampus, rhinal cortex.

Abbreviations: Delayed Matching-to-Sample (DMS); Delayed Non Matching-to-Sample (DNMS)

1. Introduction

The study of the neural substrates of memory has focused on temporal lobe structures for the past several decades. This interest was prompted over forty years ago by clinical reports of global anterograde amnesia in patients with bilateral damage to the medial temporal lobe (Scoville, 1954; Scoville and Milner, 1957; Penfield and Milner, 1958). One of the most famous patients, H.M., who had extensive bilateral removals of the medial temporal lobe, following experimental procedures to relieve his intractable epilepsy, he became unable to store and retrieve new information about stimuli and events. However, subsequent research demonstrated H.M.'s ability to store and use certain kinds of information. He can learn new motor and cognitive procedures that are skill-based and also exhibit intact perceptual priming, although he has little or no awareness of the learning event or of the context in which it occurred.

These results gave rise to a new theory about the anatomical substrates underlying memory and encouraged several investigators to attempt to reproduce the syndrome in animals. The development of a test for one-trial recognition memory for monkeys (Gaffan, 1974; Mishkin and Delacour, 1975), was followed by reports that bilateral medial temporal lobe damage in animals (involving the hippocampus, the amygdala cortical areas underlying these structures i.e. the perirhinal, the entorhinal and the parahippocampal cortices) produced memory impairments (Mishkin, 1978). Further, similar to the clinical syndrome, it appeared to be global in nature (Mishkin et al., 1982) and extended to different sensory modalities (for review, see Mishkin and Murray, 1994).

2. Neuroanatomy of the Temporal Lobe System

In the monkey, neuroanatomical studies provided evidence that temporal lobe structures were highly interconnected. Sensory information from unimodal (modality-specific) and polymodal cortical areas of the temporal, frontal and parietal lobes reaches the perirhinal and parahippocampal cortical areas located on the medial temporal lobe (Jones and Powell 1970; Van Hoesen and Pandya, 1975a; 1975b; Van Hoesen et al., 1975; Suzuki and Amaral, 1994; Suzuki, 1996), which in turn project to the entorhinal cortex followed by the hippocampus (Squire and Zola-Morgan, 1991). The entorhinal cortex and the hippocampus together constitute the "hippocampal formation". Suzuki and Amaral (1990) also described direct connections of the perirhinal and parahippocampal cortices to area CA1 of the hippocampus. The amygdala also receives a strong projection from the entorhinal as well as perirhinal cortex (Saunders and Rosene, 1988). A comparison between the anatomy of temporal lobe structures in the monkey and dog brain is illustrated in Fig 1. However, it should be

emphasized that our knowledge of the projections of neocortical areas to perirhinal, entorhinal, and parahippocampal areas in dogs is relatively unknown.

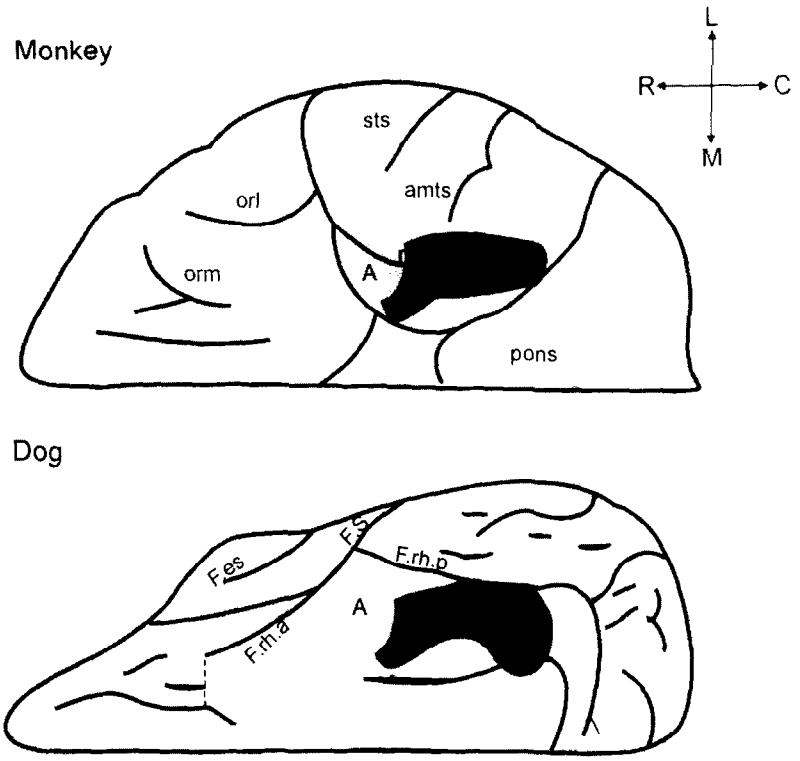

Fig 1. Schematic diagram of the ventral views of the left hemisphere of the monkey's and dog's brain. Perirhinal cortex is located along rhinal sulcus in the monkey, and posterior rhinal fissure in the dog. Entorhinal cortex is located medially to the perirhinal cortex, and parahippocampal cortex is situated caudally to the entorhinal cortex. Abbreviations: A, amygdaloid complex: H, hippocampus; amts, anterior middle temporal sulcus; sts, superior temporal sulcus; rs; rhinal sulcus; orl, lateral orbital sulcus; orm, medial orbital sulcus; F.es., ectosylvian fissure; F.S., Sylvian fissure; F.rh.a., anterior rhinal fissure; F.rh.p., posterior rhinal fissure. M, medial; L, lateral; R, rostral; and C, caudal location.

3. Emotionality and Stimulus-Reward Associations

There is considerable evidence showing changes in emotionality of animals with amygdalar lesions. In monkeys, bilateral amygdalectomy decreases their reactivity to several different sensory modalities and modified food preferences (Weiskrantz, 1956; Aggleton and Passingham, 1981; Aggleton and Mishkin, 1986; Murray et al., 1996). Significant changes in social and maternal behavior were also observed (Weiskrantz, 1956; Rosvold et al., 1954).

