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Précis of O'Keefe & Nadel's *The hippocampus as a cognitive map*

John O'Keefe
Department of Anatomy and Embryology, University College London, London WC1E 6BT, England

Lynn Nadel
Department of Psychology, Dalhousie University, Halifax, N.S., Canada B3H 4J1

Abstract: Theories of spatial cognition are derived from many sources. Psychologists are concerned with determining the features of the mind which, in combination with external inputs, produce our spatialized experience. A review of philosophical and other approaches has convinced us that the brain must come equipped to impose a three-dimensional Euclidean framework on experience – our analysis suggests that object re-identification may require such a framework. We identify this absolute, nonegocentric, spatial framework with a specific neural system centered in the hippocampus.

A consideration of the kinds of behaviours in which such a spatial mapping system would be important is followed by an analysis of the anatomy and physiology of this system, with special emphasis on the place-coded neurons recorded in the hippocampus of freely moving rats. A tentative physiological model for the hippocampal cognitive map is proposed. A review of lesion studies, in tasks as diverse as discrimination learning, avoidance, and extinction, shows that the cognitive map notion can adequately explain much of the data.

The model is extended to humans by the assumption that spatial maps are built in one hemisphere, semantic maps in the other. The latter provide a semantic deep structure within which discourse comprehension and production can be achieved. Evidence from the study of amnesic patients, briefly reviewed, is consistent with this extension.

Keywords: cognitive map; hippocampus; memory; spatial representation

In *The hippocampus as a cognitive map* (O'Keefe & Nadel 1978) we set out to synthesize four basic pieces of information: The existence of place-coded neurones in the hippocampus of the freely moving rat; Vanderwolf's (1971) finding that, at least for the rat, theta activity in the hippocampal EEG is related to some aspect of movement; our conviction that the underlying deficits in animals with hippocampal lesions consist of the absence of place learning and of exploration; and the profound retrograde and anterograde global amnesia seen after hippocampal damage in humans. The concept which enables us to tie together these different "facts" is that of the cognitive map. This notion was originally developed by Tolman to explain such phenomena as latent learning and place learning in the rat. For our purposes it was necessary to specify in considerable detail what we meant by a cognitive map, how this map was instantiated in the hippocampus, and what effect the loss of this mapping system would have on an animal's behaviour.

Theories of space

The first chapter is concerned with a brief history of theories of space, as developed primarily by philosophers. We started with the ideas of Newton, Berkeley, Leibniz, and Kant because it was with these thinkers that modern ideas about space began. The main difference amongst these writers centred on questions as to whether space was absolute or relative and whether it was an attribute of the physical universe or a property of the mind.

What do we mean by absolute and relative space? *Absolute space* is viewed as a stationary framework within which material objects can be located but which exists independently of particular objects or objects in general. Objects are located relative to the places of the framework and only indirectly, via this framework, to other objects. In contrast, *relative space* designates a set of relations amongst objects or sensory inputs which in themselves are inherently nonspatial. Objects are located relative to the observer's body and other objects; relative space does not exist independently of the existence of objects. There can be several types of relative spaces. Simple relative spaces differ in terms of the axes to which they are referred and from which they emanate: the body, the head, and the eye are the obvious ones, but there may be others. An object is to my left, in the upper left quadrant of my visual field. Higher order relative spaces locate objects with respect to other objects in terms of their mutual relations to the observer. Object A is to the left of object B.

The difference between absolute and relative spaces can be illustrated in the following way. Let us imagine ourselves standing at the edge of a billiard table on which there are only two balls: a red and a blue. From our initial vantage point the red ball is to the left of the blue one; as we walk around to the other side of the table, the balls change their relative positions, first "moving" into line with each other and then ending up on the opposite sides of each other in relative space. The blue ball is now to the left of the red one. But with reference to the absolute spatial framework of the table and the room, the balls have not moved at all. Rather, it is the observer who has changed his position from one place to another.

Newton was one of the major architects of the absolute theory of space. He believed that the physical universe consisted of atoms set in an absolute space. The mind can never directly apprehend this absolute space but conjectures its existence from experience with the

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position and movement of objects relative to each other. In contrast to Newton, neither Leibniz, Berkeley, nor Kant believed that space was a property of the physical universe. Each thought that it was constructed by the mind in one way or another. Berkeley, for example, felt that the mind was incapable of knowing anything about the physical universe as it really was but constructed ideas on the basis of sensations. One of these constructs was space, and this mental space was a relative one, based on the relations among sensations. Tactile sensations were regarded as primary and Berkeley traced many of our basic notions such as bodies and space back to these tactile sensations. The difference between bodies and empty space lay in the fact that the former offer resistance to motion while the latter does not.

Kant followed Berkeley (and Hume) in the assertion that we can have no direct knowledge of the external physical world. All that we know is the result of the interaction of the unknowable things-in-themselves and the structure of our minds. He also believed that the notion of space was a feature of the mind. Unlike Berkeley, however, he did not think it was an idea which the mind constructed from sensations but rather that it was part of the machinery of the mind, part of the mechanism by which the mind organized the sensations. Furthermore, he argued that this mechanism was not derived from sensations but was ontologically prior to them since one could conceive of a space without objects or sensations but not vice versa. Kant also differed from Berkeley in that he thought the spatial framework of the mind was absolute and not relative.

This Kantian notion that the mind contains an a priori system for organizing sensations into an absolute spatial framework is the one that comes closest to the ideas expressed in our book. The rest of the first chapter traces the development of the empiricist ideas of Berkeley and the rationalist ideas of Kant down to their present role in current psychological thought. Berkeley's most important heirs were Poincaré (1913) and Hull (1934). They both held that notions of space and spatial behaviour derived from the organism's experience with objects and the changes in those objects as a consequence of movement. The ability to use several different routes to go from one part of an environment to another was due to the development of the displacement group (Poincaré) or the habit-family hierarchy (Hull). These were both conceived as mechanisms for associating together *movements* which take the organism from the same starting position to the same finishing position [see Gyr et al.: "Motor-Sensory Feedback and Geometry of Visual Space" *BBS* 2(1) 1979].

Hull assumed that his habit-family hierarchies were learned by the animal as a result of many experiences in which different movements initiated in response to the same stimulus lead to the same goal. Neither he nor Poincaré ever faced up to the problem of how the animal knew that a stimulus or a part of the environment was the same as a previous one.

The Kantian idea of an innate spatial system that served as an organizing framework for inherently spaceless sensations had a rather more complicated history. One line of development involved attempts to answer such questions as: How are objects localized within a Kantian space (Lotze 1886, Müller in Herrnstein & Boring 1966)? How are the separate frames of the perceptual systems knitted together to form one continuous framework (Mach 1897)?

Concern with such questions led to a shift in emphasis away from the central Kantian spatial framework itself towards the nature of the peripheral sensations. An additional impetus for such a move came from the discovery that Euclidean geometry, far from being an inevitable guide to the nature of the physical world, was only one of several possible geometries.

One of the most important developments arising from the Kantian position was the nativist one. Nativists such as William James assumed that sensations were inherently spatial. That is, the extension of an object and the spatial relations between objects were regarded as being provided by the sensory inputs in the same way that colour and weight were. The most important contemporary exponent of this position is Gibson (1950, 1966), who has identified some of the features of visual sensations (e.g., texture gradients) which serve as cues to depth.

We concluded from our history that both schools of thought were correct and that there are several different ways in which the brain represents space. As Berkeley suggested, many of these spaces are relative, egocentric. They locate items in terms of coordinate systems which are centred on various parts of the body and which change with the movement of the body. In addition, however, there is one spatial system which has the properties of the Kantian, a priori, absolute system. This spatial system is not tied to the body but provides the organism with a maplike representation which acts as a framework for organizing its sensory inputs and is perceived as remaining stationary in spite of the movements of the organism.

Some properties of the locale and taxon systems

Others have used the notion of a mapping system to explain behaviour, most notably Tolman (1932, 1948), but in a rather loose and ill-defined way. We have spelt out the properties of a mapping system in considerable detail and contrasted its use with other means of finding one's way around an environment. We have called these other systems *taxon* systems and termed the strategies based on them *route* strategies. Route strategies or hypotheses can be further divided into *guidance* hypotheses and *orientation* hypotheses and these *may* be dependent on different brain structures.

A guidance hypothesis identifies an object or cue in the environment which should be approached or maintained at a certain distance. The motor component is not specified in detail by the hypothesis and can be any behaviour that successfully reduces the difference between the animal and the guide. Examples of guidance hypotheses are "go to the light," "avoid your mother-in-law," "follow that car."

Orientation hypotheses specify the motor behaviour in detail. They involve rotations within an egocentric spatial framework in response to a cue. The axis of the framework can be centred on the eye, the head, or the body. Examples of orientation hypotheses are "turn right 90° [clockwise around the body axis] at the corner," "look to the left 15°."

One of the main tenets of our theory is that much of the behaviour of animals with hippocampal lesions is due to the normal operations of the taxon systems. Therefore, although the main emphasis of the book is on the locale system and place hypotheses, we found it necessary to attribute enough properties to the guidance and orientation systems to enable us to predict the behaviour of animals dependent on them.

Two of the most important properties of the taxon systems are that information is stored on the basis of category inclusion and that changes are incremental. A category inclusion system stores two items with similar features in the same or neighbouring neural circuits. This results in interference when the same item is associated with two different items at different times or in different contexts. It follows that animals relying solely on the taxon systems should be particularly subject to interference effects. The second major property we attributed to the taxon hypotheses is incremental storage. That is, each activation of the information results in a small change in the strength of its synaptic connections and successive strengthenings or weakenings add to each other. Furthermore, we postulated that the effect of the activation of a taxon hypothesis varied with the time following the activation. The time course of this variation is expected to be complex and probably varies with the particular hypothesis. Nevertheless, this postulate predicts that the behaviour of animals relying on a taxon hypothesis will be significantly influenced by the intertrial interval.

In contrast, the hippocampal mapping system supports place hypotheses which have very different properties from taxon hypotheses. The map of an environment is composed of a set of place representations connected together according to rules which represent the distances and directions amongst them. As Kant suggested, places and locale space do not exist in the physical world but are notions constructed by the brain in order to organize sensory inputs. A small number of stimuli (two or three) occurring with a unique

spatial configuration when an animal is in a particular part of an environment are sufficient to identify a place in the map. At any given point in an environment there are usually a large number of such sets of stimuli and therefore the identification of a place in an environment does not depend on any particular cue or group of cues. The distance and direction vectors which connect the places in the map of an environment are derived from the animal's movements in that environment. Unlike the neural connections underpinning taxon hypotheses, maps are formed during exploration. Animals can use maps to locate themselves in an environment, to locate items (including rewards and punishments) in that environment, and to move from one place to another by any available route. Finally, part of the mapping system involves a mismatch mechanism which signals changes from expected sensory inputs at a particular place.

Examples of locale hypotheses are "this is a dangerous environment," "there is a safe place in this dangerous environment," "go to place A to obtain water." We postulated that place hypotheses are different from taxon hypotheses in the following ways:

(1) Information is entered into the mapping system on an all-or-nothing basis. When an animal attends to a cue it is entered in the place representation corresponding to the animal's position in the environment. On subsequent exposures to that cue in that place there is no additional increment in the strength of that representation. Of course, different cues in the same part of the environment can be attended to and entered into the place representation on different occasions, making the place representation richer but not stronger.

(2) In contrast to the taxon stores, there is little interference between identical or similar items in the mapping systems when they occur in different parts of an environment or in different environments, since they are stored in different place representations.

(3) Similarly, there are only minimal changes in the locale trace strength after activation since different pathways can be used to activate the same place representation.

(4) The place system enables the animal to act at a distance. It can go towards or away from places which it cannot perceive if they are both part of the same map.

Hippocampal anatomy and physiology

Our goal in the chapters on hippocampal anatomy and physiology was twofold: to review the literature, and to select and interpret some of the data to generate a set of "facts" on which to base a model of hippocampal function. The "pieces and patches" (as we have called them elsewhere, Nadel & O'Keefe 1974) which we selected are as follows:

(1) The hippocampus is made up of three separate systems, the fascia dentata, CA3, and CA1. Each system consists of a large matrix of similar projection cells, the dentate granules and the CA pyramids, together with a set of interneurones, the most prominent of which are the basket cells of Cajal. The interneurones control the excitability of the cell somas and probably also the dendrites of the pyramidal and granule cells. The main flow of information amongst the three subsystems appears to be in the direction from the fascia dentata to CA3 and thence to CA1. The projections are topographically precise, a narrow strip of granule cells sending their axons to the dendrites of a narrow strip of CA3 pyramidal cells; the axons of these pyramids in turn innervate a narrow strip of CA1 dendrites. These connected strips, or lamellae, form the major anatomical unit within the hippocampus. Some of these projections show an interesting pattern of termination which may be important for models of hippocampal function. The fibres do not all end at the same level of the dendrites. Instead, there is a systematic shift in the termination such that fibres end nearer the soma at one end of the lamellae than at the other.

(2) There are two major inputs to the hippocampus: one from the entorhinal cortex via the perforant path and the other from the medial septum and brainstem via the fornix. The entorhinal cortex receives inputs from many areas of the sensory cortex and it is likely that the entorhinal afferents to the hippocampus convey information about stimuli and perhaps their location in egocentric space. This projection has been studied anatomically and physiologically and has been shown to have the same precision as the intrahippocampal connections discussed under (1): small strips of entorhinal cells project only to a restricted hippocampal lamella. Each entorhinal fibre streams across the dendrites of many granule cells roughly at right angles to their orientation, making numerous synapses *en passage* in its course. With the exception of the diffuse medial septal input, this is the usual mode of afferent termination in the hippocampus.

The other major input to the hippocampus, the brainstem-medial septum pathway, has a much more diffuse termination, ending in all fields of the hippocampus and the dentate area. In the rat, this pathway appears to convey information about the animal's movements, in particular, about movements which change its position in space. In animals such as the rabbit and cat, it transmits information not only about movements, but also about arousal or attention. The major evidence for this assertion comes from studies of the behavioural and psychological correlates of theta in the hippocampal EEG, particularly the work of Vanderwolf (1969, 1971), Whishaw, and their colleagues (Vanderwolf, Bland, & Whishaw 1973). It is known that lesions of the medial septum eliminate hippocampal theta and that electrical stimulation of various parts of the brainstem elicits hippocampal theta. Thus one can conclude that one of the major functions of the brainstem-septal-hippocampal pathway is to impose the theta rhythm on the hippocampus and that this rhythm carries information about the animal's movements and, in some animals, its intention to move (arousal).

(3) The function of the sinusoidal theta rhythm appears to be that of a gate changing the excitability of the pyramidal cells from moment to moment. Intracellular microelectrode studies show that there is an intracellular rhythm inside the pyramidal cells so that the membrane potential of the cell soma varies in the same temporal fashion as the theta rhythm. Since the theta rhythm is largest in the dendrites of the pyramidal and granule cells, it seems likely that there is a theta modulation of the dendritic excitability as well. This means that afferent inputs arriving at certain phases of the theta rhythm will be more likely to activate the dendrites than those arriving at other times.

The distribution of theta within the hippocampus is different for rat and rabbit. In the rabbit, there is a progressive shift in phase as one moves across the surface of the hippocampus. An electrode penetrating through the rabbit hippocampus records two maxima in the amplitude distribution, one in the basal dendrites of the CA1 pyramidal cells and the other in the apical dendrites of the dentate granule cells. The theta recorded at the two peaks is 180° out of phase and there is a null zone between the peaks in the stratum radiatum of CA1 where the amplitude diminishes to zero. In the rat, on the other hand, Winson (1976a,b,c) has shown that although , as in the rabbit, there are two maxima in the amplitude distribution there is no null zone between them in the CA1 stratum radiatum. Instead, the phase difference between the CA1 theta and the dentate theta slowly shifts as one moves the CA1 electrode close to the dentate electrode. From this it appears that in the rat the CA1 apical dendrites are modulated in such a way that parts of the dendrite are at different phases of excitability from other parts.

These findings indicate that the theta mechanism locks into synchrony large areas of the hippocampus and entorhinal cortex so that the excitabilities of the cells have constant temporal relations with each other. In the rat, afferent inputs arriving at a CA1 apical dendrite patch that is in the depolarising phase of the theta cycle would be more likely to excite the cell than afferents arriving at a different, less depolarised patch of dendrites.

These and numerous other facts about the anatomy and physiology of the hippocampus can be combined in various ways to produce models which will act as sorters or cross-correlaters, organizing the afferents to the hippocampus according to the theta mechanism. Important questions then are: What is the theta related to? What is the principle according to which the afferents are sorted or correlated in the hippocampus?

(4) Numerous studies have correlated the hippocampal EEG with

the behaviour or psychological state of the animal. Even a superficial glance at the literature makes it immediately obvious that there are species differences in these correlates. In the rat, Vanderwolf et al. (1973) have clearly shown that hippocampal theta relates to a class of movements which includes walking, jumping, sniffing, swimming, and head movements, and which can be roughly described as "voluntary" behaviours. Large-amplitude irregular activity, on the other hand, is associated with automatic behaviours such as eating, drinking, and grooming.

The results in the rabbit and cat are slightly different. In these animals, theta occurs not only during voluntary movements but also during arousal or attention. Vanderwolf (1975) has shown that the arousal-related theta has a different pharmacological basis from the movement-related theta.

We can conclude that at least part of the basis on which the sensory inputs to the hippocampus are sorted is some aspect of the animal's movements. Experiments involving jumping and the initiation of running (Vanderwolf 1969; Whishaw & Vanderwolf 1973; Morris, Black, & O'Keefe, 1976) suggest that theta frequency may be related to the distance through which the movement translates the animal (or its distance receptor surfaces) relative to the environment. If this is so, then sensory inputs are being sorted in the hippocampus according to the distance between them in the environment.

(5) Studies of single units in the hippocampus of the freely moving animal can be divided into two categories: those which allow or encourage the animal to move around in space and provide it with a generous amount of spatial information, and those which restrict the animal's movements and limit the stimulating environment. The latter have tended to focus on a small range of stimuli, such as flashing lights, tones, and so on (Vinogradova 1970) or to examine changes during a classical conditioning paradigm (Olds 1965). Studies done in our laboratory and those of Hill (1976), Branch, Olton, and Best (1976), and Ranck (1975) find that spatial factors play a large part in the firing patterns of CA1 complex spike neurones. Some fired when the rat went to, or ran past, a place in the environment (the cell's *place field*) and others fired even more vigorously when the rat sniffed in a place because of the absence of something usually found there or the presence of something new. We have called these units "place" and "misplace" units respectively.

The pattern of firing of a second class of hippocampal units is closely associated with the hippocampal slow waves, in particular, the theta waves, and has the same behavioural correlates. Ranck (1973) has described these units and called them theta units. We have called them displace units in accordance with our belief that their major behavioural correlate is the animal's displacement in the environment.

Experiments in our laboratory have been designed to identify the factors which cause the place cell to fire in a particular part of an environment. Hippocampal unit activity was recorded from rats while they performed a place learning task on a T-maze set in an environment where there were only four cues to spatial location. The cues were controlled by rotating them and the maze from trial to trial so that they were the only cues to the location of the goal.

The results indicated that there may be two types of place unit. The first type has a place field which is built up from convergent excitatory sensory inputs each of which tends to fire the cell in the same part of the environment. Removal of some of the sensory cues, in particular visual cues, from the environment reduces or abolishes the firing of these cells in the place field.

The second type of place cell appears to have a place field which is constructed, at least in part, from inhibitory influences. Removal of some of the spatial cues causes these cells to increase their firing in the place field. A particularly dramatic example of this increased firing can be seen when the reward is removed and one is recording from such a cell having a place field at or close to the goal area. When all the spatial cues are removed this second type of place cell fires all over the environment with an increased rate.

The place fields of these neurones appear to be due partly to inhibitory influences on the cell when the animal is outside the field. The misplace cells are probably a subset of these cells. We suggest that the function of these cells is twofold: (a) to signal the animal's position in an environment, and (b) to detect a mismatch between the sensory inputs arriving in the hippocampus from a part of an environment and those which would be expected on the basis of the animal's current representation of that place in its map of that environment.

The output of these misplace cells was postulated to be one of the signals to the motor circuits that programme exploratory motor patterns such as myostatial sniffing. On this view, exploration is seen as an information-gathering behaviour which is intended first to build and then to update cognitive maps.

In addition to the output to the exploratory motor circuits, the mapping system makes available to other parts of the brain place representations which can be used in the solution of problems. For example, if an animal is hungry, the map of the environment in which it finds itself can be consulted to see if there is a representation of food there. If there is, then the map can be used to generate a motor programme which will take the animal from its current position in the map to the location containing food. A major chunk of *the hippocampus as a cognitive map* is devoted to a detailed consideration of the effects of hippocampal lesions on an animal's behaviour and the success of the major prediction of the theory that all of these data can be interpreted as a loss of place learning and exploratory behaviour.

Animals bereft of their hippocampus should still be able to learn tasks which can be solved using guidance or orientation hypotheses. As we have outlined them aboye, guidance hypotheses involve approach or avoidance of a specific cue by any available behaviour, while orientation hypotheses specify a particular rotation within an egocentric framework whose axis could centre on the body, the head, or the eyes.

One of the things which must be kept in mind is that the same problem can be solved by an animal using different hypotheses. For example, an animal which successfully learns to go to the right arm of a T-maze to obtain food could be doing so on the basis of a place hypothesis ("go to that place as defined by its position in the room"), an orientation hypothesis ("turn right 90° at the choice point") or a guidance hypothesis ("approach a cue in or behind the right goal arm; avoid a cue in or behind the left goal arm").

In a similar fashion, most tasks which psychologists assign to animals can be solved using more than one hypothesis. Even when two animals learn a task in the same number of trials, it is possible that they have solved it using different hypotheses. This can only be ascertained through the probe or transfer experiment in which the use of different hypotheses can be tested. In our example, the ability to rotate the start arm of the T-maze by 180° to the other side of the crosspiece would enable us to test whether an animal was using an orientation hypothesis (turning right) as opposed to a place or guidance hypothesis. Similarly, two goal arms which could be removed and interchanged would allow us to test for the use of an intramaze guidance hypothesis.

Where they exist, probe tests provide powerful evidence in favour of the cognitive map theory. Unfortunately, they have been used all too rarely in experiments with lesioned animals. Consequently, for much of our review of the lesion literature, we had to rely on a different strategy. Before looking at the lesion results for a particular task, we referred to the literature on normal animals and culled out the important variables which influence performance on that task. On the basis of this, we have made an estimate as to which hypotheses are used by the animals for solution of that task (and, equally importantly, which hypotheses retard solution). Where it seems likely that place hypotheses are exclusively or primarily used by normal animals (e.g., complex maze learning), we predict deficits in lesioned animals. Where other hypotheses predominate or are equally useful, there should be no deficit. Under certain circumstances (e.g., two-way shuttle avoidance), place hypotheses actually conflict with the solution of the problem, and here we predict that the lesioned rats should learn faster than normals.

Effects of damage to the hippocampal system in animals

Our analysis of the lesion literature has led us to the following conclusions:

(1) Exploration. Animals with damage to the hippocampal system do not explore new environments or novel objects. Although they are more active in open field tests they do not systematically inspect the whole environment as does the normal rat. Instead, they adopt stereotyped behaviour patterns, typically running around the outside of the environment. They do not decrease their activity over time as do normals, an indication that they have not incorporated information about the new environment into their mapping system.

In spontaneous alternation tasks, an animal is given two nonrewarded trials in a T-maze. Normal rats typically enter different arms on the two trials. We think this is due primarily to the animal's tendency to explore the less familiar parts of a novel environment. The lesioned rats either proceed randomly on the second trial or repeat their first choice, indicating an absence of this exploratory learning.

(2) Discrimination learning. Lesioned animals usually learn nonspatial discriminations as rapidly as do normals. The results on spatial discrimination are less clear because these can usually be learned using a place hypothesis or an orientation hypothesis. For example, in a simple T-maze in which the right arm is always baited, the animal can learn to go to the place in the room where the goal is (place hypothesis) or to make a 90° right turn at the choice point (orientation hypothesis). In such a spatial task, one would not necessarily expect a deficit in lesioned animals. One would expect, however, that they would have learned on the basis of a different hypothesis from normals. This is what has been found: on open elevated mazes normal rats used place hypotheses during initial training, switching to other hypotheses with continued training. Hippocampal rats, on the other hand, did not use place hypotheses at any stage of learning.

(3) Discrimination reversal training. Animals with hippocampal lesions show profound deficits in the reversal of spatial discriminations. The results with nonspatial reversals are mixed: rats and cats usually show a deficit while monkeys do not. The deficit in spatial reversal is explained by the different properties of the place system and the orientation system. Changes in the place system are all-or-none, while those in the orientation system are incremental and therefore slower. The normal animal quickly learns that there is no food in the usual place and can look in other places; the animal with a hippocampal lesion must continue to make the same body turn until enough incremental changes have summated to enable it to switch to the opposite orientation. Rats and cats fail to reverse a nonspatial discrimination (such as a black/white discrimination) but monkeys have no trouble doing so. The rats and cats fail, not because they persist in responding to the incorrect cue longer than the normal animals but because they switch into an inappropriate orientation hypothesis ("go to the left cue") rather than going to the previously unrewarded cue. When steps are taken to prevent these animals from switching into persistent orientation hypotheses, or when species are used which do not usually do so (monkeys), no deficit is seen.

(4) Maze learning. It has long been recognized that rats use several strategies in solving maze problems. Perhaps the most important of these is the goal-direction factor identified by Dashiell (1930). He found that rats seem to have a sense of the general direction of the goal and that this leads them to make more errors at choice points where the correct alley leads away from the goal than at choice points where it leads towards the goal.

Another factor which was identified as contributing to success in maze learning was Hull's (1932) goal gradient, which postulated that the effect of reinforcement was stronger as one got closer to the goal and that the later choice points in a maze should be learned more rapidly than the earlier ones.

According to the theory we are advancing, there are two ways that the hippocampal cognitive map would be useful to an animal running a maze. First, it locates the animal's position in the maze and thus underlies place hypotheses such as "go from place A (first choice point) to place B (second choice point)." Second, it locates the direction of the goal from any place in the maze and is therefore responsible for the goal-direction factor of Dashiell.

As predicted, animals with hippocampal damage are almost always worse than normals at learning complex mazes. Moreover, there is some evidence that they lack a goal-direction factor. For example, in a Lashley III maze they tend to make more errors which are sensitive to the goal direction than errors which are not. The evidence is not particularly strong, however, since the point has not really been carefully tested. Hippocampal rats nevertheless do benefit to about the same extent as normals from guidances placed at each choice point.

(5) Aversively motivated behaviour. Behaviour learned in response to threat causes considerable difficulties for theories of learning which posit that learning takes place as a result of drive reduction and reinforcement. Once an animal has successfully learned to avoid the threat, fear is reduced in the situation and it is difficult to identify the reinforcing event. [See Eysenck: "The Conditioning Model of Neurosis" *BBS* 2(2) 1979].

The cognitive map theory suggests that an important consideration is whether the threat can be identified by the animal as a place or an object in the environment. In general, frightened animals try to move away from threatening places or items and towards safe places. Under most circumstances the place system will predominate and an animal's first hypothesis will be "seek a safe place" or "avoid this dangerous place." Only when this is not possible do they select items which should be avoided.

The three most common avoidance tasks used in the study of animals with hippocampal lesions are one-way avoidance, two-way avoidance, and so-called passive avoidance. Many one-way avoidance tasks can be learned using either place or taxon hypotheses. Typically, the animal is placed in one side of a two-compartment chamber and after a short period of time the floor on that side is electrified. A directional guidance signal (e.g., a buzzer coming from the safe side) may or may not be turned on during this period as a conditioned stimulus. Where such a guidance is provided, the animal can learn to solve the task using either the guidance hypothesis "approach the cue," or the orientation hypothesis "turn 180° and run," or the place hypotheses "go to the safe side B" or "get out of the dangerous place A."

Most studies do not find deficits in one-way avoidance in hippocampal animals but about one third of the studies do. With a few exceptions, the studies conform to the expected pattern: those using a CS fail to find a deficit while those not using a CS produce a deficit. Two studies by Olton and Isaacson (1968, 1969) provide further support for the exaggerated importance of the CS to the hippocampals. In the first study, the effects of preexposure to the CS were tested; in the second, the effects of inescapable CS-UCS pairings prior to the avoidance training were tested. In both cases the performance of the hippocampal, but not the normal, animals was markedly affected by these treatments. Pre-training habituation to the CS retarded learning while prior CS-UCS pairings markedly improved it. [See Olton et al: "Hippocampus, Space and Memory" *BBS* 2(3) 1979].

In two-way active avoidance, the animal is not removed from the second compartment B, after it has fled there from compartment A. Instead, it is left there for a period of time and then required to shuttle back to the original compartment A on the next trial. Two important differences from one-way active avoidance are (a) there is no permanent safe place, only temporary respites from danger, and (b) correct solution depends upon the animal's entering a recently dangerous place. It is not surprising that this is a relatively difficult

avoidance task for animals to learn. Nor is it surprising that hippocampal animals, unhampered by conflicting place hypotheses, are almost invariably quicker than normal controls to solve two-way active avoidance tasks. They do not freeze in the situation, a natural response for the normal animal in an inescapable place. Nor do they hesitate to return to a dangerous place. This latter disadvantage shows up in the learning pattern of normal animals as a reluctance to return to one side of the box: they learn to avoid in one direction at a much faster rate than in the other. In contrast, hippocampal animals learn to go in both directions at about the same rate.

Passive avoidance tasks differ from active avoidance tasks in that correct solution requires the animal to refrain from doing something. That something might reflect a natural tendency, such as entering a small dark compartment, or it might be a learned behaviour, such as running down an alley to obtain reward. The animal is punished for performing the response and the learning consists in failure to repeat the response or an increased latency in doing so. We and others have emphasized that the term *passive* is misleading since passive immobility is not necessarily the best solution: the animal can actively engage in an alternative behaviour.

Hippocampal animals perform relatively normally on some passive avoidance tasks but are deficient on others. When they can learn to avoid an object or cue, or to cease responding as in step-down or step-through passive avoidance, they are normal; when a place must be avoided, such as in a runway task, they show a deficit. The clearest example of this is the often used approach-avoidance task. Consider the following study by Nonneman and Isaacson (1973): cats were first trained to approach a food dish and then shocked for doing so. On subsequent trials the hippocampal cats showed a passive avoidance deficit in terms of their latency to start running towards the area (place) containing the food dish. These animals did not touch the food (cue) more rapidly than control animals, however. Nearly all studies which distinguish between avoidance of the threatening place and avoidance of the threatening object show a similar result.

One final passive avoidance paradigm, conditioned taste aversion, supports our contention that there is no deficit in hippocampal animals in avoiding items, as opposed to places. Of eight studies, six found no deficit, as against one which did find a deficit. (See O'Keefe & Nadel 1978, Table A23, p. 467). In one study there were deficits only with dorsal but not ventral hippocampal lesions.

(6) Classical conditioning and operant tasks. There is little evidence concerning the effect of hippocampal lesions on classical conditioning. What there is suggests that there is no deficit with simple acquisition. For example, hippocampal rabbits acquire the nictitating membrane response more rapidly than normal rabbits. Similarly, hippocampal animals show no difficulty in learning about incentives.

In operant tasks, rats with hippocampal lesions learn to press the lever and acquire food on a continuous reinforcement schedule at least as rapidly as normal rats. This is not unexpected since there is little role for the locale system here. More complicated schedules may require the animal to use the locale system to aid in "solving" them. Take, for example, differential reinforcement of low rates of lever pressing (DRL). On this schedule the animal must withold its response for a fixed period of time before reward becomes available. Lever presses before the interval is over reset the delay and result in lost rewards. Since it seems unlikely that an animal such as a rat or pigeon can tell time, the most likely means for "solving" such a schedule is to engage in some type of "collateral behaviour" during the interval. One such behaviour is to actively avoid the area where the lever is. Other collateral behaviours rely on sequences of taxon-based behaviours. In versions of this task in which a cue signals the end of the delay period, the animal can use the guidance hypothesis "approach the lever when the light is on." Hippocampal rats have difficulty with DRL schedules when they are pretrained on continuous reward, presumably since this strengthens the taxon-based lever press hypothesis and makes it more difficult to find an alternative taxon "collateral" behaviour. The provision of a cue signalling the end of the delay interval improves the lesioned rats' performance to the normal level.

