Place navigation impaired in rats with hippocampal lesions

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The hippocampus is a structure that plays a crucial role in spatial navigation and memory. The paper discusses the effects of hippocampal lesions on the ability of rats to navigate and remember their environment. The study involved placing rats with lesions in their hippocampus in a maze and monitoring their performance over time.

Female Lister rats (n = 31) were subjected to the following procedures: total hippocampal lesions (n = 10), superficial cortical lesions (n = 13), sham surgery (n = 4), and a special adjustable head-holder (n = 1). Animals in the hippocampal lesion group had holes drilled in their skulls, and a small amount of ventral and lateral hippocampus was removed by aspiration. Oper- ators showed no hippocampal damage, and the lesions in the control group had burs holes drilled in their skulls. On completion of their skull, still suffering from concomitant hippocampus, the dorsal and control group animals had comparable lesions in the hippocampus, and the entire dorsal and ventral hippocampi were removed by aspiration. Oper- ators showed no hippocampal damage, and the lesions in the control group had burs holes drilled in their skulls. On completion of their skull, still suffering from concomitant hippocampus, the dorsal and control group animals had comparable lesions in the hippocampus, and the entire dorsal and ventral hippocampi were removed by aspiration.

On day 1, the rats were placed in a pool of water (1.32 m) and allowed to swim freely for 1 min in one of four locations in the middle of the pool. A platform was hidden (SW, NW, ND, and SE), 0.33 m from the side walls. Different clear perspex, was hidden by adding 2.31 m of milk to the water level. A second platform, 2 cm taller and of the water, was set at a later stage in its circumference around the water to include the platform. The task's route was thus followed for the next 2 days, was to find and escape from the training, and it was always in a fixed position. For a given stage of data, the two tracks, which we call police navigation, each of the experimental animals, compared to escape to the other rat was required to learn uniquely with respect to the null platform's action. All rats swam effectively using the characteristic adult swim- ming pattern. The times taken to escape from the water during the platform, and the normal cortical and hippocampal lesions groups were shown in Fig. 2. Rapidly the water with saltations, (comparable to escape of< 8 s). The hippocampal-lesion group showed a significant impairment in the place-navigation task (trials 1-20). When the platform was used, this impairment declined dramatically and disappeared when the platform was removed (trials 30-40), The platform having been placed for a rat trained previously to find the hidden platform at SE, the place navigation impairment returned to the level of intact controls (trials 3-50). Even though the platform had occupied in the preceding phase of training.

Detailed analysis of the behavioural performance of each group and the results of two transfer trials provide further insights into the nature and magnitude of the deficit after hippocampal lesioning. The animals did not show any difference in the time required to cross the platform, or the time required to cross the platform. The time to cross the platform was not significantly different between the two groups. The platform was removed after the training was continued with the hidden platform (trials 3-50). Even though the platform had occupied in the preceding phase of training.

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(Fig. 2). The directional heading of the hippocampal-lesion rats when they set off from their starting position on trial 28 was no more likely to be towards the platform than in any other possible direction. These results imply that hippocampal-lesion rats can learn a set of escape strategy (for example, that escape is possible) but are substantially poorer at learning where the hidden platform is located and, unlike normal and cortical-lesion rats, they were unable to learn to swim towards it from a distance.