Studies on dogs performed by Fonberg et al. (1969; 1972) at the Nencki Institute in Warsaw, Poland, revealed that lesions to the dorsomedial part of the amygdala led to hypoactivity and alterations in feeding behavior that ranged from hypophagia to aphagia. This lesion also reduced the responsiveness of dogs to external cues and produced an unwillingness to interact socially with the experimenter (Fonberg, 1969; 1972). Moreover, damage to the dorsomedial amygdala produced significant deterioration in the instrumental differentiation of two tones, reinforced both by food and by social-sensory rewards (Fonberg and Korczyński, 1993). On the other hand, cortico-basomedial amygdaloid lesions in dogs increased the number of intertrial responses in instrumental learning reinforced by petting (Kostarczyk and Fonberg, 1988). These findings are consistent with data from monkeys indicating a role of the amygdala in stimulus-reward associations and in memory for rewards, possibly through its interaction with the thalamus and the frontal lobe (Gaffan and Murray, 1990; Gaffan et al., 1993). More recent data indicates the critical role of the amygdala in associating environmental stimuli with the value of particular reinforcers (Malkova et al., 1997; Thornton et al., 1998) and are in agreement with earlier Fonberg's studies on dogs.

4. Spatial Tasks

There is an extensive literature from monkeys and rats describing impairments in spatial tasks after lesions to the hippocampus (for review see Barnes, 1988; Squire, 1992; Nadel, 1995). The hippocampus is hypothesized to play a role in constructing and storing cognitive maps, which are representations that capture the spatial layout of an animal's experienced environment (O'Keefe and Nadel, 1979). Hippocampal ablation in monkeys impairs their memory for place (Angeli et al., 1993; Parkinson et al., 1988). Recent data from monkeys indicate that selective hippocampal lesions can also impair learning of nonspatial tasks with similar objects presented in different contexts (Dore et al., 1998). On the other hand, there is evidence that spatial scene learning is impaired after neurotoxic hippocampal lesions that are equivalent to those observed after rhinal cortex lesions in monkeys (Murray et al., 1998). Further, studies in rats that also indicate that lesions to the perirhinal cortex can disrupt performance of spatial tasks including the water maze (Liu and Bilkey, 1998a) and radial arm maze (Liu and Bilkey, 1998b) as well as in a delayed non-matching-to-position task (Wiig and Burwell, 1998). These findings are consistent with the underlying neuroanatomy of the temporal lobe since entorhinal neurons in the monkey receive sensory information about both object and spatial locations and electrophysiological studies suggest that they encode information about objects and locations held in short-term memory (Suzuki et al., 1997). These studies suggest that within the temporal lobe, spatial functions might not be limited only to the hippocampus.

Thus, to further our understanding of the role of temporal lobe structures in spatial information processing, we trained dogs on a spatial delayed response task using acoustic stimuli in the three-choice Nencki Testing Apparatus (Fig 2).

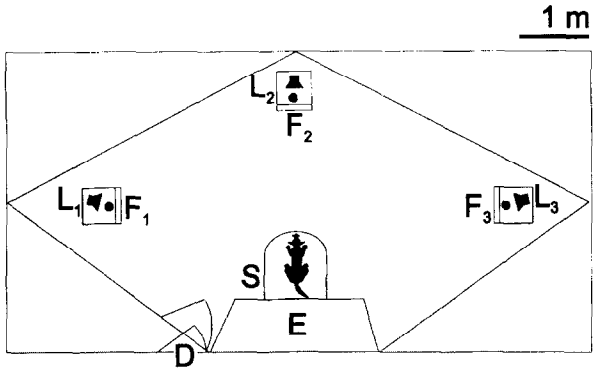

Fig 2. Schematic illustration of the Nencki Testing Apparatus. F1, F2, F3, feeders; L1, L2, L3, loudspeakers; S, starting platform, where subject is waiting; E, location of experimenter; D, entrance door.

This task used a 10 s delay interval between the presentation of an auditory stimulus and the time when dogs are allowed to make a choice and animals were trained to a criterion of 90% correct in 90 consecutive trials (6 sessions). Subsequently, 6 dogs received bilateral hippocampal lesions according to the method described previously designed using a method of aspirating the hippocampus without injuring the rhinal (i.e. entorhinal and perirhinal) cortices (Kowalska and Kosmal, 1992). Hippocampal removals in the dog are performed through a small opening in the suprasylvian gyrus, a visual association area of the parietal lobe (Fig 3). This technique cannot be used with monkeys, since the hippocampus must be approached from below, which frequently leads to damage of the underlying rhinal cortices. The results of 6 hippocampectomized dogs (group H), were compared with a group of 7 control dogs (group C) (Kowalska, 1995), and to a group of 4 dogs receiving anterior temporal removals (group AT). AT lesions involved the removal of the anterior part of the rhinal cortex and the amygdala (Kowalska, 1999). At the time of this study, we had not developed a procedure for removing the posterior portion of the rhinal cortex but recently we have developed a technique that allows the removal of the entire rhinal cortex including both the anterior and posterior regions (Kowalska et al., 1999c). Preliminary results obtained from 4 dogs with these rhinal cortex lesions (group Rh), are compared here with the other experimental groups. The location of the intended anterior temporal and rhinal cortex lesions is illustrated in Fig 4. The mean number of trials and errors for the

four experimental groups are shown in Fig 5.

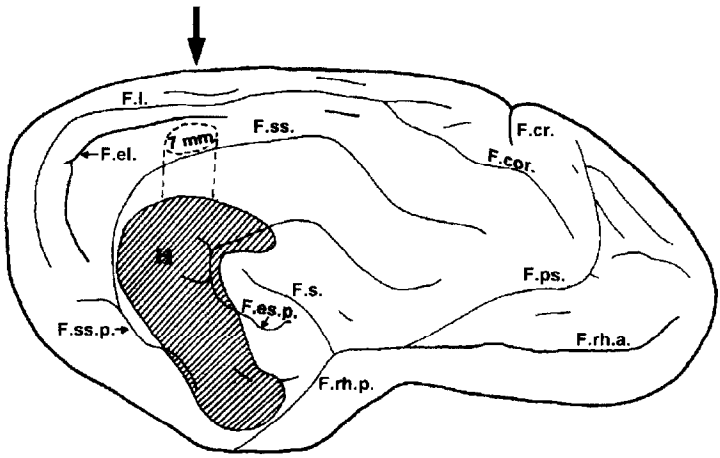

Fig 3. Lateral aspects of the dog's brain with a view of the shape of the hippocampus (shaded area). Black arrow indicates the place of entrance toward the hippocampus. Abbreviations: F.s., Sylvian fissure; F.es., ectosylvian fissure; F.es.p., ectosylvian posterior fissure; F.ss., suprasylvian fissure; F.ss.p., suprasylvian posterior fissure; F.el., ectolateral fissure; F.l., lateral fissure; F.cor., coronal fissure; F.cr., cruciata fissure; F.ps., presylvian fissure; F.pr., proreal fissure; F.rh.a., rhinal anterior fissure; F.rh.p., rhinal posterior fissure.