(7) Spatial delayed response and spatial alternation. These are both tests of spatial memory. In the delayed response task, one of two or more positions is baited within the view of the animal, which is then required to choose that alternative after a delay. Most delayed response tasks would appear to be dependent on locale mechanisms in that the animal can remember the place where reward is: as expected, hippocampal animals do poorly on these tasks. Spatial alternation tasks require the animal to alternate its choice from one trial to the next. When tested in a T-maze, the animal should make a right turn, followed by a left and then a right and so on. This task is also usually failed by hippocampal animals. When the alternation task is performed in an operant chamber, the results are mixed, some studies finding deficits, others not. One obvious difference between the operant chamber and the T-maze is that in the former an animal can reposition itself after each trial so as to be ready for the next. Holding this position substitutes for the memory of the last response. This appears to be the strategy used by the lesioned animals in those studies where they were successful.

(8) Reaction to reward change – extinction. One of the most obvious and replicable deficiencies in animals with hippocampal lesions is their inability to alter their behaviour when reward is removed from the place it is usually found. This is seen most clearly in runway tasks where the animal is taught to run from one end to the other for reward. When the reward is withdrawn, normals typically cease running within a small number of trials. In contrast, hippocampals continue to run for a great many trials, seemingly unaware that there is no longer any food at the end of the alley. Interestingly, deficits on extinction in operant chambers are much smaller and not seen at all in some studies. We think that the extinction situation is formally equivalent to the passive avoidance task. It can be solved either by a shift in the locale system or the taxon system. At the start box of an extended runway, the locale system informs the rest of the brain whether or not there was reward in the goal box on the last trial. Changes in behaviour in the start box are almost wholly attributable to the hippocampal locale system. The taxon systems contain the information about how often certain cues have been associated with reward in the past. They control the animal's response primarily to the cues of the goal box. Extinction based on these systems should be a slow decremental business, starting in the goal box and working back up the runway.

One important observation on extinction was made by Jarrard and Isaacson (1965), who reported that extinction of running in an alley in hippocampal lesioned rats depends on the intertrial interval. Large deficits were observed only when spaced trials were used. As in other tasks, it appears that the brain systems on which the lesioned animals must rely are much more sensitive to the temporal aspects of their activation than is the hippocampal system.

(9) Reaction to reward change – frustration. When an animal fails to find reward where it expects it, it may become frustrated. This frustration is assumed to be mildly aversive, to energise behaviour, and to provide cues as to which behaviours can be conditioned. It has been suggested by Gray (e.g., 1970) that the hippocampus is important to frustration. We found little evidence in the literature to support this suggestion. From studies which report either decreased or increased frustrative reactions in hippocampal animals in response to various treatments, we conclude that a direct effect of hippocampal lesions is unlikely.

(10) Other effects of hippocampal lesions. The loss of a cognitive mapping system might have subtle effects on many apparently nonspatial behaviours. Thus, for example, maternal behaviour might be affected if the dam has trouble locating the place where she left the pups or the nest. Similarly, aggression probably depends on the animal's perception of the size of its environment and its territory

within that environment. Thus one might expect some studies to indicate deficits in these tasks depending on the exact nature of the testing situation, and this is indeed what is found.

There does not appear to be any major change in eating or drinking after hippocampal lesions, although these is evidence that electrical or chemical stimulation of the hippocampus can result in stimulus-elicited drinking or eating. We think these latter effects may be indirect, resulting from the effects of the hippocampus on other structures such as the hypothalamus [see also Toates: "Homeostasis and Drinking" *BBS* 2(1) 1979].

(11) Endocrines and the hippocampus. There are subtle changes in the pituitary-adrenocortical system (involving ACTH and corticosterone in the rat) after hippocampal damage. In contrast, other hormonal systems (e.g., gonadal) have not been implicated in hippocampal function. There are three lines of evidence which support the notion that the hippocampus plays a role in hormonal function. First, uptake studies show that labelled corticosterone is preferentially taken up by the hippocampus. Second, injections of ACTH and corticosterone have been shown to modify hippocampal unit activity and hippocampal EEG activity. Third, lesions and stimulation of the hippocampus have been reported to affect both the diurnal fluctuations of plasma corticosteroids and the dynamic hormonal response to environmental uncertainty. The latter may indicate that the major relationship of the corticosteroid system is with the CA1 misplace system.

Extension of the theory to humans

One of the major goals we set ourselves in *The hippocampus as a cognitive map* was to develop a theory which could apply to the literature on human amnesia following damage to the hippocampal system, as well as to the subhuman animal literature. In order to do so, it was obvious that we would have to modify the theory to take into account the well-documented lateralization of the human brain [see Corballis & Morgan: "On the Biological Basis of Human Laterality" *BBS* 1(2) 1978]. Accordingly, we postulated that the left and right human hippocampus perform different functions, in part because of the different information they receive from the left and right neocortex. The right hippocampus of the human functions in a manner similar to that of the rat, acting as a one-trial episodic memory framework which stores items and events within a spatio-temporal context. The left human hippocampus provides a linguistic framework for the organization of narratives, a framework with properties similar to those which linguists have ascribed to semantic deep structure [see Arbib & Caplan: "Neurolinguistics Must Be Computational" *BBS* 2(3) 1979].

As in the rat, the human taxon and locale systems have different properties which stem from their different modes of storing information. The taxon system stores items in an incremental fashion, with each item listed with similar items. Consequently, taxon-based memories are prone to interference between similar items and the strength of the memory trace depends on the time elapsed since it has been activated. Retrieval of taxon information is by category name, or by association with other items.

In contrast, information is stored within the locale system as specific individual episodes within a specific context. Because of this contextual embedding there is little interference between similar items in the store; and the multiple access to each item stored in a maplike structure means that there is very little change in trace strength after activation.

The major modification to the mapping system in the human involves the inputs to the left hippocampus. In keeping with the evidence that left temporal lesions result in amnesia for verbal material, we postulate that the left hippocampus receives information about linguistic entities and sets these, rather than items drawn from the physical world, into a mapping space. This has led us to the notion that the left hemisphere cognitive map acts as a semantic map, providing something like a deep structure for connected discourse.

Work in linguistics by Chomsky (1957, 1965), Fillmore (1968, 1971), and others has suggested that in addition to the left-to-right ordered temporal structure of surface sentences, there must exist a deep structure which explains, among other things: (1) the fact that semantically related words in a sentence are often not contiguous, (2) superficially different sentences such as the active 'the boy bit the dog' and the passive 'the dog was bitten by the boy' have the same meaning, and (3) the same sound can have more than one meaning depending on the context. Analysis of different linguistic theories of deep structure suggests that they all entail the notion of a framework which specifies the semantic relations among the various entities of the sentence. In Chomsky's work, for example, this framework is explicit; in other work (such as Fillmore's) the framework is implicit and the relations among the entities are specified by a set of cases (agent, object, goal, instrument etc.) which relate the entities to each other [see Chomsky: "Rules and Representations" *BBS*, forthcoming].

We have followed up a suggestion of Jackendoff (1976) that sentences about existence or movement in physical space form a model for all sentences and that a successful semantic analysis of such sentences could be generalised to cover all sentences. After Jackendoff, we sketched out a model of a semantic map which provides the deep structure for sentences about the existence and movement of entities in physical space. For example, the sentence "the rock fell from the roof to the ground" is represented by two places in a map (the roof and the ground), the existence of an entity (the rock) in one of these places (the roof) until some unspecified time t, and the movement of the entity from one place to another at time t. Various transformation rules allow the full range of active sentences to be generated from this semantic map.

Nonspatial sentences can also be represented in semantic maps. Here, however, the mapping space does not represent entities existing and moving in physical space but in *influence* space, *identificational* space and *circumstantial* space. Consider for example, the sentence "Harry gave the book to the library." Here the action is not one of physical movement but the "movement" of the book from the possession of Harry to the library. This movement can be represented in a possessional or influence space in a manner exactly analogous to movement in physical space and the transformation rules for generating surface sentences from this map are the same as those for the physical map.

Other types of semantic spaces would map sentences about changes in identity such as "The rock went from smooth to pitted," and changes in circumstances such as "The librarian went from laughing to crying."

The human amnesic syndrome

Our understanding of the functions of the human hippocampus comes from work on two groups of patients: those with surgical removal of temporal lobe structures including the hippocampus, and those suffering from Korsakoff's syndrome, which is usually associated with damage to the mammillary bodies and the dorsomedial thalamus. Much of this research has concentrated on the memory deficit which is one of the more obvious features of the syndrome.

As it does with animals, the cognitive map theory makes two sets of predictions about changes after hippocampal damage in humans. The first set flows from the absence of a locale system while the second results from the intrinsic properties of the remaining taxon system.

(1) Bereft of the hippocampal mismatch system, humans should show *a lack of curiosity*, and its consequence, *incidental learning*. There is not much evidence on this, but Talland (1965) reported that his Korsakoff patients did not resume unsolved problems after distraction nor did they show incidental learning.

(2) Patients should not show spatial mapping abilities. Here we are referring to the locale space and not to egocentric space, which appears to be more dependent on parietal damage. One of the defining characteristics of Korsakoff's syndrome is disorientation in

time and space. In more formal tests, both Korsakoff and temporal lobe patients have severe difficulties in learning mazes. Interestingly, some of Talland's patients could learn a peg maze when the pegs had different colours, a finding reminiscent of the improvement in the performance of hippocampal rats when the cues are provided at the choice points.

(3) The hippocampus is the neural substrate for one-trial episodic memories in which events are related to each other in a long map extending from the past into the future. These episodic memories can be retrieved directly by the activation of the items themselves, or indirectly by events occurring before or after the event, or by the spatial context in which the event occurred. Without the mapping system there would still be considerable memory capacity. Short-term memory should be relatively intact. Long-term taxon memories would also be intact and could be retrieved directly or indirectly by category name or high frequency associates. The properties of this system would lead to a marked increase in interference amongst stored items.

There is a disagreement as to whether short-term memory is impaired in Korsakoff patients. For example, Baddeley and Warrington (1970) have reported intact short-term memory while Butters and Cermak (1974) have claimed there was a deficit.

With reference to long-term memory, we have concluded that a review of the literature shows that:

(a) there appears to be a selective loss in the storage of event memory;

(b) there is relatively normal storage of category memory up to and including categories based on the semantic feature of individual items;

(c) retrieval from these taxon category stores is considerably retarded in amnesics because of the powerful interference effects acting between items of a similar nature;

(d) the provision of retrieval aids at the time memories are being retrieved helps amnesics to gain access to the taxon information;

(e) the loss of event-specific memory seems to include memories formed before the onset of the disease or the surgical intervention as well as those formed subsequent to it.

Conclusions

It is our view that cognitive map theory effects a rapprochement between the infrahuman and clinical research, though there remain a number of places where the data are thin. Specific conclusions are scattered throughout *The hippocampus as a cognitive map* and can be summarized as follows:

(1) The hippocampus constructs and stores cognitive maps – representations which capture the spatial layout of an animal's experienced environment.

(2) These maps provide a basis for objective, nonegocentric, spatial cognition. Other brain systems are responsible for egocentric spaces, such as those referenced to body axes, or the eyes.

(3) Cognitive maps are useful for a variety of purposes, including successful food and water gathering; locating mates, territories, and safe havens; guiding navigation, migration, and so on.

(4) The known physiology and anatomy of the hippocampal system is consonant with the mapping notion. In particular, the existence of place-coded neurones and novelty-detectors in the hippocampus is important. The fact that hippocampal theta activity seems to relate to an animal's translocations in space provides a mechanism whereby distance information is made available to the mapping system.

(5) The consequences of damage to the hippocampus can be understood in terms of two direct effects, and several indirect ones. The direct effects involve the loss of novelty-detection/exploration and of place learning. Indirect effects, such as increased perseveration and susceptibility to interference, derive from the animal's forced dependence on alternative, often maladaptive, behavioural strategies.

(6) The spatial map/memory system also exists in humans, but the hippocampus in the language hemisphere is concerned with semantic rather than spatial mapping.

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by **Abram Amsel**
Department of Psychology, University of Texas, Austin, Tex. 78712

Hippocampus, memory and movement

A less than careful reading of this book may suggest to the reader that O & N are taking a strong stand on the old and tiresome issue of cognitive versus associationistic (particularly S-R) views of learning. Although they do slip into this posture now and then, it is important to understand that they mean to argue this point only in regard to the role of the hippocampus in learning. Their distinction between taxon and locale systems is crucial, and the strength of the book, from a behaviour-theoretic viewpoint, is that it makes this distinction clearly. Taxon systems are of two kinds, orientation and guidance. Orientation involves hypotheses such as "turn to the right," or "if you see your mother-in-law look 15° to the left." In the case of guidance systems the form is "go to the light," or "avoid your mother-in-law." Both these systems can function unabetted by cognitive maps and, on O & N's hypothesis, the hippocampus. Cognitive maps operate on the locale system: "search for what you want in your cognitive map and go to it."

The book at its best – at least the parts that concern me most – is about cognitive and noncognitive functioning, and its promise is that through a critical analysis of the literature, including their own experiments, the authors will present a convincing case for the hippocampus as the seat of the locale (cognitive maps) system. From my point of view as a learning theorist with some interest in brain function, the book can be faulted on two grounds. The first is that, whereas the authors rely heavily on experimental outcroppings of issues in learning theory from the 1930s and 1940s, they appear not to have as strong a grasp on the issues of this period as they need; they seem to rely too much on secondary sources in evaluating the status and meaning of the experimental evidence that arises out of the classical controversies in learning theory. For example, they examine the evidence from latent learning, continuity versus discontinuity in learning, and place versus response learning as though each of these issues bore equally (or at all) on the concept of cognitive maps.

In their argument about latent learning (p. 263) the authors accept *without citing a reference* the wisdom that normal laboratory rats have shown substantial amounts of latent learning; hippocampal animals are not supposed to do so. They then cite a couple of recent experiments showing no latent learning in hippocampal rats, but little if any in control rats either. They conclude that "a proper experiment" is needed that will not only fail to show the effect in hippocampals but will show it strongly in normals. A little digging into the relevant older work would have told them that the only strong latent learning ever found was in the original California experiments using complex mazes (and the magnitude of the California effect has failed to replicate on at least a couple of occasions). In simple choice situations there is often statistically significant latent learning, but the effect is never very strong. It is treacherous to build a theory on this kind of experimental quicksand, even if the presence or absence of the effect in question produces a good test of the theory – which it does not. Latent learning was about reinforcement as a principle of learning (Hull) as opposed to performance (Tolman). If latent or incidental learning can be demonstrated, this does prove that learning is possible without reinforcement; it does not prove that what is learned is a cognitive map; the learning may still be either of the taxon or the locale variety, or both.

The issue of continuity versus discontinuity in learning is not much, if any, better as a litmus test of the operation of locale versus taxon systems. Continuity in learning was at first an argument between Gestalt psychologists and associationists and the issue was the role of insight in learning. Later, it was a kind of family argument between two groups of S-R psychologists, Guthrians (formalized in the stimulus-

sampling theory of Estes) and Hullians. Here, the issue was the conceptualization of the growth of the learning curve. While Tolman favored both cognitive maps and discontinuity in learning (in the Gestalt fashion), there is no logic that requires such a map to be formed in a single trial. Nor does logic dictate that associations be formed incrementally. If continuity-discontinuity and taxon-locale are in any sense dimensions, they appear to me as orthogonal.

The place versus response literature would seem the major place to find empirical evidence on the basis of which to prove that the hippocampus is the organ of a locale rather than taxon system of functioning. Olton's work with his radial-arm maze seems a particularly promising source of such data [see Olton et al.: *BBS* 2(3) 1979]. If rats lose their ability to solve the radial-arm problem when they lose hippocampal function (and if they don't lose much else), and if they do not lose radial-arm-maze ability when they lose other equivalent amounts of brain, then O & N would seem to have scored some points for their side. The points are not yet on the board. More on this later.

The second major weakness of this book is that the evidence the authors need in order to make their case does not yet exist. O & N nevertheless undertake the task knowingly and fearlessly. They declare in their preface that they will not fool around with subtleties but will present "the boldest black-and-white case for the theory." The hippocampus seems a favorite target for black-and-white cases; and what it finally comes down to in almost all these cases is in one sense or another *memory and movement.* There are many conceptualizations of the relation of memory to movement. Among the older behaviour-theoretical ones are Hull's anticipatory-goal-response theory and Tolman's sign-significate relations theory (which does not, incidentally, *necessitate* cognitive maps). Among the more recent and more physiological views specifically related to the hippocampus are several that O & N quickly reject, such as Olds's (1972) position, which seems to combine Hullian and Tolmanian thinking with an emphasis on the latter; Gray's (1977) theory of septo-hippocampal function, which also combines elements of both classical learning-theory positions with an emphasis on the former; and the views of Altman et al. (1973), Douglas (1972) and Kimble (1969), which in one way or another find the hippocampus important for "braking," inhibition and suppression in the face of negative expectations. Clearly the hippocampus has a lot to do with anticipations, and particularly – or at least, this is the bulk of the evidence at hand – anticipations of negative events: punishments and frustrations. (When the authors cite studies that show no decrease in primary frustration in a double-alley as evidence that the hippocampus is not involved in frustration, they miss Gray's (1977) and Glazer's (1974) and my own point – that hippocampal function, and particularly some portion of the slow-wave (theta) activity, has to do with anticipatory frustration, with persistence and avoidance, not with the energizing effects of primary frustration, which experiments of Henke (1974) and others relate to the amygdala).

Of the recent explosion of learning experiments on hippocampal function, most of which are displayed in the tables of the authors' impressive appendix, not a small number involve passive avoidance ("don't go there," "don't do that") and DRL ("wait before you press the lever," "take more time," "do other things to take time"). Most of the relevant experiments on what I will call "paradoxial effects of reward" – the partial reinforcement and magnitude of reinforcement extinction effects, successive negative contrast, and patterned single alternation, as examples – are conducted in simple runways or lever boxes where taxon systems would seem to come into play more than cognitive maps would. All these experiments showing effects of hippocampal lesions can be accounted for by any of the extant theories of hippocampal function, except that the cognitive maps position would seem to have particular difficulty with the DRL results: Why should a map be involved in timing? Fewer experiments involve specifically the kind of arrangement in the solution of which a cognitive map would be required. Again, the Olton radial-arm maze is an exception, although here, too, it is easy to see how taxon rather than locale systems might be operating. The rat does not need a cognitive map to solve the Olton maze (and, incidentally, it does not solve the maze in anything like a single trial). The external-cue markers for each arm can trigger anticipations or *memories* leading to approach-avoidance movements. Or, in Tolman's language, sign-behavior-significate relationships can operate at each arm, independently. None of this requires the function of a cognitive map.

O & N took on a tough case when they decided to defend the proposition that the hippocampus mediates (in the positive form) "If you want X, find X in your cognitive map and go to X," rather than "If X is signalled and you want X go to X." A term from Scottish jurisprudence, appropriately enough, comes to mind: neither guilty nor not guilty but – "not proven."

by **Theodore W. Berger**

Department of Psychology and Department of Psychiatry, Western Psychiatric Institute and Clinic, University of Pittsburgh, Pittsburgh, Pa. 15260

Selective activation of hippocampal neurons

O & N have written a superbly comprehensive thesis concerning the role of the hippocampus in spatial mnemonic processes. It is supported both by lesion-behavior data indicating that rats with hippocampal damage show deficits in their ability to solve maze problems using a "place" (vs. a "cue") strategy, and by unit-recording data showing that hippocampal neurons are selectively activated when an animal is in a particular "place" on a maze. Different neurons respond to different places. It is on the latter unit-recording evidence that I wish to comment.

The authors report that hippocampal neurons respond selectively to a "complex" of extramaze visual cues which define extraorganism space. The cues are "complex" in the sense that a number of individual cues are necessary to sustain a cell's firing rate, and a given cell will respond equally well as long as some minimum is present, regardless of which specific cues make up that minimum (e.g., any three out of four stimuli). The argument is that the same space can be defined by several different subsets of cues, so that hippocampal cells meeting the above criteria are coding "space," not specific stimuli.

While this is an impressive piece of data, it falls somewhat short of demanding a "spatial coding" interpretation. Two things should be kept in mind: (1) the data demonstrate only that hippocampal cells can be activated by complex cues (i.e., that the cells code "spatial location" is an inference), and (2) that rats are food reinforced for entering maze arms during prerecording procedures. It becomes equally likely, then, that the data demonstrate conditioned hippocampal responsiveness to correlations between food reinforcers and multiple cues (only a subset of which need to be present to activate the cell). Through usual conditioning procedures, a given cell becomes responsive only to one complex cue composed of extramaze stimuli delimited by the organism's orientation in space (i.e., visual field). Given the amount of other evidence showing increases in hippocampal responsiveness during learning (Olds et al. 1972; Segal 1973; Best and Best 1976; Berger et al. 1976; Berger and Thompson 1978a,b), this alternative interpretation is certainly reasonable.

A second point concerns further evidence by O & N that hippocampal cells are responding specifically to "place." They discuss evidence showing that hippocampal neurons are activated when an animal is in a particular maze location – regardless of the animal's orientation within that location. For example, one cell may fire at a midway point on a maze arm when the animal is oriented either toward or away from the center of the maze arm. This type of evidence would seem to counter strongly the alternative interpretation outlined above, because the complex of multiple cues would be so different in each case. However, the reader must keep in mind that the data demonstrate only that hippocampal neurons increase their firing rate *in some manner* at a location on the maze arm. The authors have not demonstrated that the temporal distribution of increased unit spikes is the same in each case. In other words, a given cell may increase its discharge rate when oriented in either direction on a maze arm. The *pattern* of increased unit discharge, however, may be quite different when facing toward maze-center than away from maze-center. Alternative patterns could function to code (see Perkel and Bullock 1968) alternative sets of multiple, complex cues acting as conditioned stimuli. That the temporal distribution of conditioned cell firing can be a significant parameter

during learning has been shown by previous studies (see Berger and Thompson 1978a; Berger et al. 1979) using what O & N term the "neuropsychological" or "paradigmatic" approach to the study of brain-behavior relations. Differences in the pattern of cell discharge are difficult to document under the "neuroethological" conditions used by O & N, as this approach does not easily permit analysis of the temporal dimensions of brain-behavior phenomena.

A final point relates directly to the use and rationale of the "neuroethological" strategy for the study of brain-behavior relations. One of its major drawbacks has been mentioned above – its difficulty with fine-grained temporal analyses. A second drawback is its reliance on observational and subjective criteria for classifying brain-behavior correlations. The arbitrariness involved in this method of analysis can be seen in O & N's discussion of Ranck's (1973) and O'Keefe's (1976) studies of behavioral correlates of hippocampal neuronal activities. Both investigators used highly similar procedures in the same species. Ranck (1973) found that hippocampal activity correlated more with specific behaviors than with the spatial location in which the behaviors were emitted. O'Keefe (1976), on the other hand, reported that the firing of hippocampal cells correlated more with an animal's location in space than the particular behaviors emitted in that location. Confusion deepens on reading O & N, as it becomes apparent that increased firing of many hippocampal "place" cells depends on certain behaviors being performed in certain places, giving the impression that space-behavior "interactions" of some sort are the true determining variable for hippocampal cellular activation.

O & N have presented an impressive and interesting array of evidence that hippocampal cells are selectively activated by some variable – and that variable may be "place." There are a considerable number of alternative explanations for hippocampal cellular firing increases under their conditions, however, and a great deal separates the electrophysiological data from a comprehensive theory of spatial memory and cognition.

by **D. Caroline Blanchard and Robert J. Blanchard**

Department of Psychology, University of Hawaii, Honolulu, Hawaii 96822

Behavioral analysis of the hippocampal syndrome

The difficulties involved in attempts to understand behavioral changes associated with damage to specific brain sites or structures are seldom really appreciated. On the one hand are all the imponderables of brain anatomy and dynamics, on the other, the complexities of behavior. Despite (or perhaps because of) the radical changes in our views of brain constituents and their organization which have occurred in the past two decades, there is no question but that the brain side of this equation is better understood than the behavioral side.

The hippocampus is a dramatic illustration of this. In one sense, hippocampal damage ought to be comparatively easy to analyze. The behavior of the hippocampally damaged rat is so characteristic and consistent that the experienced observer cannot conceivably run "blind." The animal proclaims its status in its movements, before any choice point or aversive stimulus is encountered. However, this characteristic and consistent behavioral pattern has defied analysis by a veritable army of persistent researchers over a period of several decades.

Part of the problem is that many of the tasks and measures of experimental psychology are based on accident and analogy, rather than on a systematic analysis of the situations and population pressures under which animals have actually evolved. Another difficulty is that almost all behaviors of higher animals are mutiply determined: Variation in "passive avoidance," for example, may stem from so many sources that a change in any single passive avoidance task is basically uninterpretable.

Faced with these problems, most investigators tend to form some sort of fairly low-level and general hypothesis – that hippocampal damage interferes with perseveration or freezing, for example – and to test this hypothesis by demonstrating this deficit in a variety of tasks.

O & N's hypothesis that the hippocampus forms the neural substrate for the formation of spatial maps is clearly not one of these low-level notions. It is elegant and detailed, and has occasioned one of the most thorough and satisfying literature analyses ever to emerge from studies of a single brain structure. A reviewer usually likes everything except the part that he himself is most familiar with. In this case, we are most impressed by the O & N treatment of areas with which we are very familiar: exploration (Ch. 6) and aversively motivated behavior (Ch. 8). These chapters are outstanding in their scholarly and analytic treatment of behavior, quite aside from possible insights into hippocampal functioning.

Faced with such splendor, it seems pretty churlish to remain skeptical. We are, though. There are just too many points at which the spatial-map hypothesis alone cannot account for behavioral changes accompanying hippocampal damage.

For example, in a recent series of experiments (Blanchard et al. 1977) we found that normal rats show a fairly systematic pattern of grid grasping to minimize the pain of shock. Hippocampally damaged rats grasp the grids in similar fashion, but hold this grasp and its associated immobile posture only about half as long as normals. Similarly, these rats perform more poorly than normals when forced to balance on a narrow ledge: they fidget around and fall off. When placed on a larger platform in which active immobility is not required for balance, or on a wire in which active rather than immobile balancing is typical, hippocampal rats were virtually identical to controls.

There is no reason to suppose that spatial mapping has anything much to do with performance in these tasks. Since the apparatus items were on the same table and about the same distance above the table, there is also no reason to suppose that spatial maps would be different in the three tests even if such maps were involved in performance on the tests. Yet hippocampal damage produced a clear deficit in one test and no difference in the others. Moreover, the ledge test appeared to rely on exactly the same sort of active immobility which hippocampal rats seem incapable of maintaining in so many other situations. If spatial mapping problems were not a factor in the ledge-platform-wire test comparisons, it seems unparsimonious to interpret the acknowledged difficulties of these animals in freezing in aversive conditioning tasks as a reflection of loss of spatial mapping ability.

We are not seriously proposing that an inability to "hold still" can account for all the differences between normal rats and rats with hippocampal lesions. In fact, this particular deficit is not a function of damage to the hippocampus proper. In an as-yet-unpublished study, Haakon Sundberg and ourselves found that exactly the same motor pattern is found in rats with lesions restricted to the entorhinal cortex. However, this deficit is also seen with hippocampal damage, and it may have a profound effect on attention to specific objects as well as on the formation of "maps," since such attention is obviously impaired by a deficit in active immobility.

Regardless of one's specific perspective, this book has performed an invaluable service in bringing together the scattered hippocampal literature. It also sets a new standard for critical and scholarly analysis of hypotheses, and will lead to a more sophisticated experimental approach to behavior changes following brain manipulations.

by **T. V. P. Bliss**

Division of Neurophysiology and Neuropharmacology, National Institute for Medical Research, London NW7 1AA, England

O'Keefe & Nadel's three-stage model for hippocampal representation of space

I have given my general impressions of this remarkable book elsewhere (*Nature,* in press). Here I want to consider some of the problems attending the authors' neural model of how the cognitive map is generated.

From a physiological point of view, the main evidence for O & N's theory is that there exist place units in the hippocampus. It is not clear whether similar place units exist elsewhere in the brain – obviously, the theory would be seriously weakened if they did. Assuming that such cells are peculiar to the hippocampus, one can ask what special features of the anatomy and physiology of hippocampal neurons allow the construction of an enduring neural representation of space. This is

the problem which O & N tackle, albeit with uncharacteristic brevity, at the end of Chapter 4. Their model contains three stages. In the first, sensory information from stimuli in the environment is assumed to converge on granule cells of the dentate gyrus via perforant path fibres originating in entorhinal cortex. The details of what goes on in the dentate are not critical to the model's primary job of endowing pyramidal cells with the observed properties of place units; in fact the model would work (insofar as it does work) if the entorhinal cortex projected to CA3 pyramidal cells directly. This having been said, the system of rigidly wired collateral connections favoured by the authors (Fig. 28) is highly implausible, making huge demands on developmental mechanisms, and is in any case unnecessary. A system of random connections within the constraints of an overall lamellar projection would also serve the required purpose of yielding a population of granule cells responding only to complex configurations of stimuli.

In the second stage of the model, the axons (mossy fibres) of granule cells project, via en passant synapses, to CA3 pyramidal cells. For economy it would clearly be desirable to restrict the number of pyramidal cells responding to activity in a given mossy fibre, and to achieve this the authors ingeniously exploit the fact that theta waves travel across the hippocampus during voluntary movement, imposing rhythmical changes in the excitability of pyramidal cells. Consequently, at a given moment in the exploration of an unfamiliar environment, the mossy fibres carrying information about environmental stimuli will find only a proportion of CA3 pyramids, among the total receiving such activity, in the appropriate phase of theta to fire. At this point it will help if I give a simplified example of how, as I understand it, the model operates.

Suppose the controlled environment contains three stimuli, A, B, and C (light at 10 o'clock, buzzer at 2 o'clock, cage at 6 o'clock, for example). In the authors' model of the dentate, each pair of stimuli activates a specific granule cell; let us say that AB, AC, and BC fire granule cells G1, G2, and G3 respectively. The three granule cells in turn fire those three pyramidal cells, C1, C2, and C3, which the theta gating mechanism selects from the population of target cells of G1, G2, and G3. Note that at this stage, during initial exploration of an unfamiliar environment, the pyramidal cells have the same fields as the corresponding granule cells; in particular, if any one of the three stimuli defining the animal's place in its environment is removed, only one granule cell and one pyramidal cell will remain active. In order to transform the three pyramidal cells into true place units with the property of continuing to respond in the absence of any one of the three stimuli, the three pyramidal cells must become linked together so that if one fires, all fire. Once this essential requirement is made explicit, it can be seen to be identical to the "collateral effect" proposed by Marr (1971) in his model of the hippocampus as a simple memorizing device, a paper which, unaccountably, is not mentioned by O & N. There are two immediate consequences of this requirement. First, the anatomical substrate must exist; probably the longitudinal associational pathway would serve in this respect. Second, there must be the appropriate type of modifiable synapse in the collateral/associational fibres. It seems inescapable that Hebb synapses are required; only thus can connections between those cells, and only those cells, which have fired together be strengthened. Here we touch on a major weakness of O & N's treatment – their failure to exploit or even adequately describe the one property of hippocampal synapses which on the face of it uniquely suits them for use in a memory device, and that is their ability to sustain long-term potentiation of synaptic efficacy, of a duration and magnitude found up to now nowhere else in the brain. There is the difficulty, however, that in the several hippocampal pathways known to support long-term potentiation, the evidence suggests (if not quite conclusively) that activity in the afferent pathway alone is sufficient to induce potentiation. In other words, these are probably not Hebb synapses.