The validity of this place navigation deficit was assessed in two separate transfer tests conducted on trials 29 and 42, immediately after the four daily trials of days 6 and 8. For transfer tests, the hidden platform was first removed from the apparatus, then the rats were placed in the pool for 60 s with no opportunity for escape, and their movements observed. The results were striking. Control and cortical-lesion rats swim in a persistent and consistently across the former platform location whereas the hippocampal-lesion rats did not. The hippocampal-lesion rats did not merely swim around the side walls. To demonstrate this, animals were marked on the video screen indicating the exact surface area and former positions of the platform in each of the four cardinal quadrants. The total number of quadrant, which an individual rat passed through during the 60-s test was 7.6, 6.8 and 8.6 for the hippocampal, cortical-lesion, and control groups, respectively (F < 1). The groups were distinguished by which quadrant they passed through: an individual hippocampal-lesion rat was no more likely to pass through the quadrant marking the platform position used during training than an is at any other quadrant (Fig. 3a). We observed to tend the part of the hippocampal-lesion rats to remain in the vicinity of the training annulus once they had eventually reached it (compare with ref. 7). Thus the deficit produced by hippocampal lesions was total. Furthermore, with respect to the lack of spatial bias revealed in the annulus measure, the deficit was apparent in all 10 rats of the experimental group. Our interpretation of these findings is that, whatever their other effects, hippocampal lesions do cause a profound and lasting impairment in spatial understanding. It could be argued, however, that while matched for motor requirements, motivation and reinforcement, the place- and route-navigational tasks are not matched for task complexity. Perhaps hippocampal-lesion animals perform poorly on the spatial task because it is complex (albeit a task learned by normal animals in less than 10 trials), and perform better on the visible platform tasks because it is easier, rather than because the spatial component is then redundant. If this is the basis of the diacronie of effects in the two tasks, then at least some spatial bias should be shown by some of the hippocampal-lesion animals in a transfer test conducted after training on the ostensibly easier visible platform task. Transfer test 3, conducted immediately after trial 41 on day 8, examined this possibility. In trial 41 itself, there was no significant difference in the latency, path length, or directionality of escape behaviour across groups (P > 0.1), all animals escaping rapidly by means of short, direct paths.

Fig. 2. The actual path of the control rat (defined in terms of path (top) and error (bottom) in each group on trial 28 just before the first transfer test. The paths were traced using a video screen placed above the pool. One experimenter (FGJ) set up the platform position of the room and maintained the correct platform position. The experimenter (J. C. D.) removed the trained rats from the pool and placed them at the entry point. The path was open to the rooms which included a window, and high, bright lighting in the pool. The paths obtained by the rats were transformed from the video screen and measured. Path length for hippocampal-lesion rats took 4.64 s to reach the platform, whereas for the control and cortical-lesion groups took 2.32, 3.02 and 7.20 s, respectively. A majority of subjects showed that the hippocampal-lesion group took significantly longer than both the cortical-lesion and control groups (P < 0.001), which is not true and did not differ significantly from each other (P > 0.05). Furthermore, the accuracy of the layout of the platform was inspected as follows. We measured the angle subtended by a tangent to the circle path at a point 0.5 m from its starting position, and a few interesting this point the corners of the platform. This might be 81° from the correct direction for the hippocampal-lesion group, whereas for the cortical-lesion and control groups, the angle was 34° and 38°, respectively (Kruskal-Wallis, H = 5.87, df = 2, P < 0.033, one-tailed).
Evidence for dendritic competition in the developing retina

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At present little is known of the rules regulating dendritic morphology. Several studies have demonstrated that the shape of the dendritic tree depends on its affector supply. The ganglion cells of the retina provide a particularly useful model system for the study of dendritic development. In vitro, the ganglion cells are not only removed from their normal environment, but also deprived of afferent inputs. Thus, the shape of the dendritic tree is determined by intrinsic factors and not by external influences. In the developing retina, it is possible to examine how the absence of neuronal activity affects the pattern of dendritic growth. The results presented here suggest that the dendritic trees of ganglion cells are not only influenced by intrinsic factors, but also by extrinsic factors, which are mediated by the activity of the retinal ganglion cells.

Experiments were performed on 11 hooded Lister rats. On the day of birth, the rats were anaesthetized by intraperitoneal injection of 1 ml of 1% chloral hydrate. The retinas were removed and placed in a test tube containing Tyrode's solution. The retinal ganglion cells were stained with a combination of 1 μl of 0.1% alcian blue and 1 μl of 0.05% rhodamine conjugate. The retinas were then mounted on a glass slide and viewed under a microscope. The dendritic trees of the ganglion cells were photographed at 100x magnification. The photographs were then analyzed using an image analysis system. The results showed that the dendritic trees of ganglion cells are not only influenced by intrinsic factors, but also by extrinsic factors, which are mediated by the activity of the retinal ganglion cells.