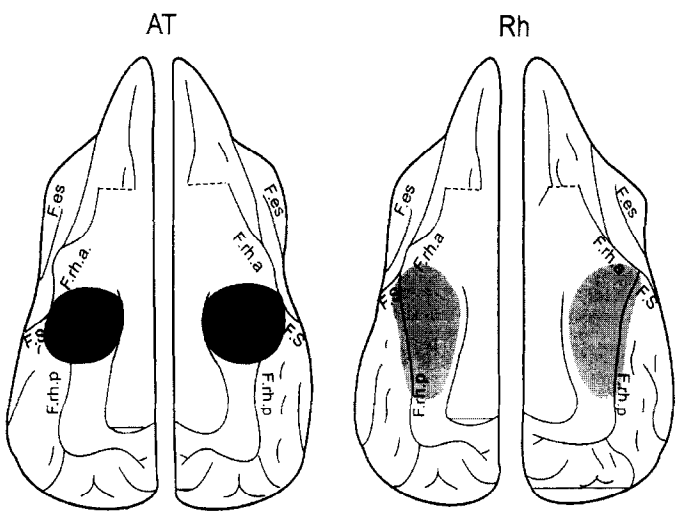

Fig 4. Ventral view of the dog's brain showing the intended anterior temporal (AT, black area) and rhinal cortex lesions (Rh, gray area). Abbreviations the same as on Fig 1.

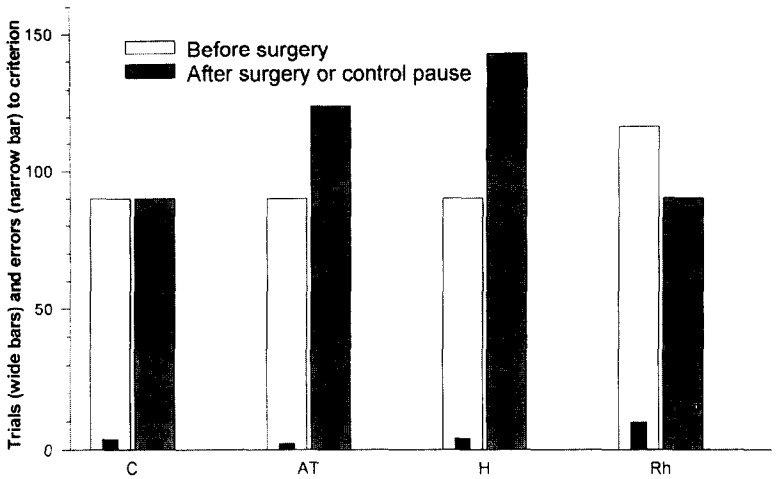

Fig 5. Preoperative learning and postoperative relearning of spatial delayed responses. The wide bars indicate the mean number of trials for each group to learn (white bar) or relearn (shaded bars). The narrow black bars represent the mean number of errors. C: control group (n=7), AT: group with anterior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

After the surgery, both groups C and Rh reached criterion on the spatial delayed response task immediately, but the other two groups had higher mean number of trials and errors to criterion. However, one dog in the AT group and 3 dogs in the H group accounted for much of this. Thus, only dogs with hippocampal lesions showed a significant increase in the number of errors to postoperative criterion. Following this, all dogs were given additional training on the spatial task but with the delay intervals extended to 30, 60 and 120 s, and subsequently with distractions (each in blocks of 90 trials). Although dogs with anterior temporal lesions exhibited some decrement in performance across delay intervals, only the hippocampal dogs showed significantly lower scores with increasing delays and with distractions (Fig 6, part A and B, respectively). These results indicate that the hippocampus is crucial for intact performance of a spatial delayed response task using acoustic stimuli in dogs.

Our next question was to determine if a combined lesion to the hippocampus and the anterior temporal lobe would be additive relative to the effects observed after either lesion alone. For this reason, at the completion of the training with extended delays and distractions, three dogs from the H group and three dogs from the AT group were retrained on the basic task with the initial training delay of 10 s and to the same 90% criterion level. Dogs from the AT group reached the criterion

immediately, while every dog from the H group, as observed previously, required significantly more trials and made more errors to reach criterion. Following this, dogs received a second surgery where

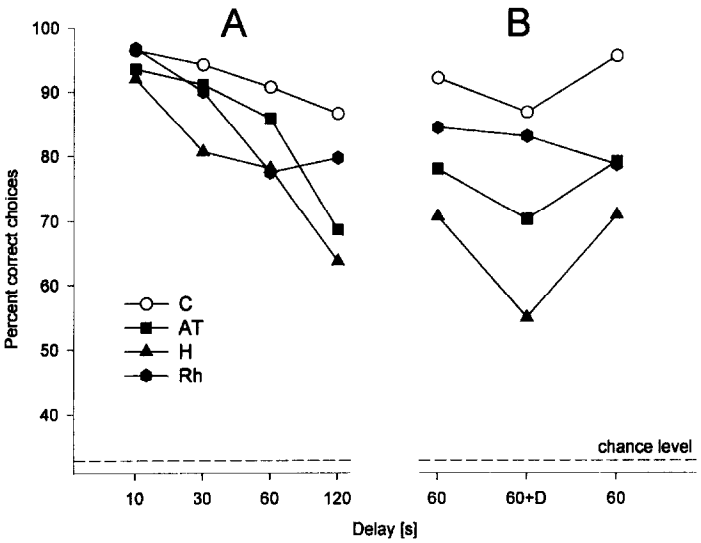

Fig 6. Mean postoperative performance in delayed responses across delays (A) and training with distractions (B) following lesions of the anterior temporal lobe (group AT, n=4), the rhinal cortex (group Rh, n=4), the hippocampus (group H, n=6), and in controls (group C, n=7).