The third stage of the model – located in CA1 – deals with two other types of unit, assumed to be driven by inputs (presumably Schaffer collaterals) from the assembly of interconnected place units. The first of these is a more complex type of place unit with the property that its place field widens progressively as the stimuli defining place in the controlled environment are withdrawn (this is in contrast to the simpler type of place unit in CA3, whose place field remains constant until the cell ceases to respond at all); the second type is the misplace unit, which fires when an expected stimulus is absent from a familiar environment. It is not a difficult matter – though the authors, like Bishop Berkeley in an earlier context, "excuse themselves on this one" – to construct neural models to mimic these behaviours, using direct axo-axonic inhibition for the former, and sensory afferents from entorhinal cortex converging via inhibitory interneurones on CA1 pyramids for the latter. But such models have little in common with conventional interpretations of hippocampal anatomy. Altogether, this last stage is the least satisfactory part of the model.

Two final points. The cognitive map is represented by sets of pyramidal cells the composition of which depends on (1) the phase of theta activity which existed at the start of exploration, and (2) the route followed during exploration. The map should be independent of both. The first problem could be overcome by assigning modifiable (preferably Hebb) synapses to mossy fibres. The second is less trivial and I see no simple solution, though a solution certainly exists as places can be recognized when approached by a route different from that followed during initial exploration.

by **Jan Bureš**

Institute of Physiology, Czechoslovak Academy of Sciences, 142 20 Prague 4 - Krč, Czechoslovakia

The "neuroethological revolution" in unit studies

In their remarkable book O & N have gathered an impressive amount of evidence supporting their view that the hippocampus is the brain structure responsible for cognitive spatial mapping. This important proposition is substantiated by philosophical, psychological, physiological and clinical arguments and espoused in an eloquent, essayistic manner which shows the authors' scientific and emotional commitment to this idea. Although sceptical readers will probably still have doubts about cognitive maps in general, and their hippocampal implementation in particular, the firm position of the authors is an important landmark in hippocampal research, which will certainly stimulate thinking and experiments in this direction. Not being a hippocampus specialist, I shall limit my comments to a methodological issue which seems to be crucial for the electrophysiological evidence for cognitive mapping.

O & N ascribe great significance to the observation that some hippocampal neurons fire more frequently when the animal is in a particular place of the experimental environment, while the activity of others is independent of the animal's location. This is, of course, a reasonable classification in view of the authors' cognitive mapping hypothesis. One would expect the authors to use conventional analytical methods to prove this point. Surprisingly, this is not the case. The discussion of these experiments (pp. 190–217) is introduced by a somewhat ironic criticism of the "neuropsychological" approach to behavioral unit studies. The authors decry the experimental paradigms which examine the incidence of various types of unit responses in a nerve center during performance of a particular task.

> "Thus, we learn that in nucleus X 31 per cent of the units responded only to the 2 KHz tone, 19 per cent to the flashing light, 12 per cent to both, and 38 per cent to neither. This may be like concluding that there is a remarkable uniformity amongst computers, caryatids, chrysanthemums, and coprophagists, since they are all polysyllabic, begin with a hard C sound, and denote entities which fall at the same rate in a vacuum" (p. 191).

They feel that the neuropsychological approach does not ask the correct questions and that its progress is too slow because of too much concern for unimportant details. They suggest that "during the exploratory phases of research into the function of a structure it is necessary to use a more information-rich methodology, the neuroethological one" (p. 192).

While one may agree that progress of the "neuropsychological" unit studies is not very fast, the superiority of the information-rich approach proposed by O & N is questionable. The basic principles of the neuroethological methodology are quite simple. 1. Unit activity should

be studied in "as naturalistic a setting as possible," preferably "in the animal's natural habitat" (p. 194). 2. Each unit should be studied as an individual with the aim "to describe the full range of its behavior in as many different situations as possible" (p. 194).

Anyone familiar with unit activity recording in freely moving animals and with the analysis of such records will be impressed by the immensity of the task implied by the above principles. It is difficult enough to distinguish unit responses to a well-defined isolated stimulus from nonspecific activity changes due to interfering stimuli, homeostatic disturbances, or the simple nonstationary nature of the spike generating process. With an unrestricted stimulus field and unlimited behavior on the part of the animal it would be almost impossible to define the independent variable (a combination of cutaneous, proprioceptive, vestibular, auditory, visual, olfactory, and visceral signals) and to correlate it with the unit activity. Rigorous correlation of recorded behavior (frame by frame photography, telerecording) and electrical activity might be one solution but would require developing new analytical methods. In all such studies unit reactions must satisfy certain statistical criteria, taking into account the signal-to-noise ratio, variability, trends, and so on. The authors' position in this respect is peculiar: In the footnote on page 195 they refuse to apply statistical methods to their findings and claim that the reactions studied are so clear that they do not "require averaging or other computer manipulation for their demonstration."

Such an attitude contrasts with the efforts of a generation of referees who have insisted on statistical documentation of even the most obvious findings. It is also an anachronism in the computer age which provides easily accessible means for statistical analysis of the type of data obtained by O & N. If the hippocampal responses are so clearcut, it should be easy to demonstrate their existence by appropriate statistical methods. Statistics would serve in this case as an objective evaluation of the data, providing an accurately defined acceptance threshold which could be applied by anyone who wished to replicate the experiments. Without specifying statistical criteria, unit classification becomes a voluntaristic affair, a matter of art or faith rather than of science. Without elementary quantitative treatment it is not clear how many times the rat returned to location A before a given unit was classified as a place unit. What was the distribution of unit responses during these returns? Were there no signs of habituation during repeated returns to the same location? etc. etc.

Do results of the "neuroethological" analysis of hippocampal unit activity stand up to the authors' expectations? The reader is surprised to find out that the yield of the information-rich approach is rather meager. Only two classes of units were distinguished in the hippocampus: the place units (with the subclass of misplace units) and displace units. The impugned "neuropsychological approach" testing the preordained hypothesis that the hippocampal units are affected by the animal's location in the environment would obviously not fare worse. On the contrary, it would also provide data about the relative incidence of these classes (information considered unimportant or even misleading by the authors) and would allow a systematic, preferably parametric, examination of space representation in such units. An obvious "neuropsychological" paradigm would be to place the animal in a transparent transport cage and to move it repeatedly from one location to the other until computer analysis of unit activity revealed (or failed to reveal) the predicted changes of firing rate. If active rather than passive movement is required, the rat can be trained in a closed (e.g., circular or rectangular) runway and the unit activity can be correlated with the animal's location in various parts of the apparatus. There are hundreds of similar experiments which can test the spatial hypothesis step by step using the classical theory of experimental planning, changing one parameter at a time, or perhaps a few parameters simultaneously, piecing together a coherent picture of the putative mechanisms underlying the cognitive mapping hypothesis.

The unit studies by O & N are an important contribution to the paradigms for electrophysiological investigation of behavior. The authors pioneer the use of the animal's location in space as a complex stimulus and develop techniques permitting electrophysiological testing of their hypothesis. Their arguments for hippocampal mapping should be more affirmative, however, leaning on intuitive demonstration of the predicted unit activity changes by conventional methods rather than on discursive analysis of the mistakes made by others.

by **Robert J. Douglas**

Psychology Department, University of Washington, Seattle, Wash. 98105

The hippocampus and its apparent migration to the parietal lobe

In presenting every scrap of evidence, every conceivable twist of interpretation that can be marshalled to support their theory, O & N have produced what may be the best brain and behavior book yet written. The naive reader might not realize, however, that the authors employ an adversary technique that leaves no room for the "other side of the story." That is, there are selective omissions of evidence and theories that might weaken the authors' arguments. The reader might also fail to realize that the evidence can be interpreted in other ways, and that it would be well to look into these before jumping on the spatial bandwagon.

One example of selective omission is the failure to mention studies showing the crucial importance of the vestibular system ("inertial navigation") to the locale system (e.g., Douglas 1966a,b; Beritoff 1965; Barlow 1970; Douglas et al. 1974). A probable reason is that the authors want the reader to see an important connection between "place units" in the hippocampus and the behavior of hippocampectomized rats. Place units fire in response to a pattern formed by two or three visual extramaze or environmental cues. Clearly it would be "neat" if hippocampal lesions had a particularly bad effect on behavior based on such cues. Alas, some of the behavior most vulnerable to hippocampal damage has been intensively studied and found to be *un*related to visual or other extramaze cues. This has been shown to be true, for example, of both spontaneous alternation (Douglas 1966a) and complex maze learning (Watson 1901).

Watson trained rats in a complex spatial maze and found his rats to behave as if they had never learned the problem when he rotated the maze by 90° in the room. O & N cite this observation in support of their idea that maze learning is based on visual extramaze cues. They neglect to tell us that Watson also dragged the maze to the far end of a long room, thereby completely changing the "scenery" as viewed by the rat. This had no effect whatever on maze performance. We are also not informed that Watson found similar behavior in normal and in blinded rats. These results, and many others of a like nature, fall somewhat short of proving that visual extramaze cues are crucially important in maze learning.

The reader is also not informed about the behavior of people with damage to the vestibular system or such vestibular-related regions as cerebellum, striatum, or parietal lobe. As Barlow (1970) describes, these patients are perfectly able to orient egocentrically to stimuli that are currently in view or in the immediate vicinity. But they cannot grasp the spatial relations between different "scenes." They routinely get lost going to or from the bathroom, for example, but immediately recognize the place they want when they stumble upon it. The defect sounds like that imagined by the authors to occur in hippocampectomized rats. The authors cite but do not discuss the work of Semmes et al. (1955, 1963) on map-following in people with parietal damage. The subjects in these studies had no apparent defect in egocentric orientation but were unable to follow either visual or touch maps. A map-following defect has also been reported in people with Parkinsonism (and thus presumed striatal damage; Bowen et al. 1972). To be sure, parietal lesions do sometimes produce a (usually transitory) defect in egocentric orientation, but a conclusion that parietal lesions produce only egocentric deficits is not, in my opinion, warranted by the evidence.

The book contains many instances of highly strained explanations for the defects of hippocampally damaged rats in various tasks. Rather than listing them, however, I will go to the heart of the problem. That is, there are numerous examples where hippocampal lesions produce the typical "hippocampal syndrome" deficit without spatial abilities or the locale system being involved in any discernible way. For purposes of argument let us assume that the taxon and locale systems exist and that hippocampal lesions abolish the latter but not the former. It would

then seem to follow that when normal and hippocampectomized rats are both forced to employ the taxon system, the lesioned animals would not be expected to have a deficit. Indeed, one could argue to the contrary that it would be the normal rats that might be defective if they persisted in trying to use the irrelevant locale system. A perfect example of such a situation is the reversal of a black-white discrimination problem. This cannot be solved by the locale system because a given alley is sometimes white and sometimes black.

It is therefore not surprising that hippocampal lesions generally have no effect on the rate of original learning, but it is most difficult to explain why the lesioned animals should be so defective in learning the reversal. The explanation is that the hippocampals have difficulty in suppressing an irrelevant turning habit. Since the normals are also using the taxon system, there seems to be no logical reason why they should not have the same difficulty. Indeed, one suspects that the authors could have more comfortably explained an improvement in reversal learning after hippocampal lesions as being due to the normals having one more erroneous hypothesis to test and eliminate.

The Olton group has repeatedly found that hippocampal system damage prevents rats from learning to avoid reentering the same location during a radial maze session. The defect, attributed to a loss of spatial memory, has been viewed as powerful support for the spatial model. Recently, however, Olton et al. (1979) have found similar results in a completely nonspatial version of this task, in which rats were trained to avoid specific alleys that were spatially interchanged. Thus, the radial maze literature, once a cornerstone of the spatial model, has become a stumbling block.

In most complex mazes the animal can "guess" which way to turn at many choice points by knowing the location or direction of the goal, and it has been proven that rats do indeed employ such information. The spatial model is supported by the fact that rats with hippocampal lesions are defective in learning such spatial mazes. The problem, however, is that the lesion appears to produce an even greater deficit in nonspatial linear mazes (Winocur and Breckenridge 1973). In linear mazes there are no choice points at which the correct answer can be guessed by knowing goal location or direction. O & N correctly attribute the defect to an inability to suppress interference (a trait of the taxon system). But the normals are also presumably using this system, and they also display behavior indicative of interference. The normals, however, manage to suppress interference more quickly.

We are repeatedly informed that the dull-witted taxon system is unable to overcome, suppress, block, withhold, and so forth. On the other hand, possession of the locale system and the hippocampus allows the animal to do these things. This could be mistaken for an inexplicit version of an inhibition idea. Inhibition also arrives by the back door in the convoluted discussion of exploration which, we are told, does not occur in hippocampectomized subjects. The authors grant the obvious, that the lesioned animals run around and sniff and look as if they were exploring. This is, however, not "really" exploration because the activity does not decline over time. Thus, the lack of an inhibitory process, habituation, is misleadingly described as a lack of the thing that is inhibited. One suspects that the authors are trying to make "misplace units" appear to be highly relevant. In Pavlovian terms these would be called "disinhibition units," since they fire in response to stimulus change. But in most studies of exploration (activity) the rat is plopped down in an open field that remains unchanged.

Finally, the authors attempt to cope with the verbal memory deficit in hippocampally damaged humans by postulating a spatial origin for the deep semantic structure of language. This is truly a stroke of genius even if the defect does not appear to be a semantic one. Milner's bilaterally hippocampectomized patient H.M. manages to converse normally and has great difficulty in remembering nonsense syllables. Further, one could also argue for the ultimate spatial origin of the temporal sense and make virtually all mental processes "spatial." The problem is that the color red becomes meaningless in an all-red universe.

In summary, there is abundant evidence (barely touched upon here) that hippocampal lesions produce the same sort of deficit in spatial and nonspatial tasks. There are also many cases where the lesioned animals appear to have learned spatial locations, though the authors claim that in such instances the animals are "really" learning something else. They fairly point out, however, that in most cases this remains to be experimentally verified. The authors claim that theta waves are crucial to the locale system, and yet Winson (1978) abolished theta waves by medial septal lesions and found this to have no significant effect on the learning of a task expressly designed to demonstrate spatial memory. There are alternative theories that can also ingeniously explain many facts and that do not make a fundamental distinction between spatial and nonspatial stimuli (e.g., Olton's "working memory," Warrington's "recognition," and Weiskrantz's "interference" ideas). The internal inhibition model, not to be confused with "response" inhibition, can also account for the anatomy, physiology, and pharmacology of the hippocampus, as well as for the lesion data (Kimble 1968, Douglas 1967, 1972, 1975). The problem for the reader is that these alternative models have not yet been elaborated in the form of well-written 500-page books. O & N are hardly to blame for this. They have done their job superbly, leaving it for others to do theirs.

I believe that there is considerable merit to the O & N model when employed on a less ambitious scale. Rats are barely removed from the nocturnal common ancestor of placental mammals. The hippocampus evolved into its present form in the brains of primitive mammals that investigated their environment by locomotion and direct contact. Of necessity they developed movement-related spatial maps, and it is probably no coincidence that hippocampal theta waves are usually associated with movements in space in rats but with attention in primates. One would predict that movement-related theta would also occur prominently in shrews and other primitive insectivores. Hippocampal mechanisms were probably first elaborated or expressed in a geographic or absolute spatial context. Many other abilities may well have evolved from the primordial spatial sense, dragging the hippocampus with them, so to speak. But there comes a point at which the descendant becomes so divergent from the ancestor as to warrant separate classification. A comprehensive model would thus ideally postulate underlying functions that can be expressed indifferently in spatial or nonspatial contexts.

by **Roger M. Downs**

Department of Geography, Pennsylvania State University, University Park, Pa. 16802

On the nature of cognitive maps

In the preface to their book, O & N liken theories to maps and the construction of theory to a voyage of discovery. These are apt metaphors. I derive as much pleasure from reading this book as I do from poring over a map. The likeness between this book and, say, a Portuguese or Dutch *mappa mundi* is uncanny. The clear division between the known world and the *terrae incognitae,* the sea monsters and sirens that lurk and beckon from beyond the safety of the known world, the unparalleled draftmanship, the understated sense of design, the rococo flourishes of the elaborate cartouche; all of these find their counterparts in *The hippocampus as a cognitive map.* But the book is more than just a source of pleasure; it offers the fascination of a global view of a landscape that had previously been glimpsed only in fragments. O & N speak of assembling "pieces and patches," of "synthesizing the facts"; one can only applaud the end result.

As a piece of scholarship, it is a *tour de force.* This is not a book for the fainthearted who like a muted, homogenized discourse, well-hedged with evasive qualifications. Instead, it presents a panoramic survey, the results of which are stated boldly and persuasively. As a participant in a year-long interdisciplinary seminar on space, I can appreciate the achievement of the first chapter, which disentangles the philosophical conceptions of space and which renders coherent the inchoate psychological conceptions. It is reassuring to read a literature review which is both critical and constructive. Overshadowing all of these strengths is the feeling that someone is trying to say something important, that O & N have caught hold of some of the evanescent ideas that have been "in the air" of late. In the process, they have forced us to take stock of some of our less-articulated beliefs. Let me

try to take stock from the point of view of a geographer interested in cognitive mapping.

A key point is O & N's attempt to find *a* physiological-anatomical locus for the cognitive map. I say *a* locus because there are significant implications that follow from the search for *any* locus: these implications transcend the choice of the hippocampus, a particular choice which is beyond my competence to discuss.

Within the group of environmental psychologists and geographers, there has been a tacit agreement not to ask, Where is a cognitive map? We have devoted attention to how cognitive mapping takes place, what a cognitive map looks like, and why it exists. The functionalist answer to this last question has taken us perilously close to the "where" question, but apart from a few tentative forays, we have remained content to view the cognitive map as a hypothetical construct, a classic form of intervening variable. There were what seemed to be good reasons for this position: a lack of expertise in physiology, a dualist approach to the mind-body problem, some muttered asides about reductionism. All of these served to hide what is an obvious question.

Where do we stand now that O & N have destroyed the convenient fiction that we do not need to ask about the locus of cognitive maps? That we had unnecessarily restricted ourselves is clear from the richness of O & N's argument. That we are in a position to consider some penetrating questions is equally clear from a discussion of but one of the issues that emerges from even a limited reading of *The hippocampus as a cognitive map.*

We must return to the perennial problem of the meaning of the term, map. I agree with O & N that Tolman was using map as a metaphor, in exactly the same way as he used telephone switchboard. O & N state that the locale system is analogous to a map of physical space. But even they seem hesitant in places – how are we to interpret their uses of "map-like representations" and "map-like structures?" At least they have more courage than many of the writers in the avian migration and homing literature who are quite happy to write about a compass and yet insist on cloaking map in quotation marks.

The status of map depends upon a characterization of the nature of the mapping process. From a cartographic viewpoint, a mapping is the transformation of an object set (physical space) into an image set (the cognitive map) via a function. Of vital concern are the set definitions and the properties of mapping function. The set definition establishes relations (distance, direction, etc.) between places; the mapping function specifies which relations will be maintained or preserved in the image set.

O & N offer a physiological interpretation of this process which suggests that the mapping function is Euclidean and three-dimensional. In this way, neural space is directly analogous to physical space. It is true that cartographers treat physical space as being locally Euclidean, arguing that the differential curvature of the two surfaces (the earth and the sheet of paper) is irrelevant at the large scale. The current model achieves that same effect by suggesting a three-dimensional character. But nowhere do O & N show why the image space must be (or is) Euclidean and three-dimensional. I suspect that it is – but the nature of the mapping function is unspecified. Without question, it is a homomorphic transformation: but how is this generalization achieved? It will have rules for scaling distance relations: but how does it transform distance-as-effort, which is non-linearly related to physical distance, into a Euclidean metric? Moreover, since all maps are models, judgments which relate neural and physical space using criteria such as veridicality, accuracy, and distortion must be made with care. We can only know (map) physical space as a model, albeit a model in which we have greater confidence.

There is an unresolved question about the number of maps of physical space that are contained within the hippocampus. One possible interpretation of the physiological model is that it is literally a microcosm and that any map is a "slice" through the microcosm. If this is not the case, then what does it mean to say that the system contains a map for each environment experienced? The idea of a mental atlas poses problems. How is the appropriate map selected? Can places appear in two or more maps? Is change of scale made possible by a series of maps or by some "enlarger-reducer" mechamism?

In focusing on the nature of maps, I have not left myself space to list all of the points at which I find myself in agreement with O & N. Their constant concern with the relation between research design strategies and the role of cognitive mapping as a flexible, hypothesis-generating function is worth pursuing. And so is their separation of the taxon and the locale system: this strikes at the heart of the conventional belief that route mapping and spatial mapping are but stages in a single learning process.

Given my interests, this is the most provocative book that I have read in years. Given my geographical background, a discipline whose "language" is said to be that of the map, it does wonders to read the casual thought buried in a footnote to page 401. That language could have developed as a means of transfering information about the spatial aspects of the environment is as refreshing a thought as the book is as a whole.

***by* Paul Ellen**

Department of Psychology, Georgia State University, Atlanta, Ga. 30303

The hippocampus and operant behavior

O & N's book comes as a welcome relief to those of us working in the area of brain-behavior interactions. The last few years have seen a spate of symposium proceedings and collections having to do with the functional significance of one or other of the various structures comprising the limbic system. In all these volumes there are contributions describing anatomical, electrophysiological, or behavioral findings, or some interdisciplinary combination of the three. Nowhere in them, however, does one find a systematic attempt to integrate the various reports. As a result, the reader is generally left with a hodgepodge of unrelated findings, many bearing only tangentially upon the purported thrust of the symposium. As a result our undertanding of the functional role of various brain structures is generally not advanced, and the most that could be claimed for such endeavors is that they provided the various contributors to the book with an opportunity to do some travelling. With the publication of the O & N book, we have for the first time a coherent attempt to relate the anatomical, physiological, and behavioral data derived from studies of the hippocampus into a comprehensive, integrated theoretical framework.

O & N, in a bold stroke, assert that the hippocampus is the cognitive or spatial mapping system of the brain. Although the concept of a cognitive mapping mechanism is not new, having originally been suggested by E. C. Tolman (1948) more than thirty years ago, it remained for O & N to breathe new life into it. They have accomplished this by linking the concept of a cognitive map to two different domains of data. By doing so, they were able to give the concept of a cognitive map a genuine theoretical status. On the one hand, the concept was linked to a variety of data at the behavioral level, while on the other, it was linked to findings on the anatomical and physiological level. Tolman's use of the concept was ill defined since he succeeded only in linking it to behavioral findings. However, the dual linkage to data in different empirical domains makes the notion of a cognitive mapping mechanism theoretically significant, since it allows it to be something other than merely a circular response-inferred construct which is used to explain the very same data from which it is inferred. For once the concept of a cognitive map is tied to more than one set of empirical referents, giving it more properties than the very data that it relates to. O & N have elegantly demonstrated that theoretical development is still possible within psychology and that one does not need to engage in naive reductionism to theorize about brain-behavior relations.

From my perspective as a psychologist, the chapters dealing with the history of concepts of spatiality, and the review of the behavioral effects of hippocampal lesions (chapters 5–11) were the most fascinating and rewarding. In the lesion review, O & N make a valiant attempt to subsume the myriad findings following hippocampal lesions into their theory that the hippocampus functions as a substrate of a cognitive mapping mechanism which enables animals not only to

localize themselves in space but also to find their way from any place in the environment to a goal location. In the absence of hippocampus, animals are forced to rely on hypotheses derived from taxon systems such as "go to the light" or "turn left or right." The success of these guidance or orientation hypotheses would depend upon the requirements of the particular task. In contrast, normal animals can rely on locale systems which give rise to place hypotheses and maps of the environment. Such maps enable animals to choose the shorter of two routes to food or to short-circuit many of the alleys in a maze. In general, O & N are successful in applying their theory to the lesion literature. Clearly, the findings of deficits following hippocampal lesions on various maze and discrimination tasks are consistent with expectations derived from the theory. Particularly ingenious is their explanation for the facilitated avoidance behavior of hippocampal animals in a shuttle avoidance task. Since place hypotheses are abolished by the lesion, the hippocampal animal is not confronted with conficting place hypotheses and hence the two-way avoidance task is not particularly difficult for it. For the normal animal, in contrast, running to a place which was previously identified with danger is particularly difficult.

O & N begin to strain their theory when they attempt to account for results of hippocampal lesions on operant behaviors. In operant situations animals are confined to a relatively small chamber, so it is difficult to entertain the notion that place hypotheses are important mediating factors in the bar-pressing performance. Of particular interest, too, is the fact that in the hippocampal literature, the results of the behavioral effects of hippocampal lesions on operant tasks have been the most controversial. For example, while Isaacson and his associates (Clark & Isaacson 1965; Schmaltz & Isaacson 1966a,b, 1968) have uniformly reported an overresponding by hippocampal animals on DRL (differential reinforcement of low rates) schedules, Ellen and his associates (1964, 1970, 1973) have shown that such an impairment actually requires the confluence of two separate factors to produce the effect. Not only must there be large hippocampal lesions, but also there must be a history of extensive CRF (continuous reinforcement) pretraining. O & N argue that the overresponding results from the failure of the hippocampal animals to avoid the *place* where the lever is and a consequent failure to engage in collateral behaviors which could mediate the required delay on the schedule. The assumption underlying this argument is that since responses to the lever sometimes lead to reinforcement and sometimes not, the animal is in a conflict situation. The normal animal has more hypotheses (place) than simply guidance ones available and can utilize these in forming collateral chains which mediate the required delay. The hippocampal animal in contrast can only resort to taxon-type hypotheses and these tend to keep it close to the lever. This treatment of the hippocampal effects on DRL behavior suffers when confronted with the fact that it isn't merely the fact of the hippocampal lesion *per se* which produces the overresponding, but rather there must also be a prior history of CRF pretraining. The fact that a hippocampal lesion by itself is insufficient to produce the behavioral deficit in the task seriously limits the generality of the O & N model with respect to this class of behavior. What this means is that response rate on DRL schedules is not reflective of the hypotheses generated by either the taxon or locale systems.

Perhaps even more important in this context is the fact that extensive CRF pretraining is a necessary condition for the behavioral effects of the hippocampal lesion to be manifest. O & N attribute this to a simple strengthening of an orientation hypothesis mediating lever pressing which occurs to a greater degree in hippocampal animals than in normals. Thus, this finding would mean that the difficulty in giving up the previously acquired CRF response pattern would simply be a reflection of the operation of the taxon system rather than the locale system and hence not indicative of any other impairment. This kind of argument reflects a fundamental problem with the book. Throughout, O & N assume that hippocampus is a unitary structure "in the sense that it has an integrated function" (p. 231). Thus, all other possible functions of hippocampus are subsumed under or considered to be subsets of the basic cognitive mapping function. This tactic was deliberately chosen by the authors; yet one questions its wisdom. Will it not set the stage for interminable controversy as to whether hippocampus has one or many functions? Will it not provide the bases for numerous attempts by others to demonstrate that hippocampus indeed has more functions than that simply subsumed under cognitive mapping? In this sense, it may be that O & N have inadvertently set up a straw man which can only function to deflect more serious evaluative attempts at assessing their theory.

Finally, I should like to comment on one other aspect of the theory as presented. O & N emphasize the role of locomotor behavior in the generation of a cognitive map. Exploratory behavior results when there is a mismatch between the representations in the map and the environment as perceived by the animal. Exploratory behavior provides new inputs to the map, which is then updated to conform to the perceived environment. Movement-generated theta rhythm sorts the various sensory inputs to the hippocampus according to distances between places in space. A major role is thus imputed to locomotory behavior in the mapping of space. O & N cite extensive literature indicating a decrease in exploratory behavior subsequent to hippocampal damage. Locomotor exploratory behavior is considered to be a fundamental information-processing mechanism whereby the animal acquires spatial information. To what extent can an animal learn about places in the absence of locomotion? To the extent that cognitive map formation is possible in the absence of locomotion, a serious blow would have been dealt to the mechanism postulated by O & N to account for how the cognitive map gets formed. In particular, the significance of the theta rhythm in the process would be severely weakened. Early work by Gleitman (1955) and McNamara, Long, and Wike (1956) demonstrated that place learning was possible in the absence of locomotion. Merely transporting animals over paths in little cars without allowing locomotory activity allowed animals to learn either the locus of food or the locus of shock termination. Similarly, Beritoff (1965) and Ungher and Sirian (1971) showed that blindfolded animals could learn the spatial locus of food simply by being passively transported to the food place. In short, cognitive maps could be formed in the absence of exploratory behavior. To the extent that locomotion does not occur, it can hardly be alleged that movement-induced theta is performing the function of representing spatial distances that O & N attribute to it.

Despite these comments, my reaction to *The hippocampus as a cognitive map* is still one of unabashed admiration. It is a scholarly achievement the likes of which we have not seen in years in this field. It will be a source of new ideas, constant stimulation, and fruitful inquiry for years to come.

by **Jeffrey A. Gray**

Department of Experimental Psychology, University of Oxford, Oxford OX1 3UD, England[1]

Spatial mapping only a special case of hippocampal function

When O'Keefe and Dostrovsky (1971) first proposed the hypothesis that the hippocampus might act as a spatial mapping system, it was based on observations of the behavior of a handful of hippocampal neurons from which they had recorded in the freely moving rat. If the hypothesis proves to be correct, this was scientific insight of a high order; if it does not, *The hippocampus as a cognitive map* is a beautifully constructed mansion with many pleasing chambers, but one nonetheless constructed of cards.

Recordings made from single neurons in conscious animals have been invaluable in the analysis of sensory systems. But one may legitimately doubt whether this approach will prove as fruitful when it is applied to structures which process information at points far removed from input or output. The classical technique, pioneered by Hubel and Wiesel (1962) in the visual system, is to search for the common properties of the set of stimuli able to activate a neuron. It is this technique that O'Keefe and his colleagues have applied to the hippocampus, albeit with important extensions in the kinds of stimuli presented to the animal. Now, it is possible to deduce in this way what information is *available* to a neuron; and, in the sensory systems, that is

the most important thing we need to know. But it is not possible to deduce what the neuron *does* with the information it receives. An analogy may make this clear. In a railway station a great many people have a copy of the timetable, but a porter, a train driver, and the station chief make very different uses of it; we would learn very little about their several functions by asking them whether they possessed a timetable. In the same way, I am sceptical of the claim that single-unit studies of the hippocampus can tell us very much *by themselves* about what this organ does.

This general doubt is reinforced by an odd feature of the data reported in such studies: experimenters seem to discover hippocampal units which respond to those particular environmental regularities that they have built into their experiments, *whatever these happen to be*. Thus, Vinogradova (Vinogradova and Brazhnik 1978) in Moscow, keeps her animals immobile, presents to them stimuli which are either novel or are repeated until they become familiar, and allows very little other variation in experimental procedure: she finds hippocampal units that are responsive to novelty or familiarity. O'Keefe (O'Keefe and Black 1978), in London, gives his animals spatially extended environments to explore, with stimuli that either remain fixed in their spatial positions or are moved around: he finds "place," "misplace," and "displace" units. And M. W. Brown (personal communication 1979), in the Department of Anatomy at Bristol, gives monkeys a complex learning task with contextual cues that tell them which of two alternative responses is to be made to a main cue: he finds units that are responsive to context. I deduce from this pattern of findings, not that experimenters notice only what they expect to see (though this may play a part), but that the hippocampus is an organ that is able to store (probably temporarily) many *different* kinds of environmental regularity, depending on those that the animal encounters. If this is so, it is a chimera to seek for the *particular* environmental feature to which hippocampal units are *always* responsive: there is none. If there is, it can have nothing essentially to do with space, for in neither Vinogradova's nor in Brown's experiments was the critical feature spatial.

It is much to O & N's credit that, though single-unit experiments provided the impetus for their theory, they recognize that such experiments must be supplemented by data of other kinds; and, indeed, much of their book consists in a valiant attempt to reinterpret the vast bulk of findings from lesion studies in a manner that is congenial to their theory. The reasoning that has gone into this attempt is often ingenious in the extreme; and future research will need to take into account both the many specific suggestions that O & N derive from their theory, and their damaging criticisms of alternative accounts of the same data. Nonetheless, their reinterpretation of the results of lesion experiments is, in the end, a failure. There are too many points at which the account that they offer is lame, far-fetched, and lacking in direct evidence. To document this criticism in detail would require a review of the evidence as long as their own (Gray and McNaughton, in preparation); here there is space for only one general and two specific points.

The general point concerns the *post hoc* quality of many of the arguments offered by O & N. This quality is a direct consequence of the strategy of explanation they adopt. They propose a theory about what the hippocampus does, and this is admirably developed from general principles, themselves derived from a thorough initial analysis of the concept of space. This is physiological psychology at its best. But, for the most part, *it is not this theory that is used to explain the effects of hippocampal lesions*. Instead, these effects are attributed to the properties of parts of the brain that remain intact after such lesions, that is, to the so-called taxon system. But the properties attributed to this system are not derived from general principles: they consist of just those properties that are able to account for the known consequences of hippocampal lesions (the discussion in the accompanying précis of the role of intertrial interval is an example of this mode of argument). This retrospective, rather than predictive, character of the discussion of the lesion data robs it of almost all conviction.

For my specific points I shall refer to two experiments on the effects of hippocampal lesions, neither of which can easily be explained by the spatial theory.

According to this theory, in a task in which normal animals are unable to use place hypotheses, hippocampal animals should not differ from them. Webster and Voneida (1964) trained cats on a tactile discrimination. Vision was totally occluded, the animal having to palpate raised patterns at the bottom of a tube into which it inserted a paw. Pairs of patterns were presented simultaneously, with position (left or right) randomised. Pushing the correct pattern was rewarded with food, pushing the incorrect one was not. I should be interested to know what spatial element O & N can detect in this task. Hippocampal cats were not impaired in the learning of this discrimination; but they are not normally impaired in simultaneous discriminations of any kind. However, they were severely impaired during reversal learning and extinction. Neither of these findings fits easily with the spatial theory; but they fit perfectly with the inhibition theory that spatial theory has attempted to replace.

The second experiment (Rawlins, Feldon, and Gray, in press) investigated the partial reinforcement extinction effect in the straight alley. The results refute two predictions made by O & N.

In their book (p. 347) they state that the partial reinforcement effect should be "superimposed upon the deficit in extinction shown by continuously rewarded hippocampal animals." Hippocampectomy, however, completely abolished the partial reinforcement effect, and this was due equally to a rise in resistance to extinction in continuously rewarded animals and to a *fall* in resistance to extinction in partially rewarded animals. The second of these changes is particularly difficult for O & N to explain, since they suppose "taxon" hypotheses to be inherently more persistent than the "place" hypotheses of which hippocampal animals have been deprived. On the other hand, the findings reported by Rawlins et al. (in press) are in agreement with a theory of hippocampal function (Gray et al. 1978) for which O & N have found "little evidence" (their précis).

The second prediction made by O & N and refuted by the findings of Rawlins et al. (in press) concerns the locus in the alley at which the effects of hippocampal lesions should be most pronounced. As O & N state in their précis, "the extinction situation is formally equivalent to the passive avoidance task." According to Black, Nadel, and O'Keefe (1977) the spatial theory predicts that, in a passive avoidance task in the straight alley, the effects of hippocampal lesions should be greater the further the animal is from the goal (in which the shock occurs). The same should therefore be true in an experiment on extinction. But in the experiment by Rawlings et al. (in press), both the rise in resistance to extinction in continuously rewarded animals and the fall in resistance to extinction in partially rewarded animals, caused by hippocampal lesions, were as clearly marked in the goal as in the start and run sections of the alley.

There are many more detailed objections of this kind that one could make to O & N's theory; given its wide scope, it would be astonishing if there were not. To put these objections does not diminish the substance and importance of their achievement. *The hippocampus as a cognitive map* is packed with ideas which, through the authors' articles and conference papers, have transformed discussions of both hippocampal function and spatial behaviour during the last decade; and these ideas will undoubtedly influence research and theory for many years to come. But to some degree their work has already been absorbed and transcended by more recent developments.

Consider, for example, the demonstration by O'Keefe, Nadel, Keightley, and Kill (1975) that fornix lesions abolish place learning in rats. This, along with Helen Mahut's earlier work with monkeys, was important in showing that spatially complex tasks are particularly susceptible to impairment in hippocampal animals. O & N, in my view, misinterpreted this finding as indicating that spatial analysis was the critical ingredient in these tasks, just as they misinterpreted the single-unit experiments on which I have commented earlier. It has now been shown by Olton et al. (1979) that, although hippocampal deficits are indeed massive in spatially complex tasks, this is due to *other features of those tasks than their spatial characteristics*. This is not the place to consider what those other features might be; Olton's own conclusions are in certain respects in good agreement both with the view of the single-unit data advanced above and with several other approaches to the mystery of hippocampal function (see Gray et al. 1978, and Rawlins 1979). These approaches have in common that they postulate a role for the hippocampus in determining the correct

response under conditions of uncertainty, ambiguity, or interference. In such a view spatially complex tasks are merely a subset (albeit a very important one) of a wider class of tasks in which the sources of interference are particularly great. It is along these general lines that a comprehensive theory of hippocampal function is likely to be created. The emphasis on space *per se*, in contrast, is difficult to integrate with data from tasks of other kinds (Webster and Voneida's [1964], for example), and it runs into insurmountable difficulties as a general account of hippocampal function.

Note

1. Address 1979–80: Laboratoire de Physiologie, Faculté de Médecine Pitié-Salpêtrière, 75634 Paris Cedex 13, France.

by **Ernest Greene**

Department of Psychology, University of Southern California, Los Angeles, Calif. 90007

On panspatial theories of brain and behavior

The hippocampus as a cognitive map is a masterfully written and thoroughly enjoyable book which will have a substantial impact on physiological psychology. The breadth of scholarship demonstrated by these authors is impressive, as is their dexterity in handling procedural details which are often lost in the review process. However, the crowning achievement of the book is in their parody of theoretical excess.

Beginning with a theoretical viewpoint which is inherently defensible, they have contrived extensions and distortions of logic to produce a delightfully obtuse and ridiculous model of hippocampal function. The humor is dry (very British, of course!), and at no point do the authors resort to slapstick or pun. They do play one or two practical jokes on their doltish colleagues who use the lesion method. In chapter 5 they say that an overview of these studies shows that they provide precious little information about the functions served by the hippocampus, but then they devote eight of the fifteen chapters of the book to an evaluation of the complex behavioral results reported by these studies. One can also imagine the apoplectic and impotent outrage of these hapless souls when they discover that their evaluation of the behavioral changes in hippocampal animals (their ideas about what the changes mean) have been neatly segregated from the data – with the latter being thoroughly cited while the theoretical concepts are presented elsewhere and without citation. Somewhat ashamedly I must admit to a chuckle, and a twinge of pity for those poor devils who were the butt of the joke. In defense of the authors of the book, however, I believe they did have a clear and decent purpose for doing the literature review in this way – to present the fruits of their lighthearted and whimsical caricature from being confused with serious ideas about how the hippocampus functions.

It would be difficult or impossible for me to give examples which adequately capture the parody in this book, since much of it derives from pushing a theoretical concept to an illogical extreme. In other cases it comes from the straight-faced presentation of an argument which is directly opposite of that which was given in a previous paragraph. For example, in discussing the steep avoidance gradient of hippocampally lesioned rats (ch. 8), the authors propose that without the spatial map provided by the hippocampus the animals are not able to judge their location in the alleyway, and are able to inhibit their behavior only when they have been physically confronted with the taxon cue (i.e., the conditional stimulus). Presumably the cue is not able to act at a distance because the organ which maps space and time (the hippocampus) is missing. This proposal follows directly on the heels of a discussion of discrimination learning (ch. 7), where the authors cite data showing that hippocampal rats have no problem running down an alley to encounter the taxon cues used in the discrimination task (requiring expectations extending over time and space).

In chapter 11 we learn that loss of the spatial map produces some abnormalities in sexual behavior (problems of knowing where?). We also learn that the hippocampus mediates hormonal responses when the animal is no longer given a reward for its actions. This is one of the many situations in which the hippocampus serves to detect novel match or mismatch (i.e., the nonreward is new because it did not occur at the right place, but a different reward would also be new if it occurred at the right place, but might also be new if it occurred at the wrong place, or for that matter a nonreward or a reward would be judged as new if it did not occur at the right *time*, or if it occurred at the wrong place at the wrong time . . .). It seems clear that detection of any new event will require the contribution of the hippocampus, and this may also be true for nonevents if the spatiotemporal relationships are not quite right.

Perhaps the most interesting wordplay comes in the discussion of the role of the two brain systems in language. The cognitive map is mediated by the hippocampus, and is responsible for judging events in space and time, and for providing all situational contexts (since these are, after all, extensions of events in space and time), and for providing hormonal responses to mild (but not strong) fear, since mild fear is usually "situational" or place-defined (p. 360). The taxon system handles other behavioral situations, producing behavior in animals which is excessive, persistent, and stereotyped. In humans, however, the taxon system mediates grammar, transformational processes, syntactic structure and lexicon (p. 401). The hippocampal cognitive map provides for the person's deep semantic comprehension of language. This model should predict that humans with hippocampal damage would suffer from aphasia. Indeed, careful questioning of these patients will show that they have no knowledge of Kant's *analytic a priori* or *synthetic a posteriori* relationships and events (ch. 1), or for that matter of the quintessential role of Leibniz's *monads* in determining our destiny. The hippocampally damaged patient seems to converse normally about his life prior to surgery using taxon-mediated automatonlike speech, but clearly he has no deep semantic comprehension of what is being said, and therefore is both amnesic and aphasic in the most profound sense.

I look forward with eagerness to a sequel to this book. Having dealt so effectively with the hippocampus, the authors are likely to elaborate further on the taxon systems – possibly resulting in the discovery of the "antitaxon" (which is, of course, everything the taxon might be, but isn't!). Given the general agility and breadth of these authors, they may choose to elaborate upon the cortex, or deal with the hypothalamic and brain stem systems mediating drives. I, for one, would choose the latter, and would be especially interested in showing that the diverse human urges, lusts, and passions are juxtaposed in the brain like the many delightful places over the surface of the body which may be licked, stroked, and suckled.

by **H. Hécaen**

Unité de Recherches Neuropsychologiques et Neurolinguistiques, I.N.S.E.R.M., 75014 Paris, France

Cortical areas involved in spatial function

Although one cannot readily discard the arguments set forth against the cognitive map theory of hippocampal function, I would by no means intervene in a debate among hippocampal specialists concerning the validity and weight of the experimental results. I would merely like to point out that the spatial cognitive map theory constitutes a useful model for the study of certain spatial disorders.

It is in fact tempting to draw parallels between the experimental animal results and the deficits in spatial learning observed by Milner (1965) in cases of right hippocampal ablation in humans. Though many of these patients had no apparent spatial deficits, they were grossly impaired in maze-learning tasks irrespective of the modality tested. Also, the interpretation of spatial disorientation in man must obviously take into account the role played by the posterior parietal region in the spatial function of primates.

Studies by Hyvarinen and Poranen (1974) and by Mountcastle and his associates (1975, 1976, 1978) have demonstrated the existence of certain properties in the cells of areas 5 and 7 of the primate cortex. Essentially, there appears to be a system at the level of area 5 which constitutes "a continually freshened image of spatial relations, of the

position and movement of the body, head and eyes in relation to each other and to immediately surrounding space" (Mountcastle 1978). Loosely associated with this system is a command apparatus situated at the level of area 7 [see Kupfermann & Weiss: "The Command Neuron Concept" *BBS* 1(1) 1978]. It would seem that this apparatus, which is sensitive to motivational factors, is responsible for the regulation of visual and manual processes in personal space as "described by the reach of arm and hand, and particularly for the direction of visual attention into the world around us" (ibid.). Robinson et al. (1978) and Robinson and Goldberg (1978) have recently provided another interpretation for the activity of these posterior parietal cells. These authors reject the role of the posterior parietal region in the regulation of movement and propose that this area should be considered the neurophysiologic substrate for visual attention only. Finally, it should be noted that the ablation of these regions in primates has produced deficits in "reaching" or in "allocentric" orientation.

A particular type of spatial disorientation has been termed "the loss of topographic memory" in view of the mnestic character of the disorder; it is more frequently associated with other deficits of spatial function produced by right posterior parietal lesions. However, cases have recently been reported in which the loss of topographic memory was virtually unaccompanied by any other deficit.

Such was the case in one of our patients, who exhibited a loss of topographic memory with no other spatial deficit along with a serious impairment of maze task learning. This patient suffered from a vascular lesion in the right occipital region as revealed by the computerized tomographic scan; note that the hippocampus is supplied by the posterior cerebral artery.

Taken together, these findings suggest that the mnestic character of some of the spatial deficits produced by lesions in the posterior right hemisphere may be due to the disruption of connections between those cortical structures and areas of the limbic system. The importance of afferent and efferent connections between the posterior parietal regions, the cingulate gyrus and the retrosplenial cortex has recently been demonstrated in monkeys (Mesulam et al. 1977; Roesen and Van Hoesen 1977).

These findings make it possible to venture a unitary interpretation of spatial function in human orientation. Like Mountcastle (1978), we would envisage a neuronal assembly at the level of the inferior parietal lobe which furnishes the coordinates of the body image in its immediate surround and is related to visuomotor coordination [see Gyr et al. "Motor-Sensory Feedback and Geometry of Visual Space" *BBS* 2(1) 1979]. The more distant visual world – what is beyond manual reach – becomes organized on the basis of these coordinates as the individual moves from place to place. Subsequently, and in virtue of some other neural substrate (such as the hippocampus) the individual establishes a spatial map based on multisensory cues. Those stimuli which, through experience, have acquired significance as spatial landmarks, serve as continual signals to the individual as he follows a familiar itinerary; also providing signals are those stimuli that are contrary indices for the particular itinerary chosen. In this way, the individual can orient himself.

Parieto-occipital lesions disturb awareness of the body's position in space. As a function of the specific lesion site within these areas, the deficit produced may affect either the manipulo-spatial domain alone or more distant, extrapersonal space which, as described here, is not simply visually appreciated but is, in fact, inferred from the data available in pericorporal space. Lesions which isolate these areas either from their contralateral sensory afferents or from their connections with motivational systems will result in attentional disturbances with respect to the contralateral half of the body and/or space. The mnestic character of many of these spatial deficits, meanwhile, is probably best understood as the result of lesions involving structures which are specifically concerned with learning, like the hippocampus, or which compromise the connections between these structures and the posterior parieto-occipital region.

Although clinical and experimental findings may be fruitfully reflected in this analysis, one must not forget that spatial deficits are, if not exclusively at least principally, the result of right hemisphere lesions. The existence of functional hemispheric specialization in humans requires certain additional considerations. In conjunction with the apparition of language, and because the latter requires special neural substrates, the necessary tissue developed predominantly in the left hemisphere in areas which are not found in subhuman primates (i.e., the supramarginal gyrus and the angular gyrus). Consequently, the mechanisms for spatial integration and holistic perception of the environment had to be subsumed by tissue which was predominantly available only in the right hemisphere.

by **Richard Hirsh and Joel Krajden**

Department of Psychology, McGill University, Montreal, Quebec, Canada H3A 1B1

Hippocampal function: logic, logic, and more logic

The hippocampus as a cognitive map will be an important and controversial book. It employs thoroughly cognitive theories to explain phenomena in the area of the physiology of learning and memory. This field has long been dominated by simple associative or connectionistic approaches, despite the fact that many, if not most, nonphysiological psychologists have found such approaches to be seriously deficient in accounting for most behavior.

Moreover, the approach of the authors is more neurological than that of more conventional work. O & N succeed in being truly comprehensive. The behavioral correlates of hippocampal EEG and unit activity are integrated with the effects of lesions of that structure upon behavior. More importantly, the authors make use of morphological and physiological knowledge in an intelligent and inventive manner to explain how the hippocampus carries out the psychological functions that they ascribe to it. All too often theory in physiological psychology is nothing more than the attribution of some psychologically defined process to some morphologically or pharmacologically defined entity, with little attention to whether that entity is capable of carrying out the assigned function. The authors have provided an example that should be followed whenever the appropriate data is available.

The authors' thesis also carries very important implications for purely behavioral theories of learning. As the late Abe Black realized, explaining the behavior of normal animals in cognitive terms and that of animals with lesions in associative terms implies that both types of systems are presented in the brain. To date most of the controversy in the psychology of learning has been whether one or the other type of theory was correct.

To a student of hippocampal function, *The hippocampus as a cognitive map* is a very perplexing book. From one viewpoint the ideas and arguments that it presents about hippocampal function are powerful, and in my estimation right. Viewed from another aspect they are wrong and almost trivial.

Brain processes may be characterized both in terms of the logical operations comprising them and in terms of the information being processed. These two kinds of characterizations are more or less independent in that one usually has little to say about the other. We couldn't agree with O & N more when they assert that the hippocampus carries out logical operations necessary for the production and use of cognitive maps. However, we think they are totally wrong when they assert that the hippocampus processes only spatial or geographic information.

The initial segment of the book compares the logic of taxon systems to that employed by the system of which the hippocampus is a part. Characterizing the learning systems typified by S-R theories as category inclusion logic and styling them as taxonomic is precisely on target. No restriction is placed upon the kind of information to which this logic may be applied. They may deal with both spatial and nonspatial problems. The formal properties of the taxon system are then contrasted with that of the locale or mapping system. This is the first misstep in that it implies that nontaxonomic logic is to be found only in systems dealing with geographic information. Such an assertion is false, as attested to by the applicability of analytical geometry to all kinds of problems, spatial and otherwise. Such tunnel vision probably accounts for the omission of dimensionality as a key formal property of mapping systems. Maps are dimensional in the sense that a relation

between a given point and two others can only obtain if a certain relation between the latter two obtains. Dimensions are quite different from inclusion categories. The latter identify a set of items having common elements and disregard the diversity, whereas the former are a means of systematically characterizing variation. This, incidentally, is the crux of the argument between Kant and the British Empiricists.

In a subsequent section of the book it is claimed that hippocampal dysfunction does not affect nonspatial problems and all reported effects of such dysfunction are attributed to the animal's not knowing where it is or where it wants to go. This assertion is also false. Quite early reports describe deficiencies on the part of hippocampally ablated animals in reversing nonspatial discriminations (Douglas & Pribram 1966; Silveira & Kimble 1968; Teitlebaum 1964; Webster & Voneida 1964) as do subsequent studies (Becker et al. 1979). There are data indicating that hippocampal dysfunction results in deficits in acquiring and performing nonspatial sequences (Caul et al. 1969). Moreover, hippocampally ablated animals, unlike normal animals, are not capable of transposition following the learning of a size discrimination. All of these effects can be accounted for by disruption of the logical operations underlying the nontaxonomic system. Close analysis of the explanation offered by O & N reveals that they rest on the formal properties of the locale system rather than the processing of its part of spatial information.

To a certain extent the authors implicitly recognize the importance of the formal properties of the mapping system. In order to account for the amnesic effects of hippocampal lesions in humans they postulate that the hippocampus of the left hemisphere carries out the same logical operations in the linguistic realm that the one in the right hemisphere performs upon spatial information.

Admiration for the authors' methods in handling neurobiological data does not preclude demurral from their conclusions and inferences. We are inclined to regard the presence of theta waves as indicating that the hippocampus is not working very hard, on the ground that such highly syncrhronized activity in hippocampal cortex is probably functionally analogous to alpha activity in neocortex.

Secondly, it is fair to describe the authors' interpretation of their unit activity studies as one holding that hippocampal neurons detect the present location of the animal. Such an interpretation is not completely suited to explaining the effects of hippocampal dysfunctions in terms of disrupted mapping processes. Cognitive maps guide behavior insofar as they describe the relation between the present location of the animal and that of the incentive, or, if you will, the place it wants to be. To date O & N have not addressed the question of unit representation of destination. Moreover, their methods during unit studies focus on whether or not firing occurs in a given place, leaving quite open the question of how the relationship between the animal's location and destination is encoded.

To date the main effect of *The hippocampus as a cognitive map* has been to engender a howling controversy over whether or not the realm in which the hippocampus operates is exclusively spatial. Our fervent wish is that ultimately this tumult will be regarded as a waste of time. The book has a great many things to say about the formal properties of the system in which the hippocampus participates. The proper role of the book is to stimulate interest in the logical operations carried out by the hippocampal system that endow it with such formal properties.

by **J. Eric Holmes**

Department of Neurology, University of Southern California, School of Medicine, Los Angeles, Calif. 90033

Waves and cells, maps and memories, space and time

My old hippocampus went: " 'Thetive'!
This theory's most innovative.
With Nadel and O'Keefe
One finds easy belief –
It's some kind of a map connotative!"

O & N's accompanying précis gives an inadequate picture of their massive opus. Ranging, as it does, from the philosophy of Kant through the anatomical controversy over efferent pathways, the neuropsychology of the theta (RSA) rhythm to an extensive discussion of lesion data and lesion philosophy, human pathology and the syntactic structure theory of Chomsky, it provides an abundance of openings for the commentator. I will use the theta rhythm, the experimental technique and the human lesions for my locale points.

"It must mean something!" Watching the sinusoidal theta rhythm rolling out of the EEG machine for the first time, one cannot escape the conviction that it contains a special message, albeit yet undeciphered. This conviction is heightened by the knowledge that the hippocampus has been associated with memory. In 1954, Green and Arduini suggested alerting and arousal and Adey (1960) suggested memory and learning. I thought it "indicates a readiness to act" (Holmes and Adey 1960). The biggest controversy seemed to be over the impersistence of theta in Grastyan's cats trained in approach behavior (Grastyan et al. 1959). Vanderwolf and Heron (1964) advanced the theory that the rhythm was related to "voluntary" movements. That it might be just a humming noise the brain makes when it is turned on was a hypothesis none of us cared to consider.

O'Keefe, Nadel, Ranck and the others, however, have demonstrated the comforting fact that there are hippocampal cells that fire bursts of spikes at the theta frequency. Surely if cells are firing rhythmically the rhythm "really does" mean something! "And thus there is a prima facie reason to believe that these slow waves are providing information about underlying neural events" (O'Keefe and Nadel, p. 143).

Some of the controversies about the theta rhythm have been resolved by assuming that there are two different theta rhythms and that it may be associated with different behaviors in different species (O'Keefe and Nadel, p. 219). The waves in the cat (the neurophysiologist's traditional animal) are quite different from the waves in the rat (the experimental psychologist's traditional S). Both of these animals differ from the rabbit, the animal in which the phenomenon was first reported, and still the most reliable theta generator (Winson 1972).

"Nonmovement" theta occurs in most species during REM sleep. Because of the association of the hippocampal rhythms with learning and memory, this has led to the interesting proposal (Pearlman 1979) that REM sleep is necessary for memory. I wish O & N had commented on this.

The strange catatonic, species-specific behavior of the rabbit – the tonic immobility or animal hypnosis response – is accompanied by low frequency theta (Harper 1971). No one has yet investigated the species-specific immobility of the pointer dog. I predict the hippocampus will be found to be in theta.

Unmentioned by O & N is another complication, the rare occurrence of anything resembling a theta wave in monkeys and men. Although Crowne et al. (1972) have shown that a theta rhythm can be leeched out of the monkey's hippocampal EEG with a computer search, even they would agree that it is not an obvious occurrence. Does this mean the monkey (and by analogy, man) does less cognitive mapping?

O & N identify the theta rhythm with the recording, or the reading of recordings, from the hippocampal "map." "Theta activity is necessary for the original construction of maps, for their subsequent modification when there is a mismatch between the map of a situation and the present sensory array, and, finally when the map is being used in the predictive mode" (O'Keefe and Nadel, p. 190). They suggest a circuit for recording place-labeling stimuli which would be "scanned" by the theta wave. Cells would then fire and – and something would happen. Like most such speculations, the proposal fades off into "activate . . . circuits in the brain stem" (O'Keefe and Nadel, p. 229). This does sound like Adey's (1967) "these wave patterns . . . underlie the initial deposition of information in cerebral tissue, and . . . subsequent recall." It is not quite as dramatic as Landfield's (1976) suggestion that the hippocampus cells form a hologram and the theta rhythm is a laser beam!

Is the map literally a physical representation of the rat's environment? Probably not. "CA1 place units recorded next to each other sometimes represent neighbouring parts of an environment, as often they do not" (O'Keefe and Nadel, p. 223). Unfortunately for us, we can deal only with mental images that have some familiarity. No one could have suggested that the hippocampus was a hologram until holograms

were invented. Maps have been around a long time, but this does not mean that the hippocampus looks like something out of Rand McNally. At best the theory suggests that these "place cells" are elements in a circuit of cells that fire when specific "place labeling" inputs occur. Should there not be "map cells" in the visual, olfactory, and auditory sensory areas?

The experimental basis of the theory is given on pages 190 to 217, with a summary following. One might be tempted to accuse the authors of publishing their 543-page monograph prematurely. The amount of excitement generated among hippocampologists by their theory more than justifies the book, however. The authors proceed to reexamine a great number of old and new ideas about the brain in a most productive fashion. The chapter on physiology alone contains at least a hundred sentences, any one of which could be easily expanded into a viable proposal for a Ph.D. thesis.

When a cell increases its firing, the investigator puts his head down where the rat is (figuratively) and tries to guess what the cell is "seeing." Results depend on the ability of the scientist and the rat brain cell to "see" the same things. O & N are imaginative, but Ranck (1973), looking at the same kinds of cell records, categorizes them quite differently. The very use of "see" in the example above betrays the bias built into our language. Wouldn't a real rat psychologist be more likely to say the hippocampal cell "smells" the input?

I find it startling that an olfactory animal like the rat would have "place cells" that would not fire just because the lights were turned off. Where are the odor identification cells? Surely the complexity of the rat's olfactory world must equal the complexity of our own world of color vision.

I am not arguing with the data, only with the interpretation. A rat brain cell cannot be credited with recording a location unless O & N can also "see" the location. And it seems reasonable to me to expect the categories "seen" by hippocampal cells to be quite different from those "seen" by psychologists. It is just because a cognitive map seems so incredible a thing for the cells of the hippocampus to be that it may be an inspired correct guess.

One of the problems in studying the relation of hippocampal electrical activity to memory or learning was the controversy over lesion effects. In hippocampal man, the memory loss was devastating. In hippocampal animals it wasn't so obvious; many did as well, or better than, the controls. O & N make a nice attempt to cram these studies into a cognitive map of some sort. They succeed by assuming, as most of us are forced to do, that there are several different kinds of memory, possibly located in different places. The authors do a fine job of illuminating some aspects of the human amnesic syndrome, but they slight the dimension of time.

It would seem probable that a neuronal circuit exists that would register events as related in temporal sequence as well as in spatial and in linguistic sequences. How are memories recognized as "older" or as "recent?" The intact brain is able to recognize something as "familiar" and then ask "where?" and "when?"

The authors point out that recent studies indicate that amnesics have a loss of "old" memories as well as recent ones (O'Keefe and Nadel, p. 434), but the retrograde effect, especially in trauma patients, is often very impressive. Can we account for the apparently complete loss of memories of a period of years and their gradual, partial recovery? Penfield and Mathieson (1974) have suggested that more recent memories require the anterior hippocampus, old memories the posterior. The farther back into the temporal lobe the lesion extends, the farther back in time goes the retrograde amnesia. This hypothesis is so incredible that someone ought to subject it to experimental test.

Things are mapped in four dimensions, not just three. Is the temporal axis innate or learned? Does the retrograde amnesia effect tell us something about the nature of the brain, or something about the nature of time itself? There is a myth among clinicians that patients with temporal lobe epilepsy have frequent attacks of *déjà vu*. I have encountered one patient with *déjà vu* of such intensity that he believed himself gifted with prophetic dreams. But questioning of epileptics, has, in general, not indicated that they are more subject to this phenomena than the rest of us.

We need another monograph on the philosophical, psychological and neurophysiological nature of time. Perhaps O & N can be persuaded to write it, once they iron a few kinks out of their theory of space.

by **James A. Horel**

Department of Anatomy, State University of New York Upstate Medical Center, Syracuse, N.Y. 13210

Lost maps and memories

The experimental results of O & N and others make an impressive case for the spatial functions of the hippocampus. The cognitive map theory presented here is an innovative development of the implications of these results. It is rich both in new ideas and old ideas seen from a new vantage. It presents a much needed fresh and promising approach to the function of the hippocampus. It also at times vastly overreaches. While I admire this imaginative work, I must contest the interpretation that is made of the human amnestic syndrome.

The attempt to accommodate the allegation that hippocampal lesions produce amnesia is exceptionally inventive but totally implausible. Among other things, it requires us to place Chomsky's semantic deep structure in the hippocampus. This extravagance is unnecessary. The amnestic syndrome is not derived from the theory of cognitive maps; the theory does not predict the symptoms, it adapts to them.

The authors embrace the current orthodoxy that blames the failure to find amnesia in animals with hippocampal lesions on a misunderstanding of the syndrome and the failure to use appropriate tasks. They argue that the amnestic syndrome is a consequence of the loss of the locale system situated in the hippocampus. The difficulty in finding comparable deficits in animals with hippocampal lesions is the result of using memory tasks that engage the undisturbed taxon system. But there is at least one experiment not discussed by the authors in which the amnestic syndrome was convincingly demonstrated in monkeys, and with a taxon task. Scoville, who had produced the amnesia in humans by removing the ventromedial quadrant of the temporal lobe, performed exactly the same surgery in the same way in monkeys (Correll and Scoville 1965). The animals had been trained preoperatively on delayed match-to-sample, a task that Correll and Scoville thought would be a sensitive measure of amnesia. The lesion had a devastating effect on performance. The animals showed no retention (retrograde amnesia) and had enormous difficulty relearning, some never rereaching criterion at zero delay (prograde amnesia). It should be emphasized that the lesion, including the surgical procedure, was the same as that performed on the famous amnestic patient, H. M., and by the same surgeon. This direct replication of the human amnestic syndrome is generally ignored because of one embarrassing outcome of the experiment: a group in which the hippocampus alone was removed performed flawlessly.

According to O & N's classification, delayed match-to-sample should be served by the taxon system and therefore should not be disrupted by removing the hippocampus. This is the finding of Mishkin and Oubre (1976) as well as Correll and Scoville. Amnesia, according to O & N, results from loss of the locale system in the hippocampus. However, the surgery of the human amnestics was not limited to the hippocampus; they had the same lesions as Correll and Scoville's monkeys, including the entire ventromedial quadrant of the temporal lobe. There is no justification for assuming that the retention and acquisition deficit displayed by these monkeys is not the result of the same processes as the human retention and acquisition deficit called amnesia. The monkeys must learn and remember this task however it is classified. The absence of a deficit with hippocampal damage is strong evidence that the hippocampus has little to do with the amnestic syndrome in monkeys or humans.

What then is the source of this deficit? Mishkin and his colleagues are investigating the possibility that amnesia occurs only with combined damage to hippocampus and amygdala. I have presented evidence that it is produced by damage to the surrounding white matter (Horel 1978). The Correll and Scoville data support the latter argument. I performed a rank order correlation between performance at match and Correll and Scoville's estimate of amount of damage to hippocam-

pus, amygdala and temporal stem (white matter of temporal cortex). The deficit correlated by far the best with damage to the temporal stem. This is consistent with the finding that lesions to the source of this white matter in the temporal cortex produce severe deficits on this task (Dean 1974, Mishkin and Oubre 1976) but lesions restricted to hippocampus or amygdala do not (Correll and Scoville 1965). Thus, it is probable that the severe amnesia produced in monkeys by medial temporal lesions is the consequence of damage to this white matter and this same white matter must also have been damaged in the cases of human amnesia (Horel 1978).