dogs from the H group received a bilateral removal of the anterior temporal lobe, and dogs from the AT group received bilateral hippocampal lesions. Thus, all six dogs received bilateral combined lesions to both the hippocampus and anterior temporal lobe and were called group HAT. Surprisingly, the dogs with combined lesion to the hippocampus and anterior temporal lobe, independently of the size of the lesions, were not impaired both in retraining of the delayed responses with 10 s delay (Fig 7), and in performance of the task with extended delays and distractions. The average scores for groups of animals who first received separate lesions and then the second, combined lesions to the anterior temporal lobe and the hippocampus are presented in Fig 8. After the second surgery, both groups that were formerly single-lesion groups AT and H, exhibited higher scores on the spatial task and during performance with extended delays (Fig 8, part A). In addition, dogs with combined lesions exhibited less distractibility after the second surgery when compared to the first surgery (Fig 8, part B).

The effects of hippocampal lesions on the spatial task appear to be a function of test experience. Thus, hippocampal lesions, which significantly affected retraining and performance of the originally acquired spatial delayed response task, did not have the same effect after extensive training even with an additional lesion to the AT. One explanation for these results is that, combined lesions to these two areas of the temporal lobe could abolish the effect of a separate hippocampal injury.

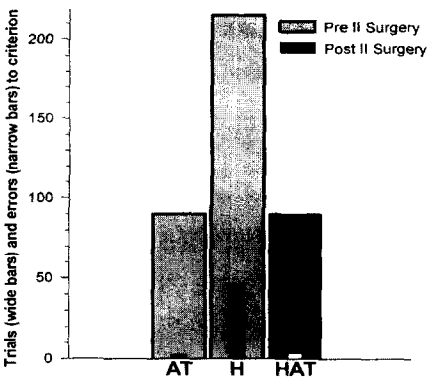

Fig 7. Mean number of trials to criterion before (wide shaded bars) for the group AT (n=3) and H (n=3); and after the second surgery (wide black bar) for the group HAT (n=6). Narrow bars represent the mean number of errors to criterion.

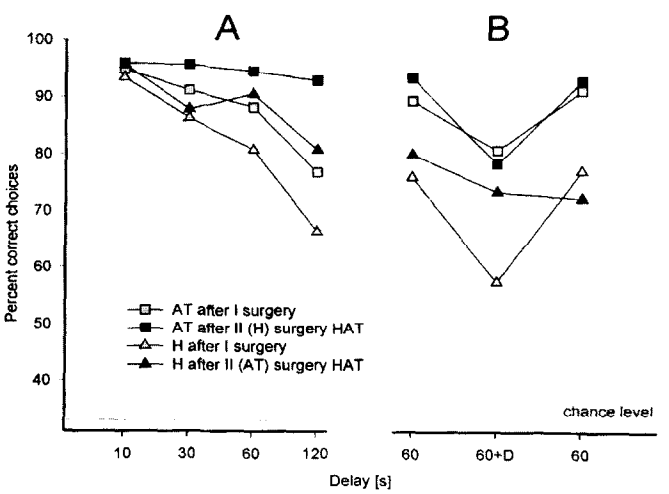

Fig 8. Mean postoperative performance in delayed responses across delays (A) and training with distractions (B) following lesions of the anterior temporal lobe (group AT, n=3), the hippocampus (group H, n=3), after first surgery (shaded squares and triangles, respectively), and for the same groups after the second surgery (black squares and triangles, respectively) forming the group HAT.

Another explanation is that in the over-trained task, the role of the hippocampus in performance of the spatial delayed responses was reduced, or perhaps eliminated, with other brain structures controlling spatial behavior. If so, this assumption could favor a view, that the hippocampus plays time-limited role in memory storage (Zola-Morgan and Squire, 1990; Squire, 1992).

5. Recognition Memory

One-trial recognition memory is only mildly disturbed or unimpaired in amnesic patients and hippocampectomized monkeys (Zola –Morgan et al., 1992, 1994; Murray and Mishkin, 1996). On the other hand, recent studies in monkeys provide evidence that the rhinal cortex (including perirhinal and entorhinal cortical areas) are critical for object identification and memory (Zola-Morgan et al., 1989; Meunier et al., 1993; Suzuki et al. 1993; Murray, 1996; Mishkin et al., 1997). This new evidence clarified previous findings that recognition memory deficits in monkeys with combined lesions to the hippocampus and amygdala (Mishkin, 1978), was probably related to damage to the rhinal cortical areas underlying these structures. However, recognition memory deficits in animals with medial temporal lesions have only been reported for the visual, tactile, and olfactory modalities; the role of the mammalian medial temporal lobe in auditory recognition has yet to be explored. Understanding the role of the temporal lobe in auditory recognition as well as the role of other structures is important for identifying the neural substrates of vocal communication in mammals and ultimately, of speech and language in humans.

5.1. Auditory Recognition in Animals

It is surprising that although processing auditory information is important for vocal signals in nonhuman primate communication (Snowdon et al. 1982), monkeys have difficulty learning auditory tasks. Although there are some reports showing that monkeys can learn simple auditory differentiation tasks (Iversen and Mishkin, 1973; Ławicka et al., 1975), most indicate a great difficulty with training monkeys on both simple differentiation and more complicated auditory memory tasks (Wegener, 1964; Stępień and Cordeau, 1960; Stępień et al., 1960; Dewson and Cowey, 1969; Dewson and Burlingame, 1975; Cowey and Weiskrantz, 1976; Kojima, 1985; D'Amato and Colombo, 1985; Colombo and D'Amato, 1986; Wright, et al., 1990; Fritz at al. 1997). Dogs, by contrast, appear to learn auditory memory tasks relatively easily (Chorążyna and Stępień, 1961; Brown and Sołtysik, 1971; Pietrzykowska and Sołtysik, 1975a; 1975b). In fact, a substantial body of evidence is growing (largely by scientists at the Nencki Institute in Warsaw, Poland) describing auditory differentiation, generalization, and reversal learning in dogs (Dąbrowska, 1971;

1975; Kowalska, 1980; Kowalska et al., 1975a; 1975b; 1981; Kowalska and Zieliński, 1976; 1980; Brennan et al., 1976; Zieliński et al., 1979). The cross-species comparison suggested not only that dogs are particularly receptive to auditory stimuli, but also that they may be better subjects than monkeys for research on auditory memory.