The cognitive map theory can stand alone without the amnestic syndrome. The authors have presented a convincing argument concerning the need for such maps to find our way among objects and places. The association of these maps with the hippocampus is good but not as convincing. A few troublesome features remain. One of these is the prediction that hippocampal lesions produce deficits on delayed response. I disagree. There are a number of studies showing no deficits on delayed response when the lesion is restricted to the hippocampus (Mishkin 1954, Orbach, Milner and Rasmussen 1960, Mahut and Cordeau 1963, Mahut 1971). An interesting point that bears on the above argument is that in the Orbach et al. study the authors predicted from their experience with human amnestics that there would be no deficit on delayed response with medial temporal lesions, and the results confirmed their prediction.

Another awkward problem derives from placing the source of exploratory behavior in the hippocampus. While it may be true that removing the hippocampus by itself decreases exploration, removing it along with the rest of the temporal lobe has quite the opposite effect. After a bilateral temporal lobectomy, monkeys exhibit an extreme compulsion to explore and react to every object in their environment (Klüver and Bucy 1939). Whatever the reason for this, it certainly cannot be said that these monkeys have lost their motivation to explore with the loss of their hippocampus.

by **Robert L. Isaacson**

Department of Psychology, Center for Neurobehavioral Sciences, State University of New York at Binghampton, Binghampton, N.Y. 13901

Hippocampal lesions and intermittent reinforcement

It has been said that there is, in most large books, a better, smaller book trying to get out. In this case it is the "bigness" of the book that provides its greatest contribution. The kernel is, of course, the spatial map theory of hippocampal function. This theory has been well known for several years and the most important data, those from chronic single cell studies, reported in many publications. Therefore, the small book hidden in the big one is already out. The big book, however, contains a great deal of additional facts and information. So much, indeed, that the book is really a "must" for the people with research or scholarly interest in the hippocampus.

Before all else, the authors are to be complimented on creating a very readable book. Their prose is excellent. The early sections on older philosophies of space are exceptionally well done. Another singular contribution is in the quantity and depth of the review of the literature in the field. Clearly, countless hours were spent reading, evaluating, and thinking about the data in many types of research reports. As a thoughtful compendium of research, it is unexcelled.

As noted, the general sense of the cognitive map theory has been known for quite a few years. In the book the authors attempt to extend the theory to encompass a wide variety of data that at first, or even second, third, or fourth, glance don't seem to be related to cognitive or spatial maps. This is usually done in an elegant and creative fashion. Yet these examples of theory building, renovation, or elaboration are sometimes less than satisfying. One example would be the common deficit found in animals with hippocampal lesions when changed from continuous reinforcement schedules to ones in which the animal has to wait 20 seconds between bar presses in order to obtain a reinforcement (DRL-20). This change was thought to be due to the intact animal's moving away from the area of the lever in order to prevent responding. I have not observed differences in such collateral acts on the part of lesioned animals. However, the fact is that the change from a continuous reinforcement schedule to one in which reinforcements are intermittent produces a great increase in the rate of responding. This hugely increased response rate interferes with the decrease in responding essential to efficient performance. Further evidence that it is the change from continuous to intermittent reinforcement which causes the impaired performance comes from studies showing a much reduced effect in lesioned animals that never experience continuous reinforcement but begin their operant careers on the DRL schedule.

Perhaps the strength of the book, namely its remarkably wide review of research, is also its weakness. In an attempt to evaluate or interpret almost every type of study, there are some weaker sections. These are areas in which neither of the authors has undertaken research. It is impossible in science today to be expert in all areas of brain behavior, even for any two people. Therefore, there are sections in which more critical comment or evaluation would have been useful.

In regard to the theory itself, I have strong reservations about it. Basically, I think it is improper to try to formulate a theory of the mental or behavioral role of any brain structure. In essence, the role of any structure depends on the state of the systems with which it is in contact. Its behavioral contribution can only be evaluated in regard to the function of other brain systems and their interactions.

The creation of such a larger theory is well beyond our capabilities at this time. In addition, I believe that there is no such thing as an invariant consequence of restricted forebrain damage. The behavioral effects of lesions are always dictated by the genetic makeup of the animals as well as their pre- and postoperative treatments or experiences. While we often pay lip service to these factors, too often we ignore them in our theories and data analysis.

While my personal objections to the basic theory (and others like it) may seem to indicate rather fundamental objections, I strongly recommend the book. Nowhere else is this information available in one place or the writing so well done. Overall, it is a worthy book.

by **Ray Jackendoff**

Linguistics Program, Brandeis University, Waltham, Mass. 02254

What is a cognitive map?

I find two of O & N's major claims convincing: first, that humans and other animals must have some innate notion of absolute space which they use to help orient themselves and find their way around; second, that the hippocampus is an important factor in long-term memory. However, the major argument of the book, that the hippocampus is specifically devoted to the encoding of "cognitive maps" that represent information about absolute space, seems to me to be seriously confused in several respects. These arise mostly in connection with Chapter 14, "An extension of the theory to humans." I will start with specifics and work my way to more general issues.

1. O & N's use of linguistic terminology, though in itself not really damaging to their argument, reveals some misconceptions about linguistic theory that are disturbingly common among nonlinguists. The error lies in their term "semantic deep structure," which conflates two distinct levels of linguistic structure: deep structure and semantic representation. The former term denotes the level of syntactic form that is described by phrase structure rules and that serves as input to the transformational rules. In the theory presented by Chomsky (1965), deep structure was also claimed to be the level of syntactic form to which the rules deriving semantic representation apply. In the late sixties, two divergent alternatives developed. One school of thought, "generative semantics" (e.g., Lakoff 1971), went on to claim that deep syntactic structure was isomorphic to semantic representation; according to this view, the term "semantic deep structure" is legitimate. However, other linguists (Chomsky 1972, Jackendoff 1972, Bresnan 1978) argue that there are aspects of semantic representation that cannot be enlighteningly derived from syntactic deep structure, and that information from surface structure also plays a role in the determination of meaning. Hence deep structure and semantic representation are indubitably distinct.

O & N's "semantic deep structure" is clearly meant to be what a

linguist of this latter persuasion would call "semantic representation" or "semantic structure." This terminological distinction is not just a quibble, for it has repercussions on how one interprets linguistic data, as will be seen directly.

2. Having spent my career discovering how difficult it is to find a mapping of any theoretical interest between semantic representation and syntactic structure, I found O & N's proposals for converting cognitive maps to language (pp. 403–409) more than a little cavalier. Most responsible linguists could not countenance a theory which derived O & N's examples (15–20) from a single underlying structure: the examples are too structurally diverse to be related by any sufficiently constrained set of transformations.

15. The rock fell from the roof to the ground.
16. The rock was on the roof and (then) it fell to the ground.
17. The rock was on the ground where it had fallen from the roof.
18. The roof had a rock (on it) and (then) the rock fell to the ground.
19. The ground has a rock (on it) which fell from the roof.
20. The roof had something fall from it on to the ground and that was the rock.

Furthermore, a linguist would have to be seriously concerned with why such relatively simple transforms of example 15 as *To the ground, the rock fell from the roof,* and *The roof, the rock fell from to the ground* are not grammatical realizations of the same information. In short, O & N's claim (p. 403) that "A variety of sentences can be generated from our simple spatial semantic map by a set of transformation rules" requires a great deal more rigorous justification than they provide.

To be sure, examples 15–20 all convey approximately equivalent information, and one would like to account for that. There are two possibilities, both of which, for the sake of constraining linguistic theory, must assume that the sentences have distinct syntactic and semantic structures. The first possibility is that the sentences are related by inference rules of the usual logical sort; the second is that the inferences are mediated by the construction of something like a cognitive map of the event being described. The latter seems more plausible to me because of the existence of numerous lexical pairs like *above/below, to the left of/to the right of,* and *inside/around,* and because of inferences about betweenness. (See the treatment in Jackendoff 1976, pp. 96–98, 116–118.) Thus, in the end I find the idea of a cognitive map appealing – but its connection to language must be far less direct than O & N would have us believe.

3. O & N's schematic cognitive maps (pp. 402, 407) are based on a mistaken assumption which I regret having perpetrated in Jackendoff (1976): that the canonical semantic structure for sentences describing motion invariably includes the endpoints of the motion, which I called the source and goal. Jackendoff (1978), in exploring the semantic resources of English further, shows that motion sentences are better characterized in terms of motion along a *path.* A path may be intrinsic to a verb of motion, as in *The flag waved,* or it may be expressed by a prepositional phrase. A source-goal pattern such as *from here to there* is only one type of path; there are at least two others, which may be called directions (e.g., *toward the mountain*) and routes (*along the river*). These different types of paths make different use of the landmark named by the object of the preposition. In a source-goal pattern the landmark is an endpoint; in a direction it fixes the orientation, but the path need not reach to it; in a route the path bears some other spatial relation to the landmark. This enrichment of the description of motion sentences cannot be accommodated into O & N's cognitive maps, which do little more than spell out the endpoints of a motion event. The proper corresponding enrichment of the theory of cognitive maps is not entirely obvious.

What ought to be attractive to O & N in this revised account of motion sentences is that it attributes existence to paths independently of whether motion actually takes place along them. For example, something can simply be oriented along a path rather than traveling on it, as in *The sign points toward New York.* Thus spatial organization as revealed by language is not tied to specific events or motor commands; it seems more to involve location and orientation with respect to landmarks. This accords well with O & N's infrahuman evidence.

On the other hand, we are left in the dark as to what a cognitive map might be like. O & N emphasize (p. 78) that "The cognitive map is *not* a picture or image which 'looks like' what it represents; rather, it is an information structure from which map-like images can be reconstructed and from which behavior can be generated." The problem is, what kind of informational structures should one propose to capture the meaning of *along, around, through, back,* and the rest of the semantically interesting words used in the description of paths? Miller and Johnson-Laird (1976) give it a serious try, but are themselves dissatisfied in the end. With the demise of the rudimentary maps O & N propose, they leave us with no specific proposal beyond the claim that organisms use landmarks to help them find their way around.

4. We now turn to O & N's most crucial claims, concerning the function of the hippocampus. O & N claim that locale (hippocampal) memory is encoded in terms of Euclidean, "absolute" space, while the taxon (nonhippocampal) system is encoded in terms of egocentric, "relative" space. Such a distinction in spatial representation is difficult to justify. In particular, linguistic evidence suggests that there is *no* clearcut distinction between absolute and relative space. Some paths, such as *from New York to Chicago,* are more or less absolute; others, such as *toward the church,* have an absolute endpoint but a direction dependent on the position of the thing in motion; others, such as *leftward,* depend on the orientation of the thing in motion; others, such as *from here,* depend on the position of the speaker. Grammatically these are treated as entirely uniform. Whether they are "relative" or "absolute" depends both on the choice of preposition and the choice of landmark. If there were a drastic difference between relative and absolute location, one might well expect them to be represented distinctly in language.

O & N also claim that locale memory is long-term and taxon memory is not. But this distinction and the absolute-relative distinction are incompatible. Surely one can have long-term memory of a route – such as the one O & N quote on p. 80 – that is specified at least partially in terms of one's orientation and one's subjective impressions of distance traveled. If so, either the locale system may contain egocentric, relative spatial memories, or else the taxon system may contain long-term memories stored in a form which may be interwoven with the locale system.

O & N further claim (p. 384, among other places) that taxon memory involves categorization by feature similarity, the abstraction of category prototypes, and the absence of spatiotemporal context information. The locale system, on the other hand, is devoted to spatiotemporal context; O & N leave unclear how this system encodes categorial information, if at all. Again, it is hard to make sense of this distinction, given the other distinctions. For one thing, how does information about spatiotemporal context reach long-term memory if not through short-term memory? Moreover, how can category prototypes and the category memberships of at least some individuals *not* be stored in long-term memory? Even the distinction between categorization and cognitive mapping is dubious: O & N, describing my theory of verb meanings in Jackendoff (1976), mention the semantic generalization from spatial to categorial change, demonstrated by sentences like *The coach changed from a handsome young man into a pumpkin.* If the content of such a sentence, which deals with category membership, is to be encoded by a cognitive map in locale memory, what does it mean to claim that the taxon system is the one concerned with categorization?

Finally, O & N claim that in humans the right hippocampus is devoted to nonverbal spatial functions, while the left is specialized for "semantic memory," that is, memory of narratives and the like. Their evidence for the distinction is derived primarily from experiments with recall of word lists and such. But memory for word lists hardly constitutes semantic memory. In fact, it is not even clear what semantic memory might be. Fodor (1975), Miller and Johnson-Laird (1976), and Jackendoff (in preparation), argue – and most work in artificial intelligence assumes – that the information conveyed by language is not encoded in a form specific to linguistic information. In order for people to be able to talk about what they see, and to be able to carry out orders, there

must be a level of conceptual structure that is modality independent; and there is good reason to believe that the semantic structure of language is not distinct from this level. Furthermore, much linguistic information we receive is inextricably mixed with nonlinguistic information, for example, in sentences such as *That man over there* [pointing] *is a spy; He's always waving his arms around like this* [demonstrating]; *The fish was this* [demonstrating] *long.* Thus, it does not appear possible to isolate an aspect of memory that might justifiably be called "semantic." In order to account for O & N's evidence of hippocampal lateralization, one must probably appeal to language-specific functions such as phonological, morphological, or lexical structure.

In short, O & N's claims concerning the functions of the locale and taxon systems are either muddled or inconsistent or both. Since, as shown above, the same is true of their claims about cognitive maps, the main thesis of the book is largely incoherent. In the absence of a clear theory, the masses of experimental evidence are hard to evaluate. Hence I found the book a disappointment, in spite of my general sympathy with its approach. My guess is that we need far better formal theories of the information that the brain processes before real progress can be made on cognition.

by **Leonard E. Jarrard**

Department of Psychology, Washington and Lee University, Lexington, Va. 24450

Considerations in evaluating the cognitive mapping theory of hippocampal function

O & N have written an important book that deserves to be read and studied by both students and investigators in the field. In an area where there are few theories attempting to integrate the results of brain and behavior studies, the book is unique. With a relatively small number of central concepts, the authors are able to apply the theory to research findings as diverse as single unit recordings, hippocampal theta, the lesion literature, and findings from the clinic; the scope of the research findings subsumed within the theory is truly surprising.

Any attempt to organize research results as diverse as those found in the hippocampal literature is bound to encounter conflicting results, and the theorizing by O & N is no exception. In no area are discrepancies more often found than in the lesion literature (the area to which the present commentary is addressed). There are a number of reasons why this is the case, and it is understandable why the authors do not insist that all results agree with the theory. In addition to problems associated with subtle differences in testing procedures when similar behaviors are being studied, the "hippocampal lesion" has varied from small lesions aimed at discrete regions of hippocampus, to extensive damage to the hippocampal formation, and damage to fimbria and/or fornix. Since the hippocampus consists of several neuroanatomically and neurophysiologically distinct regions, and these are often differentially involved in the lesions, it is not surprising that conflicting results are often reported. Considering the amount of "noise" in the lesion data, it is remarkable that the authors have been able to obtain as much support for the theory as is presented. However, one criticism of the book is that the authors fail to deal adequately with results from a number of carefully designed experiments that do not agree with the theory.

The neural model described on pages 217 to 230 of the book represents an attempt to incorporate what is known about the anatomy and physiology of the hippocampus into a model of how the structure acts as a spatial map. Specific functions are proposed for the three main systems (fascia dentata, CA3, and CA1), but it is stated that the model can be modified or rejected as new information becomes available. There now exists a considerable amount of data on the behavioral effects of selective damage to hippocampal cell fields and/or fiber tracts. Especially relevant are the effects on behavior found following damage to the CA1 pyramidal cell field. On the basis of unit-recording studies demonstrating a preponderance of place units and mismatch cells in the CA1 cell field, O & N propose that the cells are involved in cognitive mapping and novelty-detection/exploration. The available lesion data only partially support this hypothesis. As predicted by the theory, rats are more active in a novel open field following removal of the majority of the CA1 cells (Jarrard 1976, Myhrer 1975). However, performance of CA1 animals on complex spatial tasks is not necessarily impaired but rather depends on whether the task was learned before or after the operations. Specifically, postoperative performance on an 8-arm radial maze is unaffected if the task is learned before the operations, but impaired if the maze is learned postoperatively (Jarrard 1978a, b). Acquisition of a Y-maze spatial task is normal in both CA1 and complete hippocampally damaged rats, but both groups are impaired in reversal learning (Jarrard 1976). In other tasks, such as two-way active avoidance, passive avoidance, and operant tasks, performance of rats with CA1 lesions is like that of controls (Jarrard 1976, Jarrard & Becker 1977). These results do implicate the CA1 cells as being important in exploration and in the acquisition of spatial tasks, but CA1 cells are apparently not necessary for correct performance of a spatial task if it is learned before the operations. Since the CA1 cells are seen by O & N as being important in *both* the construction and storage of cognitive maps, the data only partially support the theory. As suggested by the authors, it may be that the CA3 cells are more involved than CA1 cells in mapping, but it is still surprising that interrupting the hippocampal circuit by damaging CA1 cells has little effect on retention of complex place learning.

The usefulness of the theory in thinking about lesion experiments, as well as some limitations, can perhaps best be pointed out by analyzing the results of several experiments. In one study rats were trained preoperatively in a Y-maze spatial alternation task, operations were carried out, and rats with extensive damage to hippocampus and controls were (a) retrained postoperatively, (b) tested with delays between trials, and (c) tested with and without interpolated activity during delays (Jarrard 1975). Rats with hippocampal lesions were impaired in postoperative testing (as predicted by the theory when the locale system is damaged) but they were able to relearn the task with savings (by using a taxon system?). The theory predicts that performance of animals relying on a taxon hypothesis will be significantly influenced by intertrial interval and by interference. Performance of both hippocampals and controls was impaired at longer delays but to an equal extent; however, hippocampals were more affected by the interference condition. In a second experimental procedure, animals were trained on a radial-arm maze to choose a limited subset of arms (Jarrard 1978b, Olton & Papas, in press). In our study rats learned before the operations to choose four arms that were consistently baited on an 8-arm maze and the animals were then retrained postoperatively using the same procedure. Although the immediate postoperative performance of hippocampals would suggest they were "lost in space," with subsequent training they did learn to choose only the baited arms. Hippocampals were still impaired, though, since they continued to reenter previously baited arms that had already been chosen on that trial (and thus no longer contained food). These results have been variously interpreted as evidence that hippocampal lesions result in increased susceptibility to interference (Jarrard 1978b) and a working memory impairment (Olton & Papas, in press). The spatial mapping theory would probably say that hippocampals were forced to employ a less efficient taxon hypothesis, and as a result experienced more interference between correct arms. Thus, it appears that the mapping theory predicts most of the results of experiments like the above but it does make one wonder if it is possible to design an experiment that would be a critical test of the theory.

The usefulness of a particular theory must be judged with respect to its value in integrating known facts and generating further experiments. Not only does the O & N theory integrate a diverse literature, but it makes definite predictions that can be tested. In this regard, the cognitive mapping theory would seem to be an especially promising one.

by **Raymond P. Kesner**

Department of Psychology, University of Utah, Salt Lake City, Utah 84112

Hippocampus and memory

O & N attempt in their book to integrate the vast literature on hippocampal function and incorporate the extant data within a single

theoretical framework. The introduction to their theory, which is boldly stated in the title of the book, *The hippocampus as a cognitive map,* includes an excellent discussion of the philosophical foundations of space. The anatomy and electrophysiological properties of the hippocampus are presented in a scholarly fashion. The effects of hippocampal lesions on behavior and correlates of hippocampal electrical activity have been satisfactorily integrated within the proposed theoretical framework. The extension of the theory to human data, however, is somewhat weak and the electrical stimulation chapter is poorly presented.

Since it is impossible to review every aspect of the book, I will restrict my comments primarily to the proposed role of the hippocampus in the encoding, storage, and retrieval of spatial information. There have been over the years a number of theoretical approaches associated with an analysis of the hippocampus and mnemonic function. The emphasis has been to assign to the hippocampus the role of mediating specific processes associated with information processing, including attention (Douglas and Pribram 1966), encoding (Butters and Cermak 1975), consolidation or transfer from short- to long-term memory (Milner 1968a), and retrieval (Warrington and Weiskrantz 1973) of information. O & N, however, present a new approach indicative of a changing emphasis, with an interest in the structural representation of memory. They suggest that the hippocampus encodes and contains the memory representation of absolute space. Their approach assumes that memory has a multidimensional structure with many neural systems contributing specific components or attributes to the total memory representation. They propose that the encoding of a cognitive map occurs within a "locale system" which is characterized by the storage of absolute spatial attributes of specific events within the hippocampus, i.e., the hippocampus encodes specific stimuli representing places or relations between places independent of the subject's own body schema. The encoding of all other attributes of an event becomes the property of a "taxon system" and presumably occurs in other parts of the central nervous system. The "taxon system" would include the storage of relative spatial attributes, which depend upon accurate assessment of one's body orientation in space. The consequence of this division is that the "locale system" is quite specific, with a primary emphasis upon the encoding of spatial attributes of specific environmental contexts. It appears to be somewhat akin to Tulving's notion of episodic memory (Tulving 1972), except that there is little emphasis on the encoding of time and the authors assume low rather than high interference between similar events. The "taxon system," on the other hand, appears to be nonspecific, vague, and based primarily on remaining capabilities of hippocampally lesioned subjects. It appears to be somewhat akin to one's knowledge of the world (Tulving's notion of semantic memory).

There are two major issues with respect to the "locale system" that I would like to discuss further. First, there is the question of whether one can assign the encoding of space to only one neural region, namely the hippocampus.

There are other neural regions that contribute to the encoding of space like frontal cortex, caudate, parietal cortex, and the superior colliculus-pulvinar-visual cortex system. O & N do suggest that the parietal cortex encodes space, but only relative space. Unfortunately, others have suggested that the parietal cortex encodes absolute space with relative space delegated to the frontal cortex or superior colliculus-pulvinar-visual cortex system (Dick 1976; Butters, Soeldner, and Fedio 1972; Critchley 1953; Pohl 1973; Semmes et al. 1963). The interrelations among the hippocampus, frontal, parietal, and visual cortex are unclear, but the exclusive emphasis upon the hippocampus as the only region associated with memory representation of space (i.e., cognitive mapping) might lead to a relatively simplified view of the neural circuitry mediating the encoding of space. It would be helpful if the authors had spelled out more precisely the contribution of these other neural regions.

Second, there is the question whether the hippocampus encodes only one attribute of a specific event, namely the spatial attribute. O & N stress almost exclusively the involvement of the hippocampus with the encoding of absolute space. In the chapter on human memory, they do allude to the possibility that the hippocampus might also encode time, but they don't elaborate on this further.

O & N maintain an overall interest in space primarily because of their strong interest in the lesion and electrophysiological data, which was collected within an S-R learning framework. Since this learning framework has little concern for the constructs of memory and time, it is not surprising that O & N do not discuss extensively the possibility that the hippocampus also encodes time. As a consequence, the electrical brain stimulation and human literature, which is based on an information processing framework with a special emphasis upon time, does not match well with the exclusive notion of hippocampus and space. I have recently presented data in support of the hypothesis that the hippocampus encodes attributes associated with *both* time and space (Kesner 1980).

Extending O & N's theory to include the encoding in memory of both time and space should lead to a more integrated theory of hippocampal function.

by **John W. Moore**

Department of Psychology, University of Massachusetts, Amherst, Mass. 01003

The hippocampus and informational salience

My predisposition toward *The hippocampus as a cognitive map* was decidedly negative. Earlier publications by the authors contained statements that seemed simply outrageous (e.g., the hippocampus is not involved in processing temporal information; Black, Nadel, & O'Keefe 1977), or that suggested self-serving blind spots toward contrary evidence (see Solomon 1980). In addition, the crucial distinctions between cues and places were hard to understand or accept, a fault corrected with the publication of Black et al. (1977). Moreover, I had been trained to distrust the notion of cognitive maps. Rereading Tolman did not erase this distrust but instead reinforced my belief that, while such notions may have had heuristic value at one time in the history of psychology, they were no longer to be taken seriously. In this I was quite wrong, of course, as cognitive psychologists and others have devoted a good deal of thought and effort in recent years to precisely this class of problems, i.e., how spatial information is represented mentally.

O & N have not undertaken a flippant attack on comfortable modes of thinking but rather, it seems to me, a commendable effort to bridge the gulf between present-day cognitive and physiological psychologies. Furthermore, the book corrects the extravagances of earlier treatments of the theory by being not only thorough and scholarly, but temperate and generally plausible as well. By way of specifics, while I found the last two chapters, dealing with extensions of the theory to human language and amnesia, unconvincing (although I applaud the attempt), the lesion literature from a variety of animal paradigms is treated fairly and intelligently. Unavoidably, some relevant recent works could not be included. I doubt if the electrophysiological evidence will fail to hold up, as others have already noted the existence of place and misplace units, for example.

The heart of cognitive map theory is a brilliant stroke. It is the assumption that the taxon system is more subject to interference than is the locale system. This argument neatly reconciles the theory with the literature that suggests that hippocampally damaged animals are more interference-prone than normals. Hippocampally damaged animals are simply unable to employ the more efficient and less interference-prone locale system available to normals. Those who would challenge the theory should direct their attack here, because it is here that the theory will stand or fall.

Nevertheless, I remain dubious of any theory of brain and behavior that places so much reliance on cognitive psychology. By cognitive psychology I mean the notion that behavior is "under the control" of mental entities such as images, hypotheses, memories, or cognitive maps. Behaviorism assumes that internal and external stimuli control behavior. Cognitive theories assume that stimuli evoke mental representations. These, in turn, control behavior. If behavior is caused by mental representations and not by observable events, the structure of

the mind tends to become the object of analysis. Is this appropriate or desirable?

The Zeitgeist sides with the authors – the mind is back and physiological psychologists and behaviorists are advised to adjust accordingly. My adjustments have been to gravitate toward theoretical models that portray mental entities within a system of mathematical statements linking behavior to stimuli. Such models may be wrong, but they are less likely to be misunderstood or misrepresented than purely verbal statements such as a cognitive map theory. At a recent workshop on the role of the hippocampus in learning and memory convened at Williams College this past summer,[1] I presented the outlines of a model based on the assumption that the hippocampus is responsible for reducing the salience of elements in an attentional-associative network consisting not only of points in space, but cues and responses as well. By reducing the salience of these elements under precisely specified circumstances, the hippocampus *participates* in the ongoing development of cognitive maps that contain not only information about where things are located, but also about what follows what.

While alternative interpretations of hippocampal function proceed down the pipeline, *The hippocampus as a cognitive map* should maintain its influence in the field for many years to come. Too much original data, thinking, and toil went into it for it to be dismissed lightly. In addition, it has already provoked enough thought and research so as to ensure its place as a milestone.

Note

1. Workshop on the Role of the Hippocampus in Learning and Memory, Williams College, Williamstown, Mass., June 25–26, 1979, J. W. Moore and P. R. Solomon, organizers. The authors of *The hippocampus as a cognitive map* participated in this workshop.

by **Arthur J. Nonneman**

Department of Psychology, University of Kentucky, Lexington, Ky. 40506

Time: a fourth dimension for the hippocampal cognitive map

Somewhat more than a year ago I first saw an advertisement from Oxford University Press announcing a forthcoming volume by John O'Keefe and Lynn Nadel which promised to propose, once again, that the mammalian hippocampus serves as the essential physiological substrate for cognitive-mapping functions. My initial reaction to the ad was much the same as that of one of my graduate students, who, upon seeing this volume on my desk, remarked, "They certainly have gotten a lot of mileage out of that idea." Indeed they have. But their current treatment of the topic, in the form of this book, is far more complete, more thoughtful, and more persuasive than any of their previous presentations. It is just possible that they are essentially correct. In any event, as a result of having read this book, I have been persuaded to rethink my own notions about hippocampal function. Until now, my thinking *on* the subject has been lodged somewhere between the "Palisades of Persistence" and the "Channel of Attention," as the authors characterize the perseveration and attention hypotheses respectively (p. vii).

As in most attempts to substantiate new theoretical formulations, the authors are guilty of some excess. For example, their claim in the accompanying précis for "the success of the major prediction of the theory that *all* [italics mine] of these [hippocampal lesion] data can be interpreted as a loss of place learning and exploratory behavior" is clearly an overstatement. The theory, in its present form, cannot easily handle all of the extant data. The authors themselves, in the preface (p. ix), plead guilty of a deliberate "chiaroscuro painting of the data" as a necessary first step in developing the theory.

One area in which the authors admit that the supporting data are thin is the human clinical realm. The changes in emphasis in this section (i.e., event-specific memory) and the stretching of the cognitive-map concept to include "semantic maps" in the left hippocampus are likely to elicit from some readers charges of special pleading. In fact, when one of the authors presented a preliminary attempt to explain the human amnestic syndrome on the basis of cognitive-map theory (Nadel 1977), a member of the audience commented on the "incredible chutzpah" of the presentor. But it is exactly this extension (refinement?) of cognitive-map theory that offers the possibility that a unitary explanation of hippocampal function may be possible after all. At the same time, it points up the most serious weakness of the current theory and provides direction for further development and refinement.

In terms of refinement, it is imperative that the characteristics of a semantic map be specified in the same explicit manner as spatial maps were characterized in the early chapters of this book. Merely talking about semantic maps and implying that they are basically the same as spatial maps except for the nature of their inputs is not sufficient. In addition, although the book suggests at several points that the hippocampus provides the essential substrate for storage of information in a spatiotemporal context, the temporal aspect of the locale system receives little attention except in the treatment of event-specific human memory. I see this as a general problem with the current formulation that is not restricted to the particular issue of human clinical effects. For example, in a recent *BBS* target article, Olton, Becker and Handelmann (1979) present data which clearly indicate the importance of a temporal factor in the functioning of rat hippocampus. This temporal factor has little or no relation to the temporal changes involved in intertrial or interstimulus interval manipulations. As O & N suggest, any effects of manipulating these aspects of time can be readily dealt with on the basis of taxon systems. Rather, the critical temporal factor, as I conceptualize it, provides a fourth dimension to the Euclidean cognitive map around which this book is built. It provides for the coding of discrete events in both time and Euclidean space. Thus, I would take exception to the authors' suggestion that different cues encountered on different occasions in the same part of the environment will necessarily make the place representation richer but not stronger. According to my view, such cues encountered on separate occasions could be entered into different "places" in a cognitive map with four dimensions.

In concluding, I acknowledge the fact that O & N have been thinking about this problem for a considerably longer time and at considerably more depth than I have. Therefore, there may be a serious logical flaw in the suggestion I have just made. If so, I am afraid there is a serious problem in expanding the cognitive-map theory to account for hippocampal function in a unitary fashion. Also, if there is a basic flaw in my logic, then I have to admit that I really do not understand what the authors mean when they refer to the "spatial-temporal context" of context-specific event memories. On the other hand, if my view has merit, then perhaps the next edition of this book will begin with a chapter retitled, "Remembrance of *events* past: a history of theories of *time and* space."

by **David S. Olton**

Department of Psychology, Johns Hopkins University, Baltimore, Md. 21218

Inner and outer space: the neuroanatomical bases of spatially organized behaviors

Space has been a lot like the weather; many have talked about it, but few have done anything about it. This state of affairs is rapidly changing, and the book by O & N should make a major contribution to the development of our thinking about the organization of spatial behaviors and their underlying neuroanatomical systems.