The first step was to develop a one-trial auditory recognition task that was comparable to the visual recognition tasks and used either a delayed matching- or nonmatching-to-sample (DMS and DNMS) procedure with trial-unique stimuli as was used with monkeys. An auditory recognition task using a DMS procedure has been developed for dogs (Kowalska 1997), and will be described briefly. The experimental setting (Fig 9) consisted of one central speaker located in front of the dog's head, two side speakers with nearby response pedals and one rotary food delivery system. Three hundred and twenty natural sounds, highly distinguishable to the human ear, were used as trial-unique stimuli. Sample stimuli were always given through the central speaker. After a delay of 1.5 s, both sample and testing stimuli were activated alternately through the two side speakers. Dogs were required to indicate having heard the sample sound by a bar-press response toward the side that the sample stimulus was presented and they were rewarded by food. The side where sounds were presented, as well as which sound (familiar or new one) was presented as a first after delay, were all given in pseudorandom order. The procedure used for presenting auditory stimuli is shown on Fig 10.

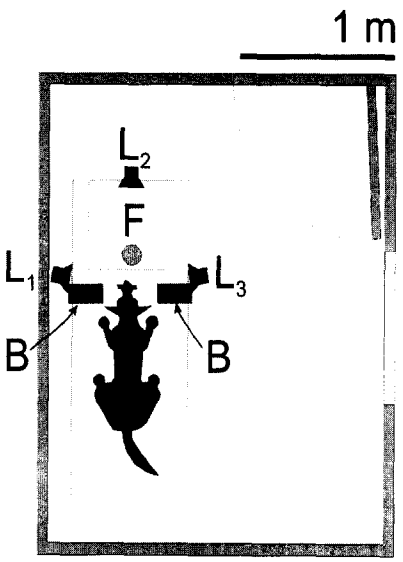

Fig 9. The experimental procedure used to test auditory recognition. F, feeder; B, response pedals, L1, L2, L3, loudspeakers.

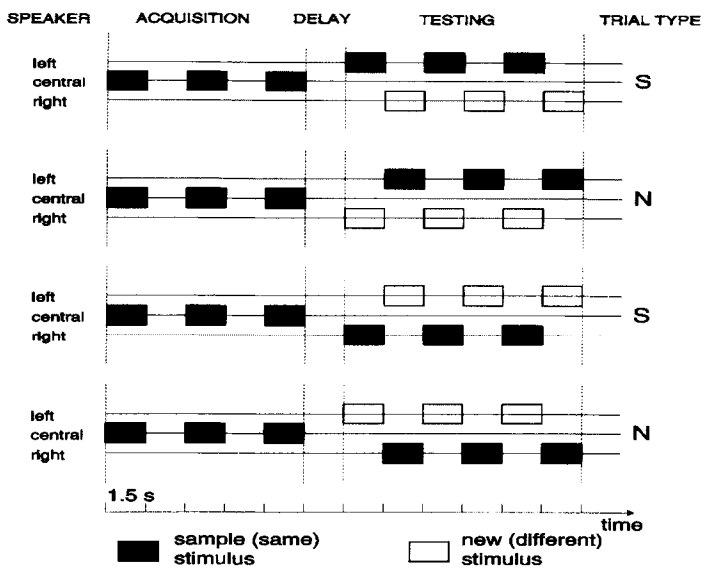

Fig 10. The technique used to present acoustic stimuli in the auditory DMS task with alternating trial-unique sounds. S, testing trial started with sample sound; N, testing trial started with the novel sound.

In collaboration with Mishkin (NIMH, Bethesda, USA) we have now made comparative studies using this task on both rhesus monkeys and mongrel dogs. To date, twelve dogs but only three monkeys have been able to learn the auditory recognition task (Kowalska et al., 1999b). Monkeys were trained in a modified, sound-insulated WGTA with movable speakers or touch-pads serving as manipulanda. Dogs were trained in a soundproof testing chamber (Fig 9) or in the Nencki Testing Apparatus (Fig 2), adapted for auditory recognition testing. In this dog test procedure, no food was given in the central feeder, which was covered during the experiment. In addition, rather than pressing a bar, the dogs were trained to approach one of the side feeders above which was located a speaker that emitted the familiar (sample) sound. Further analysis revealed that differences in the test apparatus (soundproof testing chamber vs Nencki Test Apparatus) resulted in no difference in basic learning and performance of the auditory recognition task in dogs (Kuśmierek and Kowalska, 1998).

The test procedure consisted of training directional instrumental responses to sound, followed by recognition training with short delays (1.5 or 3 s) to a criterion of 85% (monkeys) or 90% (dogs) of correct responses in 100 consecutive trials, reinforced by food. After a two-week rest, the subjects were retrained on the DMS task to the same criterion. Then they were given a performance task with extended delays. For dogs delays were extended, in 100-trial blocks, to 10, 30, 60 and finally 90 s,

whereas in monkeys delays were lengthened in smaller steps of 5 s each, starting from 5 s, and extending to delay of 60 s.

According to data obtained in our studies, monkeys acquired the instrumental responses slower than dogs. In recognition training, the average number of trials for monkeys was higher (1487, range: 960-1760) using a criterion of 85% correct choices, than for dogs (649, range: 180-1355) with a criterion of 90% correct. After another rest period, most of the animals reached criterion immediately. The range for monkeys was: 0-480, and for dogs: 0-120 (Kowalska et al., 1999b). The performance of the dogs declined gradually with extended delays: 10s – 83% (range: 73-91), 30s– 74% (62-83), 60s – 68% (59-81), 90s – 65% (53-76), whereas the behavior of monkeys was more dramatic. One of the monkeys refused to respond when the delay was extended to 10 s, the second monkey dropped to a 60% level of performance with a delay of 30 s and the third monkey was able successfully to reach a level of 67% with a delay of 60s and is currently being tested. The results of this experiment indicate that dogs are much better than monkeys in learning and performance of an auditory DMS task and are proving to be very useful subjects for studying auditory memory.

5.2. Temporal Lobe Structures and Auditory Recognition

With a newly developed auditory memory test for dogs, the next important step towards the goal of determining whether auditory recognition, like recognition in other modalities, depends on the medial temporal lobe was the development of appropriate surgical techniques for dogs (Kowalska and Kosmal, 1992). Results from dogs indicated that bilateral hippocampal removals have no effect on auditory recognition as measured either by postoperative relearning of the auditory DMS rule or by performance with increasing delays (Kowalska and Kuśmierek, 1996). This negative finding was not surprising, in view of similar negative findings that had been obtained from visual recognition tasks in monkeys with selective hippocampal lesions made with an excitotoxin (O'Boyle et al., 1993; Murray and Mishkin, 1996; Murray and Mishkin, 1998). Subsequently, however, we obtained a result that was totally unexpected (Kowalska and Kuśmierek, 1997). The multimodal recognition deficits that had been discovered initially in animals with extensive medial temporal lesions (i.e. deficits in vision, touch, and olfaction) were all found later to be attributable to damage of the rhinal, and particularly the perirhinal, cortex. There was every reason therefore to predict that the same result would be obtained for auditory recognition. In fact, however, the effects of rhinal cortical lesions, like those of hippocampal lesions, turned out to be entirely negative. This was true both for the dogs after separate bilateral rhinal cortex lesion (Fig 11, part B), and the dogs, which received rhinal lesions, combined with bilateral damage to the hippocampus (Fig 11, part A).

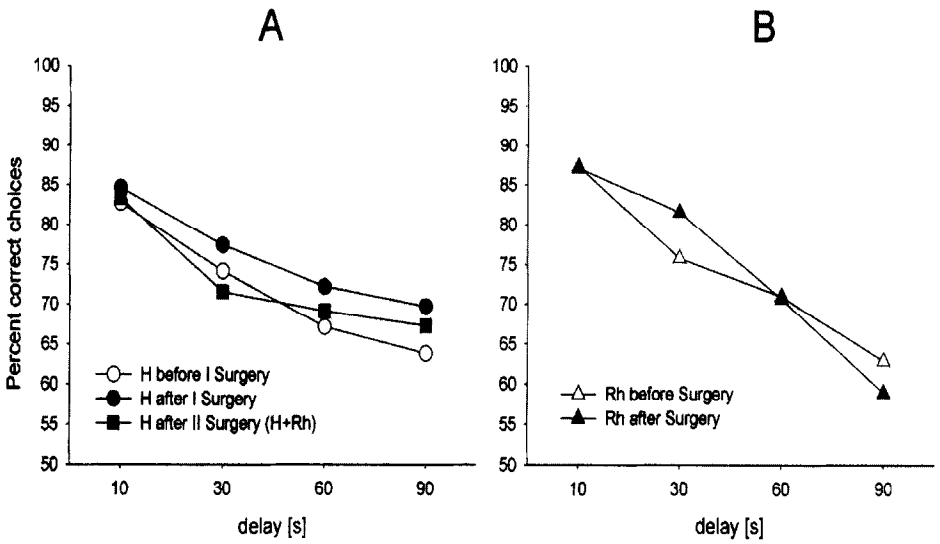

Fig 11. Mean postoperative performance in the auditory DMS task across delays. A: following lesions of the hippocampus (group H, n=4), and combined second lesions to the rhinal cortex (group H+Rh, n=4). B: following the rhinal cortex lesions (group Rh, n=3).

One possible explanation for the negative finding is that the mnemonic role of the rhinal cortex in dogs differs from that in monkeys. To examine this possibility, we began a collaborative investigation with Milgram, at the University of Toronto, who had successfully trained dogs on a visual DNMS task (Milgram et al., 1994). We have performed rhinal cortex lesions on two dogs trained on the visual recognition memory task and preliminary data indicate that the animals are impaired. One dog has a postoperative relearning deficit, while the other, though unimpaired in reacquiring the task, shows a significant postoperative decrement in performance at extended delays. These data, though still preliminary, suggest that at least for visual recognition, the effects of rhinal cortical lesions in the dog are similar to the effects of such lesions in the monkey. Indeed, it now appears as though the same may be true for auditory recognition. As indicated above, monkeys learn auditory tasks only with great difficulty and far more slowly than dogs. Nevertheless, in Mishkin's laboratory several monkeys were trained on an auditory recognition task, after literally thousands of trials in each case (Fritz et al., 1997) and preliminary data indicate that, just as in dogs, rhinal cortex lesions are without effect (Saunders et al., 1998). These results reinforce the surprising conclusion from our studies on dogs, that auditory memory in animals is organized differently from memory in other modalities. The two studies together point out the need for a better understanding of the

organization of the cortical auditory system in animals and even more importantly, how it interacts, if at all, with (a) the medial temporal-lobe structures that had been thought to support cognitive memory in all sensory modalities and (b) frontal cortical areas that may instead be the critical structures for auditory memory.

However, the anatomical and physiological organization of the cortical auditory system is still largely a mystery both in humans and in animals. The little that is known, however, allows the supposition that the cortical processing streams for audition are arranged much like those for vision and touch. In monkeys, the auditory system is composed of a number of different fields that appear to be arranged, at least grossly, in the form of parallel multisynaptic pathways (Pandya and Yeterian, 1985). Starting with the primary auditory fields in the supratemporal plane, one such pathway is directed ventrally towards the temporal pole, the medial temporal portion of the limbic system, and the ventral prefrontal region, while another is directed dorsally toward the parietal and dorsal prefrontal regions. By analogy with the visual and tactile systems (Ungerleider and Mishkin, 1982; Friedman et al., 1986; Schneider et al., 1993), it is likely that the ventrally directed auditory stream mediates perception of the quality of auditory stimuli, such as vocal signals and other complex sounds. Recent evidence in support of this assumption was provided by Rauschecker from Georgetown University, Washington DC (Rauschecker, et al., 1995; 1997). This is in contrast to the spatial location of the source of auditory information, which is likely to be mediated instead by the dorsally directed pathway. As noted, however, the analogy to the other systems breaks down when we consider the mechanism by which newly presented auditory stimuli are recognized as familiar, and, in particular, the structures with which the putative ventral auditory stream must interact with in order that auditory stimuli may be stored and retrieved. A solution to this problem requires, in the first instance, a careful examination of auditory cortical connectivity patterns.