The usefulness of the cognitive mapping theory of hippocampal function depends on the ability to take the conceptual distinctions (route or map, taxon system or locale system) and apply them to particular experimental procedures. I have difficulty doing this in some instances. For example, consider the following experimental procedure, one which has commonly been used with rats in mazes. The apparatus is a maze, as illustrated in Figure 1. The four arms are labelled north, east, south, and west, respectively. In the initial training procedure, illustrated by the top diagram of Figure 1, a rat is placed in the south arm and given a choice between the east arm and the west arm (the north arm is closed). Food is provided at the end of the east arm. The rat learns to turn right at the choice point and enter the east arm. After it consistently makes this response on every trial, it is given a

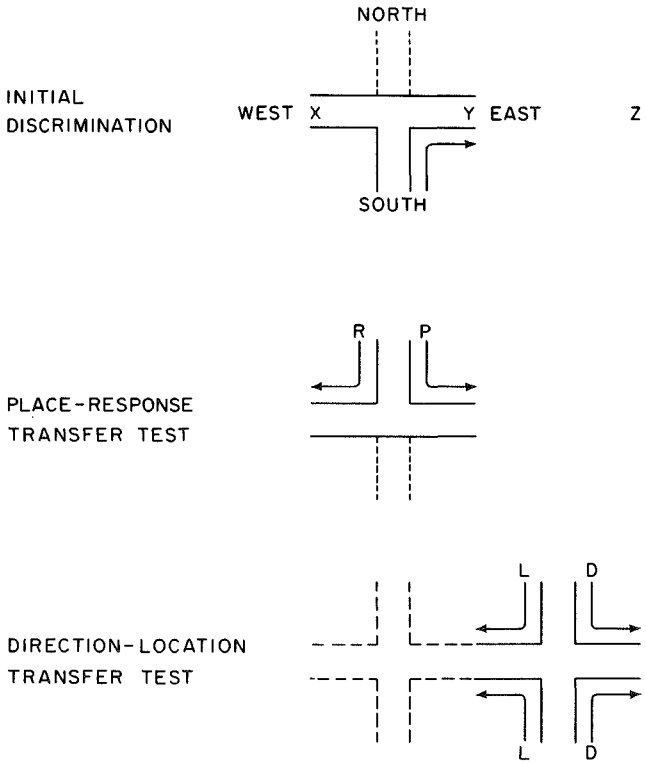

Figure 1 (Olton). An illustration of the initial discrimination (top diagram) and subsequent transfer tests (middle and bottom diagrams) discussed in the text.

"place-response" transfer test (Olton 1979, Restle 1957), illustrated by the middle diagram of Figure 1. The maze remains in the same location in the room but the rat is placed in the north arm to begin a trial; it is again given a choice between the east and west arms (the south arm being closed). If on this transfer test it turns right at the choice point and goes to the west arm, it is said to have used a "response strategy" to learn the original discrimination because in the transfer test it makes the same response (turn right) as in the first but goes to a different place (west arm). If in the transfer test the rat turns left at the choice point and goes to the east arm, it is said to have used a "place strategy" to learn the original discrimination because in the transfer test it goes to the same place (east arm) as in the first but makes a different response (turn left) at the choice point.

Presumably a response strategy is an example of the route/taxon system. But is a place strategy an example of the map/locale system? In particular, I'd like to distinguish between a "direction strategy" and a "location strategy," both of which might appear as a place strategy in the place-response transfer test just described. The difference between a direction strategy and a location strategy is similar to that between a line and a point. A direction strategy specifies only the direction of movement (i.e., towards the window on the east wall, away from the light bulb on the west wall, etc.), while a location strategy specifies a particular point in the room (i.e., 2 meters from the window on the east wall, 1 meter from the stool behind the start arm, etc.). The relative importance of these two strategies can be evaluated in another transfer test, illustrated by the bottom diagram of Figure 1. Here the maze has been moved to the right so that the west arm ends at the same place in the room (point Y) where the east arm ended during initial training. If the rat learned the original discrimination using a direction strategy, then in the transfer test it should turn in the same direction (towards the east wall) at the choice point and go to a different location in the room (point Z). If the rat learned the original discrimination using a location strategy, then in the transfer test it should go to the same location in the room (point Y) and turn in a different direction (towards the west wall) at the choice point. These results should be found when the rat is started from the north arm and from the south arm, eliminating the use of a response strategy.

As I understand the O & N theory, a location strategy requires the map/locale system. But does the direction strategy? Let's assume that the answer is "no," and that the direction strategy is an example of orientation, which is a function of the route/taxon system. If this is the case, then a rat might exhibit a place strategy on a place-response transfer test using either the route/taxon system (direction strategy) or the map/locale system (location strategy). To my knowledge, virtually all maze procedures that can be solved by a location strategy can also be solved by a direction strategy; only one published experiment has used the appropriate transfer tests to distinguish between these two alternatives (Blodgett, McCutchan, and Mathews 1949). If such is the case, then the data are not available to determine for any given task whether the animals used the route/taxon system or the map/locale system, and there is no basis for predicting the way in which the hippocampus participates in these test procedures. Alternatively, of course, the answer to the question raised at the beginning of this paragraph might be "yes," that the direction strategy is an example of the map/locale system. In that case, how does one distinguish between the use of a direction strategy as I have defined it and of an orientation strategy as O & N use the term?

Many other people are certain to be interested in understanding the practical applications of the cognitive map theory and the ways in which experiments can be designed to test its validity. O & N can help a great deal by taking a number of laboratory tasks, indicating whether they require the route/taxon system or the map/locale system, and the criteria that were used to make this distinction.

I also have a few questions about specific aspects of the theory. Do the authors consider the route/taxon system and the map/locale system to be mutually exclusive, all-or-none systems, or do these systems represent the endpoints of a continuum? If the former is the case, then at any given time the animal (and his brain) is using either one system or the other but not both, and experimental procedures are placed into one of two categories. If the latter is the case, then an animal might be using some aspects of the route/taxon system simultaneously with some aspects of the map/locale system, tasks should vary in the extent to which they engage the two systems, and experimental procedures are arranged along two dimensions.

Why is the route/taxon system considered so different from the map/locale system? Some of the distinctions are not intuitively obvious. For example, some people (Nadel, 1979b; Suzuki, Augerinos, and Black, in press) have suggested that the radial arm maze task (Olton and Samuelson 1976) is an example of a map/locale system. But there is substantial interference found during performance of that task (see review in Olton 1978; Olton, Becker, and Handelmann 1979), an outcome which according to O & N is characteristic of the route/taxon system, not the map/locale system. What were the criteria the authors used to determine what properties should be given to the route/taxon system and to the map/locale system?

I understand why the theory predicts that hippocampal lesions should interfere with performance in tasks that require the map/locale system. Yet O & N suggest that lesions will interfere with performance in tasks that require the route/taxon system, which presumably is not a hippocampal function. Does the hippocampus then have two functions, one in route/taxon tasks and a second in map/locale tasks? Furthermore, how is a comparison of the performance of animals with hippocampal lesions in route/taxon tasks and in map/locale tasks relevant to the predictions of the theory?

The authors are aware that hippocampal lesions come in many different forms, sizes, and locations, and that both the magnitude and the duration of the behavioral changes observed following these lesions vary considerably. Do they have any comments about the relevance of these variables to the predictions of their theory?

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by **Steven Pinker**

Center for Cognitive Science, Massachusetts Institute of Technology, Cambridge, Mass. 02139

Mental maps, mental images, and intuitions about space

When the subject of cognitive maps is brought up, talk of mental images seldom follows far behind. Many people use the terms as synonyms, and O & N at times seem to identify one with the other (e.g., pp. 385, 419), at other times keep them distinct (pp. 30, 390). Our knowledge about the properties of images has been increasing at a rapid rate (see Kosslyn et al. 1979: this issue of *BBS;* and Shepard 1978, for recent reviews), and it is now possible to relate the imagery system to the various cognitive mapping systems in a fairly precise way. I argue here that (1) visual images are distinct from O & N's cognitive maps and in fact cut across their locale/taxon (map/non-map) distinction; (2) the imagery system interfaces with other representations that are more closely tied to the maps that O & N describe; and (3) identifying mental representations of space with visual images may have engendered the traditional philosophical views about spatial representations that O & N eschew.

Visual images are indeed "maplike" in that they preserve interval information about all interpoint distances in the imagined scene (see Kosslyn, Ball, & Reiser 1978, and Pinker & Kosslyn, in press, for evidence to this effect, superseding that cited by O & N). People can not only scan mentally along the shortest straight path linking an arbitrary pair of imagined objects, but when an object is mentally "moved" to a new location, people can scan directly to and from that new location (Pinker & Kosslyn 1978; see also Pinker 1979). These facts are consistent with "picture theories" of imagery (e.g., Kosslyn et al. 1979: this issue of *BBS*) in which images are treated as "filled-in" regions of an internal spatial array. As such, these findings take imagery out of the domain of the "taxon" system, which is characterized by inflexible routes linking locations and by an absence of a spatial medium in which location information can be represented.

Nonetheless, images are not *true* maps in O & N's sense either, since they seem to correspond to a glimpse of a scene from a particular vantage point. Pinker and Finke (in press) review evidence suggesting that images preserve viewer-specific perspective effects such as changes of projective size with distance, changes of projective shape with orientation, alignment and concealment relations of objects at different distances, and so on. Thus images occur in *egocentric* and not in *absolute* or *unitary* spatial frameworks, and do not qualify as maps in O & N's locale system. When we consider as well the evidence that images occur in the primary or secondary visual cortex (Hebb 1968; Finke & Kosslyn, in press; Finke & Schmidt 1977), we are forced to conclude that visual imagery constitutes a representational system that is distinct from O & N's hippocampal mapping system.

Still, we seem to have no trouble forming images that correspond to the layout of some portion of the world as we know it. How, then, might the imagery system tie in with the mapping system? Kosslyn et al. (1979: this issue of *BBS*) have proposed that images are constructed from information stored in long-term memory in a more abstract format (specifically, as lists of coordinates of points to be "filled in" in the spatial array), a theory that is consonant with some of O & N's suggestions. Pinker and Kosslyn (in press) and Pinker (1979) also argue that this "deep" level of image representation preserves the intrinsic, three-dimensional (3D), perspective-independent shape of an object, and is involved in 3D mental transformations of objects (see Shepard & Metzler 1971; Pinker & Finke, in press; Pinker & Kosslyn 1978, in press) and in the recognition of 3D shapes (see Marr & Nishihara 1978). Since, as O & N argue, object recognition is intimately wedded to the use of cognitive maps, it does not seem implausible that this "deep" or "intrinsic 3D shape" level of representation is also what feeds into the hippocampal mapping system. This would give us at least three types of internal representations of space: (1) O & N's putative hippocampal mapping system: an abstract spatial framework whose locations may contain labels indexing the objects that occupy the corresponding locations in the world; (2) an "intrinsic 3D shape" level of representation, storing the information necessary to recognize objects by their 3D shape; and (3) the visual imagery system (or more precisely, the "surface array" used to display images; see Kosslyn et al. 1979: this issue of *BBS*), depicting the visual perspective appearance of an object or scene from a given vantage point.

A final observation: O & N note that many brilliant thinkers have insisted that the mental representation of space is relative and/or piecemeal. When I read the quotations to this effect that O & N cite in their historical introduction, I was struck by how apt they were as descriptions of visual images, especially in light of the research I cited characterizing images as perspective views of scenes. Thus we have Spencer writing that "every conception of space which can be formed by a single mental act is limited to such portion of space as we can have experience of at one time," and James noting that "most of us are obliged to turn around and drop the thought of space in front of us when we think of that behind"; Poincaré seems to make a related observation. Given that creative and talented people are notorious for their claims of experiencing or "thinking in" visual imagery (McKellar 1965, Shepard 1978), it seems plausible that these writers were describing their *visual images* but drawing conclusions about the *entire* human ability to represent space mentally. O & N wisely reject these conclusions, for as they have shown most effectively, the mental representation of space is a many-splendored thing.

by **James B. Ranck, Jr.**

Department of Physiology, Downstate Medical Center, Brooklyn, N.Y. 11203

On O'Keefe, Nadel, space and brain

I read an early version of the manuscript of *The hippocampus as a cognitive map* in 1972. I read an almost final version in June 1977. The following is a letter I sent to O'Keefe and Nadel then.

Dear John/Lynn:

I have just finished reading your book, and it has been a thrilling experience. The book is a major and excellent work in many respects. The two basic ideas–the existence of spatial maps, and their manifestations in the hippocampus–are of tremendous importance. While the spatial map idea is not new, your extensive development of it makes it yours. The idea that they are in the hippocampus is brilliant. Others–Greene, Mahut, Olton–had suggested spatial functions for the hippocampus, your version of it is distinctive and the degree to which you develop it is untouched. All ideas have antecedents, but you have made a major stride beyond anything before.

I am not about to say that your ideas are true, but I suspect "the truth" is probably something very much like that. What you say is probably closer to the way it is than anything else, and it gives us a powerful conceptual entry with which to work.

The scholarship is overwhelming. I have taken particular care with the anatomy, physiology, and single unit data (chapters 3 and 4) with which I am in a position to judge the scholarship and it is almost always an excellent presentation accurately stated. . . .

The scope of the book is remarkable. No one else would even think of trying to cover all the ground you do. Few people will be able to find the whole thing comprehensible, for few have that much scope, and you do not try to write any section for the reader naive in any part. I think, however, you do a good job of making it comprehensible to someone who just knows a little about any part. I felt I understood everything you said. I doubt if there is anyone who can read the whole thing critically.

Some of the parts I liked best were discussions of approaches, especially that for lesions. The book is just filled with wise little comments. . . . The book by Hutt and Hutt (1970) says many of the same things you say. It is one of my favorite books.

It has been personally exciting for me to see the ideas, the data and the book develop – and to understand it better myself. I am proud to have contributed some of the data. . . .

I am obviously much more enthusiastic about the book now than I was about the 1972 one (which I liked very much). I think this is

because the book is now much better, the data is stronger – and I have grown wiser.

My own thinking is expressed in the grant proposal I sent each of you. Much of it takes off from or revolves around your thinking. It is your style to be much more speculative than I feel comfortable with. However, I am impressed that almost all your speculation is testable. Even when it gets pretty wild – as in the cellular model – it is testable and not just a flight of fancy.

One of the greatest contributions of your book may be to show that cognitive concepts can be handled concretely in organic terms and in terms of cellular mechanisms.

. . . I think Pat Wall deserves tremendous credit for sticking by you while you worked out your unorthodox ideas in unorthodox ways. I am sending him a copy of this letter as a statement of thanks and appreciation that he stuck by you. . . .

After being as complimentary as this, I feel I can criticize as fussily and vigorously as I like.

As I have argued elsewhere, one cannot properly argue that because one finds a particular behavioral correlate of a cell, the function of that cell is to produce that behavior. The receptive field of a neuron in the lateral geniculate is, roughly, a spot of light or an annulus of light, but the function of the lateral geniculate is not to respond to or to sense spots or annuli of light. The "function" of a part of brain is the transformation which occurs in it, that is, the changes in behavioral correlates between inputs and outputs. The data on firing of single neurons and place in neurons of Ammon's horn gives us a hunch that space has something to do with the function of the hippocampus (indeed, this finding was what got O & N thinking of spatial maps in the first place), but is not evidence for spatial maps. We must know the behavioral correlates of entorhinal cortex and dentate before we can start to argue the function of the hippocampus from single cell data.

The work of Berger and Thompson (1978a), Deadwyler et al. (1979) and Segal (1973) seems to indicate that under some circumstances hippocampal cells fire under conditions which are not clearly spatial; however, these workers should try to look for spatial features.

It is remarkable that the firing of hippocampal neurons has been reported to be related to so many different things. This may be because the neurons really do change the relationship of their firing to behavior (i.e., "learning" occurs), or it may be because the real relation of the firing of the cell to behavior is something more fundamental than anything that has been described yet, and we are only looking at a particular manifestation of it in each case.

by **Larry R. Squire**

Veterans Administration Hospital, San Diego, California, 92161, and *the Department of Psychiatry, University of California School of Medicine, La Jolla, Calif. 92093*

The hippocampus, space, and human amnesia

This comprehensive book develops the argument that the hippocampus stores information about the spatial environment. Although it was derived originally from lesion and unit recording studies in rats, the argument has been developed in considerable detail so that it might extend as well to human brain function. The book contains excellent reviews of hippocampal anatomy and physiology and a complete, best-ever presentation of the voluminous animal lesion literature. This note focuses on the spatial hypothesis itself, and considers how well it fits the known facts of human hippocampal function.

Information about hippocampal function in man comes primarily from the study of patients who have sustained bilateral or unilateral resections of the medial temporal lobe, including hippocampus (Corsi 1972, Milner 1972). In addition, transient temporal lobe dysfunction seems to result from ECT, electro-convulsive therapy (Inglis 1970, Squire & Slater 1978a), so that the neuropsychological effects of ECT may also provide useful information about hippocampal function. Patients with diencephalic damage (e.g., patients with Korsakoff syndrome [Talland 1965] and case N.A. [Squire & Moore 1979]) are often included in discussions of hippocampal function because their neuropsychological findings resemble in some respects the findings following medial temporal damage and ECT. Nevertheless, the relevance of these patients to problems of hippocampal function must be regarded as tenuous, and they will be considered here only for purposes of comparison.

The hippocampus as the substrate of specifically spatial functions. The hypothesis supposes that the hippocampus is concerned primarily with spatial information. Yet patients with unilateral or bilateral hippocampal damage and patients receiving ECT consistently exhibit amnesia. The amnesia is global in the case of bilateral hippocampal damage and bilateral ECT, and material-specific (i.e., affecting primarily verbal or nonverbal material) in the case of unilateral hippocampal damage or unilateral ECT. It seems doubtful that this memory defect can be considered spatial in any interesting way, since the defect includes memory loss for material that is not spatial in the ordinary sense. Thus, patients with right temporal lobe resections have material-specific defects in image-mediated verbal learning (Jones-Gotman & Milner 1978) and facial recognition (Milner 1968b), and the severity of these defects correlates with the extent of hippocampal removal. It is difficult to see what is spatial about visual images or faces. In order to apply the spatial hypothesis to these findings, one could perhaps make the concept of space more abstract so that it somehow encompasses faces, image-mediated learning, and the variety of things that amnesics cannot remember; but at this level of abstraction, it would be difficult to distinguish a spatial hypothesis from a memory hypothesis.

The fact that the well-studied, globally amnesic patient H.M. has a defect in maze learning (Milner 1965) has been cited as support for the spatial hypothesis. While it cannot be disputed that maze learning requires spatial abilities, this defect is easier to understand as an example of global amnesia. Thus amnesic patients do have difficulty learning mazes, finding their way around, and reproducing floor plans of their houses. But they also have difficulty remembering a variety of other things, including temporal information, tactual impressions, colors, sounds, and odors. The defect is not peculiarly spatial.

Whereas patients like H.M. and N.A. can perform normally on a variety of perceptual and other cognitive tests, it could be argued that they have a spatial impairment superimposed on amnesia. To identify it, however, would require something different from asking patients to learn mazes or reconstruct floor plans. That is, since amnesic patients have difficulty retaining almost any kind of information across a delay, most spatial tests would not permit a spatial defect to be distinguished from a memory defect. Amnesic patients do, however, have normal immediate memory (e.g., for digits and for block tapping sequences [Corsi 1972]). A spatial defect might therefore be searched for by administering a spatial task that could be performed entirely within the span of immediate memory.

The hippocampus as a storage site of information. The spatial hypothesis also proposes that information about the world is *stored* in the hippocampus. For man, this notion has been elaborated to the effect that the left hippocampus stores linguistic information about the world, and the right hippocampus stores spatial-temporal information. These ideas lead necessarily to the prediction that left or right hippocampal removal in man should destroy some kinds of information about events or places that have been learned previously.

In recent years this possibility has been tested using objective tests of remote incidents, asking about famous events, faces, or former television programs. Case H.M., who became amnesic in 1953, was profoundly impaired in his ability to name famous faces that came into the news in the 1950s or 1960s, yet he scored as well as or better than normal for faces that came into the news during the 1920s, 1930s, or 1940s (Marslen-Wilson & Teuber 1975). Case N.A., who became amnesic in 1960, was profoundly impaired in his ability to provide details about famous events or former television programs that occurred in the 1960s or 1970s, but scored as well as normal for the 1950s (Squire & Slater 1978b). Finally, the retrograde amnesia associated with ECT is typically graded, such that events that occurred a few years prior to ECT are forgotten, and events that occurred many years prior to ECT are not affected (Squire, Slater & Chace 1975;

Squire & Cohen 1979). Taken together, the evidence from three different types of amnesic patients indicates that detectable loss from the premorbid period does not occur, except for the few years just prior to the onset of amnesia.

Even if one questions that the critical structure in human bitemporal amnesia is the hippocampus itself (Mishkin 1978, Horel 1978), it is still the case that damage to the hippocampus in man is not associated with a detectable loss of information from the premorbid period; nor is hippocampal damage associated with any known defects of a spatial nature that are not associated with a detectable loss of information nature that are not readily explained as a more general memory problem.

The hippocampus and the formation of memory. Analysis of bitemporal amnesia and the amnesia associated with ECT has led to some specific ideas about the organization of memory processes and about how the hippocampus might be involved in them (Wickelgren 1979, Squire 1980, Squire & Schlapfer 1980). First, the finding that hippocampal damage produces a sharp discontinuity between premorbid and postmorbid memory (Marslen-Wilson & Teuber 1978) suggests that the hippocampus is involved in new and recent memories, not old ones, and that memory storage occurs elsewhere in the brain. Second, the finding that ECT produces a gradient of retrograde amnesia selectively affecting memories formed in recent years (Squire, Slater & Chace 1975, Squire & Cohen 1979) indicates that memory gradually becomes resistant to disruption for a period of years after learning. The change that occurs as time passes could provide a basis for the formation of schemata and for the gradual reorganization of memory (Bartlett 1932, Norman & Rumelhart 1975).

The fact that the retrograde amnesia is transient (Squire, Slater & Chace 1975, Squire, Slater & Miller 1980) suggests that, while resistance is developing, the hippocampus might be required for effective retrieval of memory. By this view, as time passes after learning, resistance develops, forgetting occurs, the structure of memory is changed, and retrieval of information gradually comes no longer to require the hippocampus. The hippocampus might provide input to cortex necessary for restructuring to occur, or could protect memory from interference that would ordinarily disrupt this process. It is also possible, though perhaps less parsimonious, that information is stored initially in the hippocampus and then gradually reconstituted elsewhere in a form that is resistant to disruption.

The hypothesis that the hippocampus has spatial functions might be made relevant to this fundamental idea that the hippocampus must relate in an orderly way to other brain regions that store memory. Such a relationship would presumably require spatial information about where memories are located. Although the available data do not permit us to be very specific about this relationship, they do suggest that the hippocampus provides an organizing mechanism that allows memories to become resistant to disruption, restructured, and thereby enduring. This function of the hippocampus may also permit the effective retrieval of memories while the gradual process of restructuring is under way.

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by **L. W. Swanson**

Department of Anatomy and Neurobiology, Washington University School of Medicine, St. Louis, Mo. 63110

The anatomy of a cognitive map

Only someone with the wit and philosophical insights of Bertrand Russell could provide an incisive critique of O & N's ambitious and interesting attempt to resolve an important aspect of the mind-body controversy. I certainly cannot, though I think it unfortunate to conclude sweepingly that the hippocampus constitutes a built-in neural memory system providing an objective spatial framework (Euclidean in nature) within which the items and events of an organism's experience are located and interrelated, without considering the development of Descartes's ideas before launching into Newton and Kant. But this is quibbling. The main issue raised by the book is whether philosophy and neurobiology can be successfully integrated, and I am not convinced that they can be. As the chapters on the anatomy and physiology of the hippocampus make clear, it is not even possible at this time to explain place units in terms of known anatomical circuitry. While it may be true that different units in Ammon's horn preferentially fire when a rat is in a specific part of its environment, there is no evidence that the units are wired together in a way that provides a topographically ordered map in the usual sense of the word.

The authors present a structural model of the "cognitive map" that is based on what was known about the anatomical and physiological properties of the hippocampus in 1976. It is obvious, however, that our current understanding of the anatomical organization of the hippocampus contributes virtually nothing to an understanding of the structural and functional architecture of a cognitive map. In the final analysis, however, this has little bearing on the hypotheses advanced by O&N, which are primarily physiological in nature, and may or may not provide an adequate framework in which to interpret the effects of hippocampal lesions. Nevertheless, since they provide an anatomical model for the cognitive map, the major conclusions of their synthesis of the literature deserve comment.

First, it is clear that for a cognitive map to function properly, specific information from each sensory system must reach the hippocampus. The authors point out that pathways from each primary sensory cortical field reach the hippocampus in the monkey. However, they fail to note that: (1) sensory information that eventually reaches the hippocampus appears to be "polysensory" in nature, since the pathways converge on single cells in association cortical regions first; and (2) despite several attempts, similar pathways have not been demonstrated in rodents. In fact, the major connections of the hippocampus in the rat are with the cingulate gyrus, the anterior thalamic nuclei, and the hypothalamus, which are most commonly associated with homeostatic functions.

Second, the authors view hippocampal circuitry in terms of a series of narrow lamellae, stacked together like a roll of coins, along its longitudinal axis. This is certainly true for the mossy fiber projection of the dentate gyrus upon Ammon's horn, but it is now clear that other intrinsic connections provide for rather widespread divergence. This is particularly true for the bilateral projection from field CA_4 to the dentate gyrus, but is also evident in the projection from field CA_3 to CA_1, and in the perforant path from the entorhinal region to the dentate gyrus and Ammon's horn. The anatomical evidence now suggests that while the hippocampus has a broadly lamellar organization, its subdivisions are richly interconnected along the longitudinal axis by both associational and commissural connections.

And third, the authors assume that the pyramidal cells in a given field constitute a "large matrix of logically similar cells." Recent evidence based on the use of retrograde transport methods makes this seem unlikely since, for example, only a fraction of the cells in field CA_3 project to the septum, or to the hippocampus on the other side.

Despite such caveats, this unique book is sure to stimulate interest in the most enigmatic part of the brain as its insights are digested in the years to come. It contains a thorough and insightful review of the literature, and presents clearly a hypothesis which is sure to stimulate a great deal of fruitful research.

by **Garth J. Thomas**

Center for Brain Research, University of Rochester, Rochester, N.Y. 14642

Cognition, memory, and the hippocampus

On first reading the O&N book I found it interesting and provocative. It represents a substantial forward step toward realizing that the *behavior* of organisms is not directly caused by stimuli (environmental energies that fire receptor cells). The S-R approach has been around a

long time. It made a major contribution–to exorcise mentalistic animisms from our understanding of brain-behavior relations–but its view of neural function as related to behavior was inadequate. An opposing view, now of importance in behavioral neurobiology, regards the causes of behavior as hypothetical (at the moment) constructions that are "built" by the brain from information derived from multiple-inputs and which are integrated over time by memorylike capacities that cover a range of milliseconds to years. The point of view is old, especially in the more rationalistic and mentalistic psychologies: the organism responds, not in terms of stimuli but in terms of "cognitions" (*perceptions* and *knowledge* about the environment). The book by O&N represents an ambitious effort to spell out in neural terms what in mentalistic psychology is called *cognition*.

Incidentally, one can appeal to the concept of a cognition without positing an animistic "ghost in the machine"—to use Koestler's phrase. O&N propose no such "explanation" of organisms' capacity for responding in terms of spatial properties of the environment.

Ordinarily a "cognition" is postulated when the organism is first exposed to relevant stimuli (information-gathering phase), and then the *cognition* is called on to account for the transfer of ways and means, short-cuts, and detours that the organism is able to display when allowed to perform in a situation where it can *use* the information acquired in the earlier phase (the "latent-learning" paradigm). For example, Menzel (1978) observed that the chimp who was carried around and allowed to watch where the experimenter hid bits of food was able to find those bits of food later with a higher frequency than the uniformed chimps. Furthermore, the informed chimp went about its search in an efficient way that did not involve backtracking the specific route over which it had been carried during the acquisition phase. O&N would say that its brain had a "cognitive map" of the environment, built up during "exploratory" exposure (the acquisition phase, in this case), and furthermore, that the ability to construct such a map depended on the hippocampus.

At this stage of "the story," this reader began to balk. In the first place, many of the invertebrates, lower vertebrates, and birds often have a remarkable behavioral capacity to find their way around their spatial environments. To postulate from their behavior that they have a "cognitive map" might be very fruitful at the behavioral level. Yet they have no unequivocal hippocampal formation in their central nervous systems (alleged homologies–maybe!)

A second mildly negative reaction arose from the introduction dealing with the spatial concepts of philosophers and scientists of long ago. Establishing continuity with past thinkers who were working with inadequate information might be the scholarly thing to do, but, it seems to me, for the authors to imply that rats are little Kantians with a "category" of absolute space helps us little in understanding brain and behavior.

A more useful line of introductory speculation, which could be bolstered with some empirical fact, would, I submit, focus on the general biological problem that all locomotive animals had to "solve" the problem of guidance (more or less efficiently) as they moved around their environments, accumulating calories and doing other things necessary for them to pass through the "natural-selection filter" so as to contribute their unique samples of genes back to the gene pool.

One requirement is that the "information processor" (brain) must function quickly. An organism, to survive, does not have time to build a cognitive map by the slow accumulation of S-R associations by trial and error (Hull's "habit-family hierarchy"). To cope adequately, its cognitive map must, in a sense, comprise a *spatial constancy* (like the perceptual constancies) that can be constructed from multiple inputs during a brief "acquisition phase." For example, the cognitive map must represent the two billiard balls (mentioned by O&N) as constant in space. When viewed first from the "north," the blue one is *right* of the white one. When viewed later from the "south," the blue one is *left* of the white one. Obviously, use of only egocentric cues will not achieve constancy. (I would say "intraorganismic" because I don't think rats have egos.) The cognitive map must be built by taking into account not only information about present and recent locations of the observer, but also other spatial information available to sense organs such as the table and the spatial frame of the room.

In general, as with the perceptual constancies, the spatial constancy must be achieved quickly, even though it is sometimes crude. Organisms (including people) do get lost. Spatial behavior improves as familiarity with a given environment increases.

A third criticism that can be raised against the notion that the hippocampus is *the* substrate for cognitive maps comes mostly from work with contingently reinforced alternation. I would argue that the ability of organisms to alternate successfully depends on a very simple cognitive map (the location of only two significant places in the environment needs specification), and it depends crucially upon a time-binding (memory) characteristic of brain (the organism must "remember" *where* it was fed last!). Many years ago Jacobson and Nissen (1937) reported that the frontal lobes of primates were necessary for delayed alternation. Subsequent research has narrowed down the critical brain structure to the projection field of the mediodorsal thalamic nucleus (the "banks and depths of the sulcus principalis"). Similar observations have been made in rats (Wikmark, Divac, & Weiss 1973; Thomas & Brito, in preparation). My own research also indicates that the whole hippocampal formation is not involved (Thomas 1978). Lesions transecting the postcommissural fornix are without effect on spatial delayed alternation, but lesions in posterodorsal septum (precommissural fornix?) have a devastating effect on alternation behavior.

Clearly, more than (and not all of) the hippocampus is involved in constructing a spatial cognitive map. Part of the hippocampal formation may well be a *necessary* brain substrate, but I don't think there is yet enough data to say that it is *sufficient*.

Similar arguments can be directed against the "map interpretation" of the unit-recording studies that O&N find crucial. Important circuits for spatial behavior (and, by inference, the map) are located partially in the hippocampus, so it is to be expected that place-specific units will be located there. However, if other parts of the brain are significant in mediating spatial cognitive maps, then it seems likely that one would find place-specific units in those parts of the brain also.

Another weakness that I see in the theory concerns how to account for the plasticity that apparently characterizes the cognitive map. Research in my laboratory has repeatedly found a rather marked recovery of function after critical brain lesions. The recovered function is fragile and more easily disrupted than in normal rats by increasing the difficulty of the problem (lengthening the intertrial intervals), or by injecting irrelevant information into the hypothetical cognitive-map circuits (intertrial rotation; Thomas 1978). The apparent recovery also occurs in rats after lesions in medial frontal cortex, but the recovery is perhaps slower. Normal animals can "recover" from the random choices produced by sudden increases in intertrial intervals from about 5 sec. to 90 sec., but rats with posterodorsal septal lesions cannot recover when faced with the same kind of jump in intertrial interval from 5 to 90 sec. (Thomas & Brito, in preparation). It is my belief that the construction of a cognitive map by the brain does indeed involve the hippocampal formation, but the phenomenon of the "recovery of function" strongly suggests crucial involvement of other brain structures.