Such an examination on dogs was undertaken by Kosmal and her colleagues at the Nencki Institute, Warsaw, Poland. Their earlier studies have shown that cortical differentiation in the dog is more advanced than that in cats, particularly in the temporal and frontal lobes (Kosmal, 1981; 1986; Kosmal and Dąbrowska, 1980; Kosmal et al., 1984; Markow-Rajkowska and Kosmal, 1987; Stępniewska and Kosmal, 1986). More recently, Kosmal began a research program designed to directly compare the auditory-system anatomy of dogs with that in monkeys. Starting with an analysis of thalamocortical projections, Malinowska and Kosmal (1994) have shown that in dogs, the dominant projection is from the tonotopically organized ventral division of the medial geniculate

body (MGB) and is directed to the middle ectosylvian area (EM, see Fig. 12), which constitutes the dog's primary auditory cortex (A1).

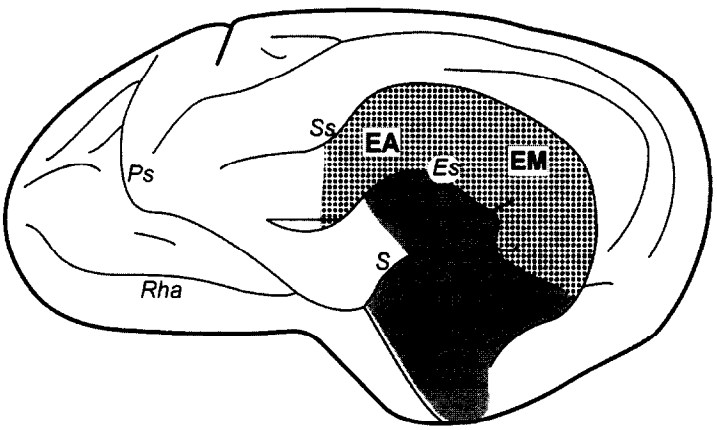

Fig 12. Scheme of the lateral aspects of the dog's brain showing intended lesions within the auditory cortex. Dotted area: primary auditory cortex (EM) and anterior ectosylvian gyrus (EA). Shaded area: auditory association cortical areas (EP, CP, S). Abbreviations: S, Sylvian fissure; Es, ectosylvian fissure; Ss, suprasylvian fissure; Ps, presylvian fissure; Rha and Rhp, anterior and posterior rhinal fissure, respectively;
S, sylvian gyrus; **EA**, anterior ectosylvian gyrus; **EM**, middle ectosylvian gyrus; **EP**, posterior ectosylvian gyrus; **CP**, posterior composite gyrus.

By contrast, the posterior part of the ectosylvian gyrus (EP) as well as the posterior composite (CP) and posterior part of Sylvian (S) gyri receive inputs mainly from the nontonotopic divisions of MGB, and so may be tentatively designated higher-order auditory fields (see Kosmal, 2000, article in this journal). Finally, the dorsal and anterior Sylvian areas, as well as the anterior part of the ectosylvian gyrus, receive significant projections originating mainly in polymodal thalamic nuclei. In the monkey, the density of MGB projections decreases gradually from lower-order to higher-order auditory areas, whereas the density of projections from polymodal nuclei of the posterior thalamus increase substantially along this same gradient (Kosmal et al., 1997). Moreover, data on dogs indicate that one of the projections from the primary auditory cortex, A1, is directed rostroventrally to the auditory area located in the posterior ectosylvian gyrus (EP), which projects in turn to the posterior composite gyrus (CP). Auditory areas are interconnected exclusively by local corticocortical axons and, on that basis, may be considered to be modality-specific (i.e. unimodal) auditory fields. This projection may constitute the neural basis for the perception and discrimination

of auditory stimulus quality. Another projection from A1, directed rostrally to the anterior ectosylvian gyrus (EA), could mediate perception of the spatial source of auditory stimuli.

Thus, it is possible that the ventral pathway, described above, would be involved in auditory stimulus recognition. To test this hypothesis, auditory cortical areas were removed in the dogs trained before on the auditory DMS task with the method described earlier (Kowalska, 1997). To stabilize dogs' behavior, additional control break and performance tests were introduced in preoperative training. Then, four dogs (group EP+CP) were given bilateral lesions of auditory association areas including the ventral part of posterior ectosylvian gyrus, posterior composite and posterior part of Sylvian gyri (Fig 12, shaded area). In two dogs (group EM+EA) the remaining auditory cortical areas: medial and anterior ectosylvian gyri, which consist of primary tonotopic auditory cortex, and an area with reverse tonotopy, respectively, were removed (Fig 12, dotted area). As shown on Fig 13, before surgery, there were no significant differences between groups in learning, retraining of DMS with original 1.5 s, or on the task with extended delays. After surgery, two dogs from group EA+EM and one dog from group EP+CP failed, whereas three other dogs from this group reattained the criterion with an elevated number of trials and errors, in comparison to their presurgery performance (Kowalska et al., 1998; 1999a). Next, two dogs, after EA+EM ablations and three dogs with EP+CP lesions, were trained to respond toward a natural sound (CS+), and then to differentiate between CS+ and another natural sound (CS-), to the 90% criterion in 100-trial block.

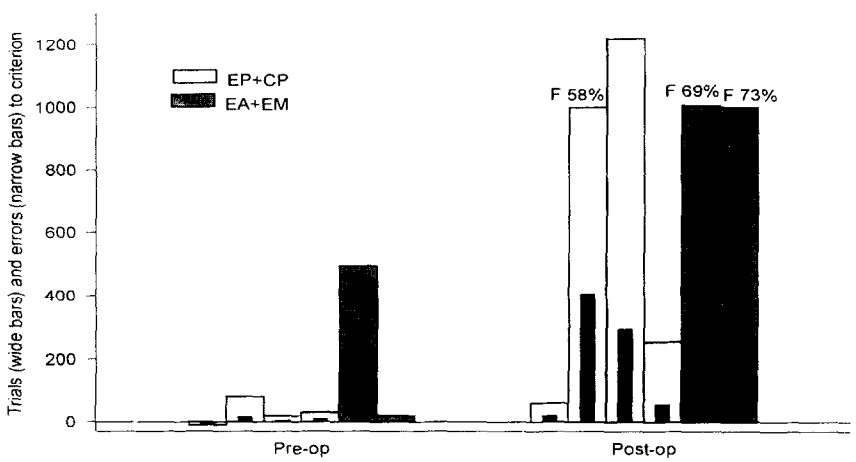

Fig 13. Numbers of trials (wide bars) and errors (narrow bars) to criterion before and after auditory cortical lesions on an auditory recognition task. F, means that animal did not reach criterion, % indicates the level of performance in last 100 trials of training.

As Fig 14 shows, dogs who received the lesion to auditory association cortical areas (EP+CP) learned to localize the acoustic stimulus and they were able to make the differentiation, whereas dogs who had damage to the primary auditory cortex (EM), together with area EA, were not able to localize the sound (Kowalska et al., 1999a). These preliminary data are consistent with earlier findings in dogs showing a significant impairment of a go-left, go-right differentiation task with two locations of an auditory frequency cue, after ablation of the EM and EA areas (Stępień et al., 1990). It was also shown that a combined lesion to the EM, EA and mostly posterior part of the Sylvian gyrus resulted in impaired go, no-go differentiation of two differently located acoustic stimuli, both for metronomes and tones, whereas, lesions limited to the Sylvian gyrus did not impair the task (Szwejkowska and Sychowa, 1971). The extensive lesion to the ectosylvian and Sylvian gyri (area EA, EM, EP and S) also impair the ability of dogs to localize brief sounds, only when the goal boxes were located more than 125 cm away however, they could successfully discriminate brief sounds even when the goal boxes were located 250 cm away (Heffner, 1978). More recent data indicate a deficit in the perception of location of auditory stimuli after lesions to the ectosylvian cortex (Stasiak et al., 1993). Even the borders of the lesions differed between studies, it seems probably, that impairments of the tasks, involving localization of sounds, might be caused by injury to the dorsal pathway directed to the anterior ectosylvian gyrus, which could mediate perception of the spatial source of auditory stimuli.

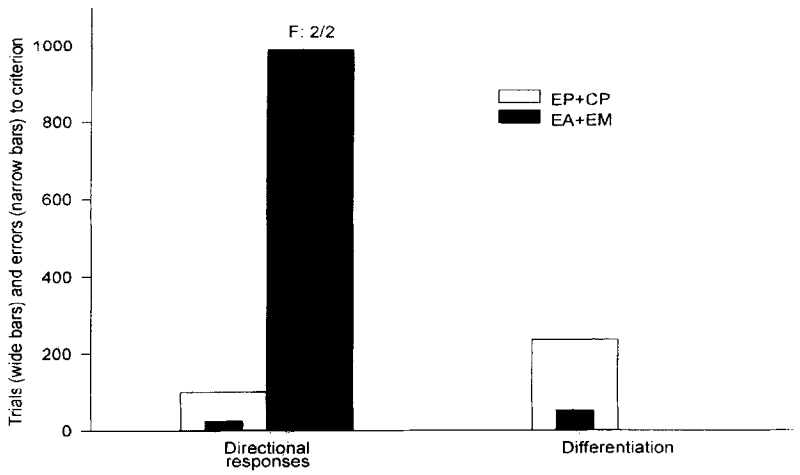

Fig 14. Numbers of trials (wide bars) and errors (narrow bars) to criterion in postoperative training of simple response toward the auditory stimulus (directional responses), and in training of differentiation between two natural sounds, for the groups of dogs given auditory cortical lesions. F: 2/2 means that two out of two animals did not reach criterion.

Our results on dogs suggest a diverse contribution of several auditory cortical areas to localization and recognition of natural sounds. The assumption about the possible role of the auditory association cortex in recognition of auditory stimuli is supported by recent results obtained in monkeys in Mishkin's Laboratory, showing impairments on the auditory recognition task after lesions to the auditory association areas (Saunders et al., 1998). Other support came from preliminary data obtained on rats in our collaborative study with Brown and his colleagues from Bristol University in UK, showing a significantly higher expression of the immediate early gene c-fos produced by novel rather than the familiar sounds in temporal auditory association cortex. This difference did not reach significance for the rhinal cortex as well as for the primary auditory cortex (Wan et al., 1999). These results are consistent with those from lesion studies in both the dog and the monkey suggesting an important role for the auditory association cortex in auditory recognition memory.

In humans, clinical data indicate severe asemantic recognition of sounds after temporo-frontal and temporo-parietal lesions (Clarke et al., 1996), however, detailed analysis of the function of specific areas in the human brain is difficult, because of the large variability in the size and location of lesions. Recent fMRI studies on human auditory cortex indicate activation within supratemporal plane caused by a matching-to-sample task with variable tones (Scheich et al., 1998). Although the human auditory areas are involved in much more complex auditory processing including verbal communication, there is very little information concerning basic neural processes underlying speech and language. Animal models could help to understand these basic phenomena.

In addition to studies that have been completed, further studies need to be performed in order to evaluate and elucidate more detail concerning the neural circuits underlying both auditory recognition processes as well as perception of auditory location.

6. Conclusions

The temporal lobe of the dog is involved in a variety of learning and memory functions. Spatial memory is impaired after bilateral hippocampal lesions but not after anterior temporal lesion or rhinal cortical injury, however hippocampal lesions have little effect on well-consolidated and over-trained spatial delayed responses. Auditory recognition memory is not impaired after hippocampal and/or rhinal cortex lesions, but it is impaired after a lesion to auditory association cortical areas. This indicates that auditory memory is organized differently from memory in other sensory modalities. The current review clearly indicates that research in dogs can substantially impact our understanding of processing of sensory information in the brain.

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