A substantial part of the book by O&N concerns a detailed and critical survey of much of the hippocampus-behavior literature, which the authors reinterpret in terms of their own hippocampus-cognitive map hypothesis. One can only agree with their frequent statement: Most of the cited data are not critical. Earlier experiments were often aimed at other hypotheses, and O&N's *post hoc* interpretations are, at best, speculative or suggestive.

A weakness of the theory presented in the book stems from its treatment of the time-binding (memory) capability of the brain required for an organism to build a spatial cognitive map (or any "cognition," for that matter). The problem does not go unremarked by O&N, but it perhaps needs more emphasis. The time-binding characteristic of brain function in its integration of the information necessary to build a cognition is still vaguely understood in neuroscience. We tend to postulate an animistic factor (a memory) suggested by an introspective

view of the "human condition." The time-binding characteristic would seem to be a critical consideration in the brain-construction of all cognitions, and it needs more attention.

O&N's extension of their theory of hippocampal function as a cognitive map to the human condition to explain the amnestic symptoms seen after hippocampal damage strikes me as very speculative. However, in my case at least, any attempted rebuttal or comment would be equally speculative, and thus not a very nutritious object of attention, to paraphrase William James.

To sum up cryptically, the book is interesting, scholarly, and provocative. Many experiments are suggested (probably the primary benefit from hypotheses) but the particular theory advanced in it seems premature to me.

by **Richard F. Thompson, Paul R. Solomon[1] and Donald J. Weisz**

Department of Psychobiology, University of California, Irvine, Irvine, Calif. 92717

"Model systems" versus "neuroethological" approach to hippocampal function

O&N's book is an extensive, scholarly effort and an important contribution to the study of hippocampal function in behavior. We were particularly impressed with their detailed and current review of the anatomical literature. Their theoretical position – that the hippocampus functions as a "spatial-cognitive" map of the organism's environment – is creative and provides a very good model against which to compare other general theories of hippocampal function.

In their book, O&N emphasize a "spatial map" function for the hippocampus as opposed to a "cognitive map" function. The more general notion that the hippocampus is involved in the more complex or cognitive aspects of information processing, which was independently suggested by O'Keefe and Nadel (1974) and by Hirsh (1974), seems to us to be of considerable value. The much more restricted spatial map hypothesis does, however, have the advantage of being directly testable. We have recently done so in an electrophysiological study where we were able to train hippocampal units in a temporal single-alternation paradigm (rabbit nictitating membrane [NM] conditioning) under conditions where there are no conceivable differential spatial-contextual cues (Hoehler & Thompson 1980), a result contradictory to O&N's more restricted spatial map hypothesis.

A related problem involves the apparent dichotomy O&N establish between spatial and temporal information processing in the hippocampus. According to their theory, the hippocampus is involved in the coding of spatial but not temporal relationships (see O'Keefe & Nadel 1978, p. 95). Although this point appears to be crucial to their theory, it also poses difficulties. Thus, it assumes that time and space are independent dimensions of the learning situation. As Moore (1980) has pointed out, there is likely to be a temporal component to all "spatial" tasks. Perhaps more to the point is the evidence, stemming from work in the rabbit NM classical conditioning preparation, which indicates that the hippocampus is involved in the processing of temporal information. As indicated elsewhere (Solomon 1980), learning in this preparation is primarily temporal in nature. Nevertheless, data from electrophysiological (e.g., Berger & Thompson 1978a) and lesion (see Moore 1980, Solomon 1980) studies indicate profound hippocampal involvement in learning in this preparation.

Another point of dispute we have with O&N's book is their less than comprehensive and sometimes cavalier treatment of data on the activity of hippocampal neurons in the context of learning, especially where the data do not appear to agree with their theory. This is particularly evident in their very critical and limited review of the pioneering studies of Olds, Segal and associates (Olds et al. 1972, Segal 1973).

Still another point of disagreement concerns O&N's characterization of neuropsychological versus neuroethological research strategies in the study of neural activity in the intact, behaving animal. They categorize our approach as "neuropsychological" and their own as "neuroethological" and, not surprisingly, favor theirs. We would prefer to term our approach a "model systems" or "paradigmatic" strategy. The basic issue here has to do with the kinds of phenomena one wishes to explore and one's view of how the nervous system functions. O&N's interest is in the "function" of the hippocampus; ours is in brain substrates of learning and memory. Our model system approach involves selection of a "prototypic" behavioral system having the properties of associative learning that is well defined and characterized and that exhibits robust learning. The model systems approach has been employed very successfully for neural analysis of simple forms of behavioral plasticity like habituation, where reflexes of the neurally isolated spinal cord and reflex pathways in simpler invertebrates can be shown to exhibit the behavioral properties of habituation (Kandel 1976, Thompson & Glanzman 1976).

For the study of associative learning, we selected the preparation developed by Gormezano (Gormezano et al. 1962) – classical conditioning of the rabbit NM response to a tone conditioned stimulus (CS) using a corneal airpuff unconditioned stimulus (UCS) – as a simple and discrete model of mammalian learning. The rabbit NM system has a number of advantages for analysis of brain substrates of learning, which we have detailed elsewhere (Thompson et al. 1976; see also Disterhoft, Kwan, & Lo 1977). In the course of the project, we discovered a rather remarkable form of neuronal plasticity in the hippocampus – in classical conditioning, neuronal activity in the hippocampus grows very rapidly to form a "temporal model" of the actual amplitude-time course of the behavioral response being learned (Berger, Alger, & Thompson 1976, Berger & Thompson 1978a, Berry & Thompson 1978, Thompson et al. 1976). Once this learning-dependent neuronal process has been identified, it is then necessary to determine the generality of the process in terms of stimulus and training variables, species, other learning paradigms, and its relations to the "laws" or variables of learning. In the end, this approach will result in definition and analysis of the brain mechanisms of learning.

O&N approach the problem from the other end, using their "neuroethological" strategy. In essence, they wish to determine the "function" of the hippocampus by study of the hippocampus, more or less in isolation, in a wide range of behavioral situations (O'Keefe & Nadel 1978, pp. 190–196). A difficulty with their "neuroethological" approach is of course that neither the hippocampus nor any other brain structure exists in isolation. Further, it is doubtful if a complex system like the hippocampus can be said to have *a* function. Another difficulty is the fact that their approach to behavior, in common with classical ethology, is basically observational. No time-locked simultaneous measurements of neural and behavioral events are made, nor is the fine structure of behavior measured. Our basic finding concerning the learning-dependent neuronal process in the hippocampus described above would not have been possible without such measurements.

In the end, the relative merits of the "model system" versus "neuroethological" approaches to brain and behavior will be determined empirically. At this point we would simply note that in the classical conditioning paradigms we have used, neuronal activity in the hippocampus becomes massively engaged in a time-locked manner to the *learned* behavioral response; at least 80 percent of pyramidal neurons appear to become so engaged (Berger & Thompson 1978b, Thompson & Berger, 1979). Furthermore, as noted above, the pattern of increased frequency of hippocampal neuron discharge actually models the temporal fine-structure of the learned behavioral response. In marked contrast, neuroethological studies of hippocampal neuron activity indicate only modest engagements in the behavioral observations and situations employed to date (O'Keefe & Nadel 1978; Ranck 1973, 1975, 1978). This is not meant in any sense to deny the usefulness and importance of the extensive empirical studies of hippocampal unit activity in the freely moving animal by these authors and by others (e.g., Vinogradova 1975). In particular, the observations of "spatial" units are most intriguing. We would, however, suggest that the ultimate interpretation of the significance of such unit responses must await a more fine-grained behavioral analysis.

Our final point concerns the logic of the lesion approach to brain substrates of behavior. O&N imply (p. 194) that because hippocampal

lesions do not impair simple learning, therefore the hippocampus plays no role in simple learning. This would seem to be an example of a "linear causality" argument. The motor systems of the mammalian brain provide a helpful analogy. There are a number of brain structures and systems categorized as "motor." They interact at all levels from the cerebral cortex to the final common paths. In animal studies, the effects of even large lesions of the motor structures and systems on motor behavior can range from severe, to moderate or mild, to virtually nondetectable. Yet these systems are all characterized as motor. Even a motor structure as well developed in primates as the pyramidal tract can be ambiguous. Studies by Evarts (1964) show a precise coding of the force used in making hand and wrist movements by cells of origin of pyramidal tract fibers in the motor cortex of the monkey. Towe (1973), on the other hand, emphasizes the minimal nature of motor deficits in monkeys who have had complete bilateral section of the pyramidal tract. No one would argue from this absence of a lesion deficit that the pyramidal tract therefore plays no role in the control of movement.

In general, the lesion-behavior approach, per se, has not been overly helpful in detailed analysis of the roles of brain structures in behavioral movement. Why, then, should we expect so much more from the lesion approach with regard to far more complex aspects of behavior like learning and memory? Clearly, deficits in various learned behaviors following hippocampal lesions are of interest; indeed, there is a wide variety of such deficits. However, the absence of a deficit does not necessarily imply an absence of function. To illustrate, O&N note that hippocampal lesions do not impair simple acquisition of the classically conditioned NMR (e.g., Schmaltz & Theios 1972). However, they neglect to cite the lesion-behavior evidence which shows that the hippocampus is very much involved in NM conditioning in more complex paradigms (e.g., latent inhibition and blocking), where the animal is required to learn temporal relations between the CS and the UCS (Solomon 1977, Solomon & Moore 1975). These experiments make it exceedingly difficult to argue that hippocampal lesion deficits involve *only* spatial learning.

Note

1. On leave from Department of Psychology, Williams College, Williamstown, Mass. 01297.

by **Holger Ursin**

Department of Physiological Psychology, Institute of Psychology, University of Bergen, 5000 Bergen, Norway

Anatomical units in psychology

Even if it turns out that the hippocampus is no cognitive map after all, neuropsychology will never be the same after this book. It is a penetrating, challenging and educational theoretical work which is and will remain obligatory reading for neuropsychologists for many years. It is also a very elucidating treatment of spatial learning and cognitive maps in normal, unoperated brains.

My evaluation is not affected by the fact that many of us think the authors are wrong. It may even be said that the book has probably shortened the spatial cue era in hippocampus research since it has made this theory so explicit and testable. Recent work seems to bring us back to square one. In their original work on memory deficits following hippocampal lesions in rats, Kaada, Rasmussen and Kveim (1961) found a deficit in the learning rather than the retention of learned material, and they also pointed to the greater effect of distraction in hippocampal or fornical lesioned rats. These seem to be reconfirmed as the essential elements in the primary working memory deficit suggested by recent work by Jarrard (1975) and Gaffan (1977). The deficit does not specifically affect spatial cues (Olton, Becker and Handelmann 1979), but I doubt that our insight would have progressed as fast without the book, the impact of which was felt long before it finally appeared from the publisher.

Essential elements of cognitive map theory remain even if the most extreme formulations regarding space cues may be wrong. In particular, there is still a possible specific role for the hippocampus in more complex or cognitive types of learning as opposed to simple S-R associations. If there are specific brain mechanisms for the recognition of complex cues, important issues are raised also for contemporary learning theory. Unfortunately, animal work on compound stimuli is complicated and difficult to perform.

The anatomical substrate discussed by O&N consists not only of the hippocampus, but also of entorhinal cortex and the septal area. It remains puzzling that each element in these flow charts seems to have different functions. For instance, the effects of lesions on orienting behavior, habituation and exploration differ, septal lesions do not produce the same change as hippocampal lesions, which again differ from entorhinal lesions (Køhler 1976a,b, Sundberg and Køhler 1979). Livesey (1975) found differences among the pyramidal cells. Self-stimulation properties differ between dentate cells and hippocampal pyramidal cells, in spite of the fact that the dentate cells feed directly and only into the pyramidal cells (Ursin, Ursin and Olds 1966). The *raison d'être* for neuropsychology, to my mind, is to compare and, if possible, combine two sets of variables, one anatomical and one psychological. There are problems in defining the units in both sets of variables. Is it meaningful to treat the hippocampus as a whole? Are the real units CA1, CA3–4, and the dentate area? What about the possible difference between the ventral and dorsal parts? For the psychology matrix the questions are even harder. Are memory or cognitive maps useful constructs? What are our tests really measuring? How much of the variance we observe is really explained by our theoretical constructs, and are such constructions valid across tests? In other words, if we claim to measure "spatial learning" in spatial test 1, do we also measure the same thing in spatial test 2?

I believe we should take the multivariate nature of the phenomena we study more seriously. I believe we have been trying to do canonical analysis for a long time without really doing it. It should be admitted that our own attempts to follow this strategy for the septal complex produced distressing results (Ursin et al. 1978). In particular, our fairly standard test battery did not produce consistent factors across tests. This does not suggest that the approach is useless. But it does suggest that the present theoretical framework is inadequate, or that the relations between variables are so complex (for instance, curvilinear) that ordinary correlation and regression studies are inadequate. If so, the theoretical task may require very complex mathematical methods.

Finally, our understanding of the interaction between structures appears to be inadequate. The belief in flow charts and pathways does not fit the facts. Summation of lesions is also problematic. Do combined lesions of A and B produce the same effect as the sum of lesions of A and B separately? One would hope that mathematical techniques could also advance our understanding of such interactions. Mishkin (1978) has recently found that amygdala and hippocampal lesions combined produce a much more pronounced memory deficit than either lesion alone. This also suggests that we should take vertical connections on the traditional hippocampus slice more seriously, in particular the angular bundle.

The cognitive map hypothesis may be wrong, at least in its most extreme formulations. If so, O&N have helped us through that phase quickly and intensively. In any case, they have offered us a broadening and sharpening of our theoretical framework which I think the field needed right now, whether we pursue this by classical tests or sharpen our logical faculties by mathematical methods.

by **C.H. Vanderwolf**

Department of Psychology, University of Western Ontario, London, Ont., Canada N6A 5C2

Is hippocampal rhythmical slow activity specifically related to movement through space?

O'Keefe and Nadel have proposed an interesting theory of the function of the hippocampus in animal behavior. One of the assumptions of the theory is that hippocampal rhythmical slow activity or RSA (especially atropine-resistant RSA) is uniquely related to behaviors which translate an animal's position in space. The frequency of RSA waves is assumed

to be directly proportional to the velocity of such movement and/or the distance to be moved.

Although I am favorably impressed by the O&N theory as a whole, I doubt that the function it assigns to RSA is correct. First, some behaviors, such as digging in sawdust or struggling when restrained, are accompanied by clear 7–12 Hz RSA even though they do not involve translation in space in the sense intended by the O&N theory. The RSA accompanying struggling has been shown to be resistant to large doses of atropine but the RSA accompanying digging has not been challenged with atropine as yet. Another example of RSA accompanying movement in a fixed location is the fact that the RSA accompanying walking on a treadmill is indistinguishable from the RSA accompanying ordinary walking (Whishaw & Vanderwolf 1973). This is true in rats even when the head is rigidly clamped in a stereotaxic frame by means of a metal attachment fixed to the skull (unpublished observations). The RSA generated under such circumstances is resistant to atropine. On the other hand, atropine-resistant RSA does not occur when a rat stands motionless, regardless of whether the substrate is motionless relative to the earth's surface or whether it is moved about (passive movement). Thus, if one examines location, immobility, movement in space and absence of movement in space in all four possible combinations, it is apparent that atropine-resistant RSA is associated with the locomotion itself rather than with translation in space.

O'Keefe (personal communications) tends to counter these objections by suggesting that treadmill walking is an unnatural situation which subverts the normal function of walking as a means of moving from one place to another. Similarly, strugging when restrained may represent an attempt to escape from the place of restraint. Although such defenses of the O&N theory may seem rather *ad hoc,* they do have a certain plausibility. Other data are needed to settle the matter.

The precise correlate of variations in RSA frequency has never been entirely resolved. When a rat makes sudden quick runs in a small experimental enclosure, the associated RSA has a higher frequency than the RSA associated with walking or slow runs. The RSA frequency during the first second or two of running in a treadmill (i.e., after the treadmill is started suddenly) also exhibits a positive correlation with the velocity of the treadmill running surface. These facts may suggest a relation of RSA frequency to running velocity or distance run. However, when rats or gerbils run steadily at different speeds in a motor-driven treadmill there is no relation of RSA frequency to velocity relative to the running surface. Perhaps high frequency atropine-resistant RSA is related to movements with an abrupt onset (i.e., with sudden increases in velocity).

When rats are trained to jump out of a box of varying depths, the higher jumps are associated with greater initial velocities as well as with a greater distance traversed as compared to lower jumps. RSA frequency begins to rise prior to a high jump and continues to rise after the rat is launched, reaching a peak at a time when frame-by-frame photographic analyses (Whishaw, personal communication) indicate that the rat is engaged in a violent scramble to grasp the edge of the box with its forepaws. Peak RSA frequency is directly related to the height of the jump.

According to the O&N theory, RSA frequency should be correlated with movement velocity and/or the distance jumped. If RSA frequency is related to instantaneous velocity, it should peak early in the jump, when body velocity is highest, then decline progressively during the movement to the top of the box. Alternatively, if RSA frequency is related to the distance to be jumped, the RSA occurring just prior to launching should display particularly high frequencies on high jumps, since it is at this time that total force (impulse) and acceleration are adjusted to suit the height to be jumped. (Once launched, the rat's behavior cannot affect the velocity or distance of the jump.) Since neither of these predictions corresponds well to the observed data, I conclude that RSA frequency is not related either to instaneous velocity of movement or to distance moved. It remains possible, I think, that high frequency RSA is related to movements with high acceleration.

I think the significance of RSA is to be found, not in some function attributable to the hippocampus alone, but in the functional organization of the forebrain as a whole. Type I movements, such as postural adjustments, head movements and all forms of locomotion, are accompanied not only by RSA but also by atropine-resistant neocortical low voltage fast activity and by changes in the amplitude of neocortical evoked potentials (Pond & Schwartzbaum 1972, Vanderwolf, Kramis & Robinson 1978). Comparable alterations in hippocampal and neocortical activity do not accompany Type 2 behaviors such as waking immobility, licking, chewing, face-washing or shivering. Although the precise significance of these electrical events for brain function and behavior remains to be determined, it does seem likely that Type I motor patterns are controlled by a complex set of forebrain circuits which are not directly involved in the performance of Type II behavior. These Type I behavior control circuits may be involved in a wide variety of functions including spatial navigation.

***by* J. Jacques Vonèche**

Department of Psychology, University of Geneva, CH-1211 Geneva 4, Switzerland; The Ontario Institute for Studies in Education, Toronto, Ontario, Canada M5S 1V6

Cognitive maps: dimensionality and development

For all the intelligence and knowledge put together in this book, it comes as a shock to the cognitive developmental psychologist of Piagetian inclination. On the basis of very thin evidence spread from rats to humans, the authors claim "that the brain must come equipped to impose a three-dimensional Euclidean framework on experience. . . ." Furthermore, they "identify this absolute, nonegocentric, spatial framework with a specific neural system centered in the hippocampus."

These bold statements raise a certain number of questions that are not adequately answered in the book. The first one is that of the reason for this imposition of a three-dimensional framework. Why not a four-dimensional or a six-dimensional one? It does not even seem to dawn upon O&N that this could be possible, and that some of the authors reviewed in the first chapter, such as Poincaré, have convincingly argued in favour of such manifolds. What is experience for them? The daily routine of the average human adult, the world of the theoretical physicist or that of the child, or the animal, or all of them? This is not clear in the book either, although it is a central question, because the world of the physicist encompasses more than three dimensions, for instance, and they seem to have a brain like anyone else. Did Lobatchevski and Riemann not *experience* their own non-Euclidean geometries? How would the invention of new geometries be possible if the brain were imposing a three-dimensional Euclidean framework upon experience?

As far as children are concerned, how could one speak of an absolute, nonegocentric, spatial framework in them when – to take the example given once again in the accompanying précis (so that the reader can see that I am not truncating the quotations) – children consider that two billiard balls of different colors have exchanged their positions, since the color order is different on opposite sides of the billiard table? How can one speak of such an absolute space, when children are furthermore incapable of thinking that a person sitting in front of them sees a perspective different from their own when they are both looking at the same three-dimensional landscape? How can one speak of such an absolute space when it is the experience of every parent that the young child mirrors the gestures of the adult he or she is facing when dressing, eating or gesturing?

Moreover, Euclidean geometry represents the most elementary form of geometry available, and that explains why it was so primitive in the history of human thought. It only considers intrafigural elements of space (as do the authors, by the way) and not the interfigural or transfigural ones. This explains, among other things, why children represent the level of water in a tilted beaker not as horizontal (interfigural reference) but as forming a 90° angle with the sides of the beaker (intrafigural reference). They remain purely Euclidean in their thinking.

I also find the book rather faulty at the level of the more specialized literature. The authors dispose quite promptly of the remarkable experiments of Don Stein at Clark University on the recovery of function

in hippocampal rats. This is a truly developmental line of research which, if taken seriously, would undermine considerably their own position. It shows that rats, operated in two or more stages, compensate for the loss of hippocampal matter in a remarkable way. This indicates that the localization theory of space perception should at least be reconsidered.

I have the utmost respect and admiration for those of us who write books. It is a difficult and exhausting task. But I would wish that academic pressures towards publishing were less effective so as to permit more thinking to go into book writing. Obviously, O&N have some very good ideas, such as the "spatial" analysis of language, for instance; but they do not go far enough into their analysis to cover the passage from displacements in the various spaces they identify: identificational, influence, and circumstantial, to transformations. Rules of transformation are at the root of the making of a stable universe of experience in a flow of stimulation; this is the central philosophical problem of our authors, who, unfortunately, like many new psychologists, find that a return to Kant is novel and appealing.

Authors' Response

by **John O'Keefe**
Department of Anatomy and Embryology, University College London, London WC1E 6BT, England
and **Lynn Nadel**
Department of Psychology, Dalhousie University, Halifax, N.S., Canada B3H 4J1

The cognitive map as a hippocampus

Here we languish, a bunch of poor scholars,
Battered by extremes of hunger and cold.
Out of work, our only joy is poetry:
Scribble, scribble, we wear out our brains.
Who will read the works of such men?
On that point you can save your sighs.
We could inscribe our poems on biscuits
And the homeless dogs wouldn't deign to nibble.
Han-Shan, "Cold Mountain," No. 10

Our theory was meant to do two things: we wanted to show (1) how a physiological theory could be interesting (and fun) when it addresses cognitions, and (2) that by so doing it could serve as the thin edge of the wedge, the base from which a broader cognitive neuropsychological model could be constructed. In contrast to several commentators (ISAACSON, GRAY, SWANSON, THOMAS), we feel that theories are premature only when there are no data. From the initial study, the first handful of units, the scientist builds theories. These can be explicitly stated or implicitly held, vaguely specified or precisely laid out, changed or discarded (or even upheld) as new data are generated, but they are always there. We find it hard to think about experiments in vacuo. One can, of course, quibble with the kind of theory we propose, as many rightly do, or even the way we chose to propose it, but to suggest that we propose nothing at all seems misguided.

The role of theory

The broadest objection, as voiced by ISAACSON, is that it is improper to formulate a theory of the role of a given brain structure because this role depends on the function of those other neural structures with which it interacts. While we can agree in general with the latter point, we fail to see why this rules out theorizing altogether. Only in a "tightly-coupled" brain must one "explain" all or nothing at all. In a tightly-coupled mechanism, removal of a part results in large changes in the working of all the remaining parts; in a loosely-coupled mechanism, subsystems are relatively independent and can be removed without great disturbance to the remainder. Adoption of the loosely-coupled view seems necessary if one is going to do lesion work at all.

HIRSH is quite right when he notes that brain entities can be characterized both in terms of the operations they carry out and the kinds of data upon which they operate. The issue here is that of content versus form. Kant held that space was form, a way of perceiving rather than what was perceived (see quote on p. 22 of the book; subsequent references to page numbers also indicate the book). We have followed this Kantian line by proposing that "spaceless" data enter the hippocampal system and cognitive maps come out. The spatial relations among objects which are embodied in the map are constructed within the mapping system: this is the operation it performs, its function, and our quest.

Another objection, voiced by SWANSON, is that philosophy and the brain are not ready for each other quite yet. We think our colleague, whose beautiful work on all aspects of hippocampal anatomy should be stapled directly into the book just after Chapter 3, is being far too modest here. Research on the role of experience in determining the functions of the sensory systems has the most profound implications for philosophers today, as does work on language acquisition in primates, the effects of commissurotomy, and so on.

The job of theory is to piece together an extremely complex jigsaw puzzle. Even though we have only a small sample of the pieces, there are benefits to be had from starting to put them together. A fragmentary theoretical framework helps us choose among the available data those which are of especial interest, and tells us what kinds we need to generate in "future research." Several commentators (and some passing, nodding, acquaintances) felt that we might have put the puzzle together prematurely, or with unseemly haste (GRAY, THOMAS, VONÈCHE); let them try convincing our publishers (spouses, parents, children) of that. Others seem to feel that we put the wrong puzzle together, more patches than pieces, and much of what we have to say here will be addressed to these commentators.

Why a cognitive theory?

Our commitment to a *cognitive* theory of brain and behavior is openly stated, and just as openly queried by some. What exactly do we mean by cognitive, why do we need it (as MOORE wonders), and what does a cognitive theory of brain function look like? By cognition we refer to knowledge acquired by the organism in the course of its experiences – "knowledge that" – which cannot be characterized in terms of stimulus-response linkages, or other similar forms of association. The importance of the distinction between cognitive and noncognitive modes of operation seems well understood by some (AMSEL, THOMAS, Olton 1979). Others (MOORE) worry that this must force us towards some form of dualism, or interactionism, in our attempts to understand the brain. But the alternatives to linkages between stimuli and responses are not *merely* mental representations, and hence of limited interest. The structure of the mind is one of the core problems of psychology, and can be tackled by assuming a limited identity theory, as Thomas notes.

We need a cognitive theory of brain function for one simple reason: other kinds do not work. Of course, not all the animal's brain, nor the brains of all the animals, are cognitive in the sense described above. Our contention is that both cognitive and noncognitive systems exist in the brains of higher animals, fulfilling different functions. They are neither mutually exclusive, since they can act in concert, nor simple points on a continuum, since they are different in kind (to answer one of OLTON's queries). A large part of the theory, as several noted (AMSEL, ELLEN, GRAY, MOORE, and others), is about these two kinds of systems, and their features and functions. And while the theory might not exactly stand or fall with the precise distinction we drew between locale and taxon systems, this is a central point in our analysis.

To briefly reiterate: the locale system, comprising the hippocampus at its core, *but also including neighboring structures* such as entorhinal cortex, septum, and subiculum (THOMAS, URSIN, others), is a cognitive system, which we take to mean that it acquires informa-

tion without the need for typical rewards, does it (probably) in a single "trial," transforms this input into maps, and makes this "new" knowledge available for future use. The way in which these maps get used depends upon the way they are accessed, but it is always the case that they are formed rapidly and used flexibly. The taxon systems, comprising the remainder of the brain, are really many separate systems, all apparently conforming to certain rules of operation – incremental storage and little flexibility in usage. Added to these characteristics, we attempted to specify as clearly as possible the properties of behavior based on these systems. As AMSEL points out, there is no necessary connection between cognitive systems and rapid, nonreinforced, learning. Nor must it be true that the locale system is relatively interference-free. The properties we attribute to cognitive maps were proposed for specific reasons, much as we assume they evolved to fulfill particular needs (Thomas's argument about the ecological pressures responsible for the evolution of a fast-acting cognitive mapping system could be an example of this). The motivation for the particular properties we gave to the locale and taxon systems must be strong to overcome the objections of GRAY, who argues that we simply put the inexplicable deficits which follow hippocampal lesions in a box labelled "taxon systems" and took this as an explanation for these well-documented, if variously interpreted, effects.

There were four sets of considerations which influenced the assignment of properties to the locale and taxon systems. First, we analyzed the features of maps and routes. In an insightful book unfortunately published too late to be included in our writing, Downs and Stea (1977) make many of the same points we raised concerning the distinction between locale and taxon learning. Second, we made the assumption that the behavior of an animal after a brain lesion at least partially reflects the normal operations of the remainder of the brain (note how this assumption would probably not be made if one held, even implicitly, the "tight-coupling" theory of brain noted above). This assumption, as DOUGLAS saw, raises the possibility of what we call an "environmental lesion"; by arranging test conditions such that locale function is unnecessary, one should be able to duplicate in intact animals the behavior seen in animals with hippocampal dysfunction. One of us has some preliminary data in support of this notion from studies applying the logic to the problem of persistence (Nadel 1979a; unpublished observations). Third, the known facts of hippocampal physiology and anatomy constrained the possible properties of the locale system; in particular the data from studies of unit activity were important. Finally, fourth, the fact that synaptic changes can be rapidly induced in the hippocampus (Bliss and Lømo 1970) dovetailed with our conviction that the map is specialized for the rapid acquisition of information.

Of course, it remains for these verbally specified properties to be translated into statements about how animals behave, as OLTON notes. There are situations in which it is easy to say that locale or taxon systems are involved; there are situations in which it is very hard. This is partly because the two can act in tandem, or even in each other's stead in certain cases (see later discussion of the unit data in another context). The idea is to plan experiments which make it easy to say what kind of learning is taking place, rather than ones which wait until after the data are collected to ask what the animals might have been doing, as we were forced to do in analyzing the available studies for the book. This, of course, is why theories are crucial: they tell one what kinds of experiments to do, and not to do.

Since we are on the topic, we might digress for a moment to consider those commentators who addressed themselves to our view on how experiments should be done. The issues at stake are very important; anyone who reads the whole of the hippocampal literature cannot fail to be impressed by the need for better experiments. It seems hard to believe that after many hundreds of lesion studies (and countless thousands of animals) we are still arguing over the most basic of questions concerning hippocampal function. There is a lot of data, and if much of it tells us very little about what we need to know, we should stop and ask ourselves why.

Our feeling, in agreement with the BLANCHARDS, is that too many experiments are designed without adequate consideration of the kinds of behaviors a particular species displays in its natural setting, or any thorough understanding of the forms of behavior elicited by the specific, artificial, test situations which are used. The elegant work of the Blanchards and their colleagues over the past decade stands out as one of the rare exceptions.

Neuroethology vs. neuropsychology

Our views on single unit methodology induced several commentators to take us to task (BERGER, BURES, THOMPSON et al.), but they seem to have missed our main point. Our claim was not that the neuroethological approach is inherently superior to the neuropsychological one, only that they are appropriate at different stages in our understanding of the functions of a given brain area. In our early ignorance, the neuroethological approach is more appropriate because it makes fewer assumptions about the behavioral correlates of unit firing and should give us a better overall picture of the patterns of these correlates – the "mountains" as we called them. Once we think we have identified the overall pattern we can begin to ask specific questions. Here we will find ourselves moving towards neuropsychological paradigms. Thus, in the book we said: "The advantages of the neuropsychological approach are clear: it affords a degree of control over the experimental situation which will eventually be necessary if the laws governing the operations of the nervous system are to be discovered. Its drawbacks result from its premature application" (pp. 193–194). It is our belief (perhaps not shared by the commentators) that the study of many brain areas (excepting perhaps some visual areas) is still in an early enough stage to demand a neuroethological approach. THOMPSON's example of the pyramidal tract is an unfortunate one since we are not as convinced as he is that its function is understood or that labelling it "motor" is very illuminating. To us, the apparent discrepancy between lesion and unit work suggests that the function of the pyramidal tract is not clearly understood. If it is a motor system, why does it project to "sensory" areas such as the dorsal column nuclei, and influence transmission through these nuclei (Jabbur and Towe 1961). When Ranck (1973) and O'Keefe (1976) found units in the hippocampus which were related to motor behaviors, we did not jump to the conclusion that the hippocampus was a motor system. The existence of other classes of units which might not have been seen in simple motor tasks suggested a wide function for the hippocampus. Perhaps a neuroethological approach to the pyramidal tract would reveal a wider function concealed to date by the narrow emphasis on its "motor" function.

Several commentators remark on the discrepancies amongst the various laboratories studying hippocampal units (BERGER, GRAY, HOLMES, RANCK). There are, however, two different types of discrepancies: those of observation and those of interpretation. Ranck and O'Keefe seem to be describing similar behavioral correlates, but give them different interpretations. On the other hand, there does seem to be a more fundamental discrepancy between this work on rats and that on rabbits (BERGER, THOMPSON et al., Vinogradova 1975). Unpublished work by one of us (O'Keefe) now indicates that this is due at least in part to a species difference in the behavioral correlates of the theta units. In both rat and rabbit these cells increase their firing rates during certain movements. In the rabbit, however, they also increase their firing in response to arousing stimuli. Thus, Vinogradova's results may be due to an emphasis on this aspect of the theta cell firing pattern. Whether or not the changes in unit firing during the conditioning of the rabbit nictitating membrane response have a similar explanation is not yet clear.

BURES has some hard words to say about the neuroethological approach, in particular the use of relatively unstructured environments and the reluctance to use statistical analyses. To some extent, these objections rest on a metatheoretical presupposition: that the coding of information in the nervous system is subtle, too subtle to be detected by the gross analyzers, eye and ear. If the neural code really is so uncertain that random fluctuations in the biophysical properties of the neuron spike generating process are an important factor (to take one of Bures's examples), then clearly the neuroethological

approach is doomed. If, on the other hand, even a significant minority of cells in the nervous system transmit information by large changes in their firing rate or pattern, then the neuroethological approach is not only feasible but preferable. Bures is also afraid that the neuroethological technique will introduce free will into science. But there is really no way to purge it. In the neuropsychological approach, the experimenter chooses the set of stimuli to be presented, the response the animal must make to them, the statistical confidence levels, and so on. The "hard" numbers which come out at the end are only as good as the voluntaristic decisions made before the study begins.

BURES suggests several techniques we could use to record more objectively the relationship between behavior and single unit activity. He also suggests some neuropsychological experiments one could do to test the place correlate hypothesis (put animals in trolleys). We thoroughly agree with him that having used the neuroethological approach and convinced ourselves that we had a good overview of the behavioral and physiological correlates of hippocampal neurons, we should now switch to the neuropsychological approach. We think he will find that recent papers from our laboratory and those of others (e.g., Ranck, Olton, Best) have done just that (see O'Keefe 1979b, for a review). He will also be reassured to know that several laboratories have, for many years now, used the technique of simultaneous recording of behavior and single unit activity on videotape as an aid in the analysis of behavior-unit correlations, much as he suggests. Frame-by-frame analysis does enable one to answer questions such as whether the theta units increase their activity before the onset of movement and whether the place fields of a place unit are the same when the animal faces in different directions. To reiterate then, we think the criticisms levelled against the neuroethological approach are misdirected. We are not against the quantitative neuropsychological approach, only its premature application. Bures may be right that the appropriate neuropsychological experiment would have revealed the place correlate of the hippocampal cells. We certainly would not have selected it a priori from the thousands of possible correlates. How many psychologists are studying classical conditioning with a grandmother as the conditional stimulus?

Anatomy of a cognitive map

How well does the theory fit with the anatomy of the hippocampal system? Perhaps there is a cognitive map, but it is not localized in the hippocampus (see DOWNS). It may be localized elsewhere or it may be distributed across several brain systems. Several commentators have reminded us that lesions to other parts of the brain are associated with spatial deficits (DOUGLAS, HÉCAEN, KESNER, THOMAS). Frontal cortex, caudate, cerebellum, parietal cortex, and the superior-colliculus/pulvinar/visual-cortex system are prominently mentioned in this regard. Several authors chide us for failing to discuss adequately the work of Semmes et al. (1955, 1963), which purports to show a generalized deficit in route following with parietal lesions. We did, in fact, analyze these data closely, but chose not to describe our conclusions. Briefly, the primary deficit seems to have been shown in the tactile version of the study, and correlated well with a deficit in two point discrimination; the deficit in the visual version of the task was no more noticeable following parietal lesions than it was following temporal or occipital lesions, all of which seemed somewhat more debilitating than frontal lesions. We think these particular data are more consistent with the assertion of a deficit in tactile discrimination in the patients with parietal lesions than with the multimodal spatial deficit advanced by the authors. We still hold to the view that many of these other spatial areas are involved in egocentric spaces (see Hécaen), in which objects are located relative to the organism (see pp. 381–383). One of the main tenets of the book is that it is not sufficient to characterize the function of an area as spatial since there are different kinds of space. Some evidence as to the nature of the spatial processing in these areas might be obtained by studies of single-unit activity therein. Clearly, the units recorded in visual cortex and superior colliculus indicate egocentric functions, while those recorded in frontal and parietal regions may suggest egocentric or locale functions (see O'Keefe 1979b for a discussion).

One of our goals was to provide a plausible wiring diagram for a cognitive map which was consonant with the known anatomy and physiology of the hippocampus. One reason for attempting what may appear a premature effort is to enable us to address ourselves at some point to the type of partial lesion data which JARRARD and others are generating. Jarrard finds that while in most tasks a lesion of the CA1 field is equivalent to a total hippocampal lesion, this is not true in the eight-arm radial maze. His results suggest that CA1 is necessary to establish a map originally but that the map must be established outside the CA1 region. Since total lesions of the hippocampus disrupt the task even when it is acquired preoperatively, it would seem likely that the map is in the hippocampus, perhaps in CA3 or the dentate gyrus. As Jarrard notes, this is consistent with one of the models outlined in the book. One problem with this analysis is that it does not specify how the CA1 area, which generates and changes maps, acts upon the CA3 area, which contains the maps. Is there an anatomical pathway between these two which remains to be discovered, or is the circuit through the entorhinal cortex? Note that Mitchell and Ranck (1977) have reported place cells in the entorhinal cortex.

SWANSON points out several inadequacies in our description of hippocampal anatomy, due to the ever-changing picture. He is right in his assessment that we would like to find sensory information entering the hippocampus via the entorhinal cortex, and the absence of a set of neocortical inputs to entorhinal cortex in the rat similar to those demonstrated in the monkey may prove an embarrassment. We will just have to wait until the anatomists tell us how the sensory information reaches the hippocampus in the rat. We are less worried about the minor changes in the topography of the intrahippocampal connections, since the theory requires longitudinal connections between the lamellae, and we had already mentioned the longitudinal association bundle as a possible substrate for that (p. 118).

BLISS asks some tough questions about the details of the model of a mapping system. He notes certain similarities in the formal properties of our model and that of Marr (1971). We appreciate and welcome his interest. Our main project in the book was to present a plausible model which was consistent with what was known at that time. We are well aware that the details must be wrong, and are prepared to consider changes to the model where it conflicts with the data. Part of this process will undoubtedly lead us into a closer consideration of the formal models proposed by others such as Marr, and McLardy (1960) and his coworker, Kilmer (Kilmer and McLardy 1970, 1971).

Finally, OLTON asks us to comment on the effects of differing sizes of lesions in behavioral studies. This is easy. Big lesions make big holes in the map; small lesions make small holes. Because of the redundancy within the mapping system, however, there should not be a linear relationship between lesion size and behavioral deficit.

Hippocampal place units

We do not agree with GRAY that unit data on the hippocampus are fruitless.[1] Nor, for that matter, do we concur with THOMPSON's dismissal of lesion studies. Rather, we try to take the more moderate position that all techniques have something useful to contribute to our understanding of the brain. One of us has recently reviewed the latest work on the hippocampal place units (O'Keefe 1979b) and many of the questions raised by the commentators are answered in that article. Here, we will briefly summarize some of the more important points. BERGER and DOUGLAS seem to have gotten the impression that the place field of a place unit was determined solely by distal extramaze visual cues. Although this is the case for some, others are clearly multimodal (e.g., the unit shown in figs. 26 and 27, pp. 205–210). There is, as yet, no evidence that the collection of place cells representing an environment is knit together into a map (HOLMES, SWANSON). The postulate that there is a map rather than

merely a collection of place representations goes beyond the physiological data but is necessary to explain some of the behavioral results (e.g., the fact that normal, but not hippocampally lesioned, animals seem to know in the start box of an alley what has happened to them on the previous trial in the goal box; on this point, we refer GREENE to pp. 342–344 of the book for a solution to the conundrum that might tickle his fancy). We have not found the destination cells suggested by HIRSH in the hippocampus or elsewhere, but would dearly love to do so. Their existence would simplify the mechanism by which the map guides behavior. To our knowledge place cells have not been found in any other brain region except the hippocampus and entorhinal cortex (THOMAS). But there are still many unexplored regions of the brain, so this is more an assertion of ignorance than anything else.

BERGER wonders whether our crude recording techniques obscure subtle differences in place unit firing patterns, e.g., when the animal is facing in different directions. We have seen some evidence for this and it should clearly be investigated in detail using the neuropsychological method. Notice that even if it does turn out that the place cell is "responding" to different sets of cues when the animal faces in different directions, one is still left with the problem of explaining why the cell responds to the cues perceived from that single location rather than two different locations. How does it put together the two sets of cues into a single place representation?

Finally, several commentators take us to task for giving such little weight to the results of single unit experiments performed by others (such as Olds). Our problem here was that these experiments were done in such a different way from our own that it was hard for us to make any meaningful connection between the two. Whereas we (and Ranck) looked at several different behaviors, watched our animals, and did not have strong preconceived notions as to what the units might respond to, Olds trained his animals on a single conditioning task in a sensorily restricted environment, did not make any behavioral observations, and was interested only in the relationship of unit firing to the different aspects of the conditioning process. Since his group did not try to classify their units according to any physiological criteria (except perhaps for firing rates), it was difficult to relate their findings to ours in any way at all. Since hippocampal lesions had no obvious effect on the conditioning task used (but see THOMPSON et al.'s commentary on this point) and their results did not bear directly on our theoretical position, we had little alternative but to keep our references to that body of work to a minimum. The same is less true of the unit work of Thompson, Berger, and their colleagues. They have classified their cells according to several criteria (firing rates, presence of complex spikes, activation from the fimbria), and it should be possible to relate their findings to those of others (including our own). Thus, there are good prospects for a reconciliation of the apparent differences between hippocampal units in eye-blinking rabbits and place-coding rats in the near future.

Before leaving the topic of unit recording we should consider an important question raised by OLTON which might yield to a study of unit behavior. Olton asks us to distinguish between a place strategy and a direction strategy, and correctly points out that most place tasks can be solved using a direction strategy. In fact, Blodgett et al. (1949) reported that place learning was much more difficult for normal rats than was direction or response learning. This would seem to suggest that direction learning is different from place learning, that many "place" tasks are really learned through the use of a direction strategy, and that the place system does not have the properties which we ascribed to it (e.g., rapid, all-or-none learning).

What, then, is the difference between direction and place learning? As OLTON correctly suspects, there is no unequivocal answer to this question. To see why, let us do a thought experiment on one of the place cells. Imagine that we are recording from a place unit in Olton's example (his fig. 1, top) and find that the cell has a field on the east arm. The cell fires whenever the animal enters the arm and regardless of the direction which the animal faces on that arm. Swapping physical arms around has no effect on the unit response so there is prima facie evidence to consider this a place unit. Since it fires regardless of the direction which the animal assumes it cannot be a direction unit. What might the results be if we perform Blodgett et al.'s transfer test as proposed by Olton. The cell might continue to fire in the same part of space as defined by the room: in this case it would fire in the west arm. Alternatively, the cell might define its field partially in terms of the apparatus and partially in terms of the extramaze cues. In this case the cell would continue to respond in the east arm on the transfer test. This type of cell would not be a pure extramaze place cell, but it would not be a direction cell either. It would define a place in terms of the configuration of the apparatus and the orientation of the apparatus relative to the environment. Animals acting on the basis of this information would make a direction choice rather than a location choice in the Blodgett et al. experiment. They would also not be affected by the translation of the maze within the room after task solution, but would be affected by maze rotation. Thus, Watson's maze translation results are not evidence against our position, as suggested by DOUGLAS.

Furthermore, DOUGLAS's (1966a) own spontaneous alternation experiments might have a similar explanation. He found that rats would alternate if the first trial was given in one room and the second in another, adjacent room. He concluded that this ruled out distal cues but this may not be true if these cues were common to the two rooms (e.g., distant traffic noise, magnetic fields). We do not accept his conclusion that the vestibular system has a prominent role to play in place learning, although we agree that an animal deprived of place information might have to rely on vestibular cues, as perhaps was the case in Beritashvili's (1965) interesting neuroethological experiments. The study mentioned by THOMAS is of some relevance here. He showed, in a T-maze alternation task, that hippocampal lesions disrupt preoperative learning but that relearning is possible. However, this relearning, presumably accomplished on the basis of alternative sources of information (vestibular?), is affected by variables which do not particularly influence animals learning with their brains intact. This indicates that relearning in the lesioned animals involved a new strategy with different properties. According to Beritashvili (1971), vestibular cues by themselves can support performance only across rather short delays, which would explain why Thomas's lesioned rats could not perform when delay intervals were lengthened.

Let us conclude this section by acknowledging that OLTON has focused on what remains a major problem: the difficulty in separating the two possible strategies for solving the direction task. The place strategy (dependent on the mixed place cells described above) and a guidance strategy (where a distant cue is approached) would both work here. Although we must continue to search for behavioral techniques with which to separate these two strategies (such as the cue-controlled environment), it remains possible that an unambiguous behavioral technique does not exist and that the distinction can only be made at the physiological level.

Behavioral correlates of hippocampal theta

What is the relation between hippocampal theta and one's theory of the hippocampus? One possibility is that one could read the function of the hippocampus off the behavioral correlates of theta. Thus, VANDERWOLF thinks that a correlation of theta with movements indicates some function in motor control. Alternatively, the theta system might provide a clue to the operation of part of the hippocampal system which, taken in isolation, does not indicate the overall function of the hippocampus. The latter is our position, and from this perspective HIRSH's revival of the question as to whether theta is an active or inactive state of the hippocampus is inappropriate. The hippocampus is "working" in both the theta and large amplitude irregular activity states: many place cells fire when the rat is in the place field regardless of whether it is engaged in theta behavior or not. So what does theta do? We have suggested that it shifts the focus across the map as a function of the displacement associated with movement. It does this in an a priori fashion: that is, it calculates the distance that a movement would take an animal under normal circumstances, rather than operating on the basis of the

distance through which the movement actually translates the animal (which is how the theta mechanism gets around one of the problems raised by DOWNS). Here we are really following and extending Vanderwolf's own reasoning concerning the relation between theta and voluntary movements in general. For example, he concludes that the relation holds even during REM sleep, but that the movement signals which accompany theta during this state are blocked at the level of the spinal cord. Similarly, we would argue that the occurrence of theta during struggling is not a valid counterexample since the motor activation would translate the animal were it not restrained. The rat nervous system did not evolve to function in an environment of treadmills, skull clamps, and moving carts.

VANDERWOLF focuses attention on jumping as a good paradigm for the study of the relation between movement and theta. Although we (R. Morris and J. O'Keefe unpublished) have repeated the main findings of their earlier paper (Whishaw and Vanderwolf 1973), there are certain differences which bear on the present discussion. In our experience, animals only scrabble at the end of the jump when they are required to jump too high. During lower jumps the landing is as smooth and coordinated as the take-off. If the rat scrabbles at the top it defeats the purpose of the jump situation, since there are no measures of force, acceleration, and so on available for this behavior. Contrary to the results cited by Vanderwolf, we have not found an increase in theta frequency during flight in our animals. Instead there is either a peak frequency at the moment of launch followed by successively lower frequencies until landing, or a peak frequency at launch which is maintained (but not exceeded) until landing. Thus, our observations are consonant with the asserted relation between theta frequency and velocity (which is maximum at the launch and decreases during the flight to zero). We should point out, however, that even were Vanderwolf's assertions true they would not necessarily lead to his conclusions unless one accepted only strict cotemporaneous correlations. We would be quite happy with a good correlation between velocity at the launch and a peak theta frequency some 200 msec later. There is no theoretical reason why the correlation must be cotemporaneous.

HOLMES wonders why no theta activity has been recorded from the monkey and whether this implies that monkeys (and humans?) do less cognitive mapping. After all, the rat is a highly spatial animal, and might be peculiar in this way. We (and Menzel 1978) think not. There are several reasons why the slow waves might not be an accurate reflection of the underlying unit mechanisms. In the case of hippocampal theta, there is the possibility that increasing levels of theta unit activation result in an EEG amplitude function which first increases but then decreases, perhaps even to the point of desynchronization (see p. 153). Furthermore, the generation of synchronous EEG waves depends on the geometry of the hippocampal elements and generators. There is some suggestion that a theta mechanism exists in CA3 of the rat although good EEG theta cannot be recorded there. We predict that there is a theta mechanism in both monkeys and humans, albeit one more sophisticated than that found in the rat.

Other functions of the hippocampus

As we argued earlier, the neuroethological approach is useful when little is known for certain, and one wants to keep an open mind. There can be little doubt that this was the case concerning the hippocampus in the early 1970s, and many seem to feel it remains that way today. The commentators, and other recent writers, have managed to find a large number of functions other than spatial mapping to attribute to the hippocampus. These include such things as temporal mapping (HOLMES, KESNER, NONNEMAN, THOMPSON et al.), salience reduction (MOORE), general context coding, (HIRSH & KRAJDEN, Winocur and Olds 1978), working memory (URSIN, Olton et al. 1979), holding still (BLANCHARDS), anticipation of negative events (AMSEL), internal inhibition (DOUGLAS), dealing with uncertainty, ambiguity, or interference (GRAY), and allowing memories to become resistant to disruption (SQUIRE). Can all these possibly be right?

We have addressed a number of these suggestions elsewhere, and our intention in the book was to avoid constant comparisons of one of the above hypotheses with another. As suggested in an earlier paper, there are differences between theories and hypotheses (Nadel and O'Keefe 1974), and speaking to 300 rather than 30 or 3 studies is one of them. Nonetheless, and contrary to our view that science is not an adversary process, we feel compelled to consider some of the issues raised by these suggestions, and by some of the studies mentioned in the commentaries.

1. Internal inhibition (DOUGLAS). We have strongly criticized this notion elsewhere (Nadel and O'Keefe 1974; Nadel, O'Keefe, and Black 1975; Black, Nadel, and O'Keefe 1977). Perhaps Douglas's claim that this idea remains viable should be bolstered with a reasoned reply to these criticisms. We persist in seeing the inhibitory failure as an effect, not a cause.

2. Holding still (BLANCHARDS). While we are uncertain about the decreased immobility observed in studies 2 and 3 of Blanchard et al. (1977), study 1 raises the likelihood that the immobility being measured is, as the authors point out, a reflection of the shift from flight to freezing, which presumably occurs when the animal concludes that escape to a safe place is impossible. The interesting question then becomes: what controls this freezing? We concluded (pp. 299–302) that place fear is largely responsible for such immobility, and that its absence in animals with hippocampal lesions should lead to a loss of freezing. Blanchard, Blanchard, and Fial (1970) reported data consistent with this idea. The decreased ability of lesioned rats to hold onto hot grids, or balance on ledges, could be due to a similar mechanism, or possibly to shifts in reaction to low-level shock in the first case (Blanchard and Fial 1968), though the data here are ambiguous. Woodruff and his colleagues (Woodruff and Bailey 1979; Woodruff, Hatton, and Meyer 1975) have shown that restraint-induced immobility is *increased* after hippocampal lesions in rats and rabbits, indicating that in some cases animals without a hippocampus can hold still even better than intact animals.

3. Anticipation of negative events (AMSEL). We certainly agree with the view that the hippocampus has a lot to do with anticipations, but fail to see why this should be restricted to negative events such as punishments and frustration (though the latter do seem to be prominent concomitants of studying the hippocampus). Since there is strong evidence that the mapping system is involved in match-mismatch operations of all kinds, we prefer to see its role in anticipatory frustration in terms of the anticipation rather than the frustration. The assumption that the hippocampus is only involved in certain aspects of frustration, without providing any justification for partitioning frustration into separate functions in this way, makes little sense to us.

4. Dealing with uncertainty (GRAY). That the hippocampus somehow provides the basis for flexible behavior is uncontested; what is called into question by Gray is our proposed source for this capacity. According to Gray, there exists a system in the brain whose function is to help out when an animal is in an ambiguous situation, or one in which high interference is likely. Exactly how this sytem is triggered into action, how it "knows" that the animal is in a tenuous situation, or in one in which interference is likely, remains unstated. We await the promised exhaustive treatment of the literature on hippocampal lesions, and an explication of how uncertainty/interference are detected by the animal. It is our guess that the mechanism which accomplishes these functions will look rather like a mapping system.

5. General context coding (HIRSH & KRAJDEN, Winocur and Olds 1978). Hirsh argues for the view that the hippocampus performs a certain kind of logical operation on all forms of data, and that by so doing it underlies all manner of contextual effects. This plausible

suggestion runs into one major stumbling block: putting all contextual functions into the hippocampus leaves little for the remainder of the brain to do. One of us has considered the problem of context at length (Nadel and Willner, in press), and concluded that the hippocampus plays a role in spatial, but not other, context effects.

6. Working memory (DOUGLAS, URSIN, Olton et al. 1979). In several recent papers Olton and his colleagues have advanced the view that the hippocampus is a working memory system capable of temporarily holding any kind of information. This view has led some to conclude that map theory is outdated already, as wilted as yesterday's greens. We think this burial, even if well-attended and carried out with requisite ceremony, somewhat premature. As we have stated our views on this in several places (Nadel 1979a and b, in press; O'Keefe, 1979a), we refrain from further comment here.

7. Temporal mapping (BLANCHARD & BLANCHARD, HIRSH & KRAJDEN, HOLMES, KESNER, NONNEMAN, THOMPSON et al.). A number of commentators feel that a cognitive map should encode time as well as space. Even though animals without a hippocampus have little difficulty learning about temporal relations, Thompson et al. remain convinced that the hippocampus is involved in such learning, arguing from data on unit activity and on the effects of lesions on complex Pavlovian paradigms such as latent inhibition, conditioned inhibition, and blocking. We have recently addressed these issues (O'Keefe, Nadel, and Willner, 1979; Nadel and Willner, in press) and concluded that hippocampal involvement in these tasks does not require the assumption that the hippocampus is involved in temporal mapping.

More generally, we do not agree with those who would wish to accord time the same status as space. Holmes suggests that we should write a monograph on time to complement the one on space – we suspect such a book would be mercifully thin. Not that time is uninteresting philosophically or unimportant psychologically. On the contrary, time is very important, but we see little evidence that it is coded directly in the way space seems to be.

The book was somewhat ambiguous on the role of time in the mapping system, eschewing it for rats while allowing it for humans. Even in the latter, it remains unclear how judgments about temporal "location" are accomplished beyond the recent past. Such work as exists on the ability to assess when in the past something happened indicates that our unaided performance in this domain can be quite primitive (Linton 1975). A sharp distinction can be drawn between this kind of temporal mapping, in which the locale system is involved, and the kind which is concerned with the ongoing temporal texture of events – the CS-US interval, for example. The lesion data rule out an essential hippocampal role in such textural analyses, though they do not rule out the possibility that the information about such temporal intervals and sequences is normally made available to the hippocampus for some purpose which is not essential to the conditioning process itself. Models of this sort (MOORE, THOMPSON et al.) give the hippocampus a role in overseeing certain consequences of temporal pairings, such as the loss of salience which CSs undergo when presented without USs (latent inhibition). Since lesion data cannot currently invalidate this position, we must await physiological data from appropriately designed unit studies.

8. Salience reduction (MOORE). In a recent article Moore (in press) has provided a model for how temporal mapping might be accomplished, and what uses it could be put to: he proposes that the hippocampus participates in the development of maps which code both location and time. The hippocampus does this by acting to reduce the salience of entities (places, cues, or responses) under specified conditions. This position would seem to predict a crucial role for the hippocampus in the "overshadowing" of a CS poorly correlated with the US by a CS highly correlated with the US, for such an arrangement of temporal pairings results in the reduction of the poorly correlated CS's salience. P. Garrad, J. Rawlins and N. Mackintosh (personal communication) have recently explored the effects of hippocampal lesions on overshadowing, and found that they did not impair rats' ability to show the effect. This seems to directly contradict Moore's position. In addition, it deprives the temporal mapping view of its most likely function.

This overview of alternative suggestions (we take up Squire later) provides much the same picture as our earlier assessments. Though specific suggestions based on the results of one, two, or even a dozen studies are often capable of accounting for some portion of the data, they fail to go beyond this preliminary step. As several commentators point out, the future success, if any, of these other perspectives rests on their ability to wade through the mine-field of hippocampal lesion effects and emerge relatively unscathed on the other side. We wish we could provide our intrepid colleagues with a good map to the location of the live mines, but ours is somewhat tattered by now.

Even though we cannot agree with those who seek to expand upon the functions attributed to the hippocampus, there remain a number of specific studies mentioned by various commentators which must be noted. As was the case in our analysis in the book, not every study falls neatly into place. For example, we have no ready explanation for the data reported by Rawlins et al. (in press) concerning the effects of hippocampal lesions on the partial reinforcement extinction effect. While we are not surprised that hippocampal lesions increase resistance to extinction in continuously rewarded animals (p. 342) and decrease it in partially rewarded ones (p. 347), we cannot readily explain why these two groups fail to differ from one another. Admitting this does not, however, indicate acceptance of GRAY's conclusions, which may be mitigated by differences in terminal acquisition rates (as seen in the goal speeds) or unexplained sources of variability (such as that which produced greater differences between the control groups of studies I and II than those between the control and experimental groups within either study).

Somewhat less difficult to answer are those who query our analysis of the effect of hippocampal lesions on the operant task known as the differential reinforcement of low rates (DRL). In this task, animals must refrain from responding for a period of time before being rewarded for a response. This conflict situation has been interpreted as involving timing behavior, though we prefer to see it as an example of the need to develop strategies which help the animal avoid the site of the manipulandum. Thus, intact rats can formulate the strategy "go to a place on the other side of the box" and thereby decrease the likelihood of temporally inappropriate responses. Animals with hippocampal lesions sometimes have problems with this kind of task, as both the temporal mapping and inhibitory views might suggest, but this occurs, as ELLEN points out, *only* when there is a conjunction of relatively large lesions *and* pretraining on continuous reward. Exactly why the hippocampus should be involved in "timing" only in these circumstances is unclear, and we have little to add here to what we have already said (pp. 323–325). What is still needed is a careful study of the precise behaviors – be they place strategies or other – used by normal animals to help wait out the interval. If there are no such differences between intact rats and a group of lesioned rats, as ISAACSON suggests, then our analysis is wrong. A study similar to that reported by Osborne and Black (1978) should settle this issue (as well as bear on the temporal mapping proposal).

Many commentators (DOUGLAS, GRAY, HIRSH & KRAJDEN, JARRARD, and others) wonder about the deficit in reversal of nonspatial tasks, taking this as an indication that the deficit after hippocampal lesions is not specifically spatial. Let us consider these data, which are indeed difficult for our theory to explain, in some greater detail. Animals are trained to perform a visual or tactile discrimination with the discriminanda shifted from location to location so as to avoid the reinforcement of a particular place or orientation strategy. Having learned to approach one of the cues, presumably through adoption of a guidance strategy, the animals are confronted with a reversal of the reward contingencies. Rats and cats with hippocampal lesions have a well-documented deficit in this reversal procedure (see Table A19). The defect, however, is of a certain form. Upon reversal, animals stop

responding to the previously rewarded cue (the extinction phase) rather soon; this is typically true for both intact and lesioned animals (the Webster and Voneida [1964] study quoted by several commentators is the exception that proves the rule, and is also the only study using split-brain animals with unilateral hippocampal lesions). Some trials later, animals begin responding to the previously unrewarded cue. In the interim, other strategies are adopted and discarded. The bulk of the defect in lesioned animals arises at this stage; they seem to adopt the strategy of approaching a particular side of the apparatus, and stick with it through an inordinate number of trials. Why do lesioned animals fall into this trap while intact animals manage to avoid it?

DOUGLAS makes two points concerning this situation. First, he states that the locale system cannot be involved in simultaneous discrimination (an assertion he also makes concerning the linear maze). Second, he suggests that lesioned animals should be at an advantage in reversal according to our analysis, since they have one fewer incorrect strategy to try out. These seductive suggestions (see GRAY, GREENE) are, unfortunately, a bit too simple. We opined in the book (p. 280) that the learning of even simultaneous discriminations could proceed differently in intact and lesioned animals, and provided some support for this view. (Douglas is also wrong in his assertion about the linear maze – see pp. 286–290). Devoid of place strategies, lesioned rats choose among fewer strategies, and as a consequence adopt orientation strategies more often during original learning than do intact rats (Kimble 1975). Though such a strategy is rewarded only 50 percent of the time, and is subsequently jettisoned for a guidance strategy, this experience during learning could increase the relative likelihood of the adoption of an orientation strategy by the lesioned rats after reversal of the reward contingencies. This being the case, we would predict that more lesioned than intact animals would fall into the persistent orientation trap. Only an animal-by-animal analysis of the strategies used during initial learning and subsequent reversal can definitively answer the question of why there is a defect. Such an analysis of performance in successive discrimination, another task which is ostensibly nonlocale, made sense of what otherwise seemed to be rather confused data (see pp. 284–286).

Finally, VONÈCHE raises the question of the "recovery of function" after multistage lesions. We treated these data in the book (pp. 377–379), where we suggested that "recovery" was only possible when tasks which could be solved by either locale or taxon strategies were used. In these mixed tasks, such as the ones Stein et al. (1969) used, many interpretations of the data are possible, a point emphasized by OLTON. THOMAS's data on recovery support our view that lesioned animals relearn using different, nonlocale strategies (see also Isseroff 1979).

What kind of maps?

A number of commentators found themselves in general agreement with, or were at least sympathetic to, the notion that animals form cognitive maps, but questioned our views on the means by which these maps are formed, the precise nature of the spatial information they contain, or the relationship of these spatial representations to other kinds of spatial codes.

We suggested in the book that maps are multimodal representations constructed during exploration, and that these maps are not pictorial images, though portions of a map can be "projected" onto the "mind's eye," to use current terminology. These maps were taken to represent Euclidean, three-dimensional space, forming the basis for our concept of absolute, unbounded space. One objection to this formulation, raised by ELLEN, is that active exploration may not be needed for map formation, raising doubts as to the role of the movement-theta system in map construction. However, the studies cited by Ellen in support of his contention are not particularly convincing. Gleitman's (1955) rats acquired only the barest of representations, and because distinctive cues were available to differentiate each arm, it is possible that the learning in this study was not place learning at all. This position is supported by McNamara et al. (1956), who also compared rats allowed to run in a T-maze with others passively transported. In their first study, obvious extramaze cues which could support guidance strategies were available, and passive transportation was as effective as active movement. However, when these guidances were removed, transported animals were no longer able to learn; active movement became essential. Thus, these data indicate that, at least for the rat, active exploration *is* necessary for place learning and, presumably, map formation. Data suggesting that map construction can proceed in higher animals (cats, dogs, primates) without movement are intriguing, but they require systematic replication. Perhaps a study along the lines of those reported by Menzel (1978), but concerned with map acquisition rather than utilization, would settle the issue.

Taking a somewhat different tack, DOWNS raises a number of interesting questions about the kinds of maps formed in the hippocampus, questions we cannot meaningfully answer at this time. As Downs points out, the very assertion that there is a neural locus for cognitive mapping provides the basis for asking these questions, and it was certainly our hope that researchers in allied fields would begin to investigate some of these issues. Our choice of a Euclidean metric for the map was influenced of course by the considerations sketched in Chapter 1, but it also has the merit of permitting easy testing. Such testing seems hardly necessary for VONÈCHE. Having overlooked our footnote (p. 23) where we admitted that we had no explanation for why absolute psychological space was three-dimensional, he asks what is experience for us? We cannot pretend that it is the daily routine of the average human adult (whatever that statistical fiction might be), but that is not the point. What is important is the environment which the brain (and behavior) have evolved to handle, and that physical environment is most simply characterized in three-dimensional Euclidean terms.

It is our guess that the inventors of non-Euclidean geometry did not, in fact, directly experience these spaces, but that this would not prevent them from discovering their existence. Experience is a tricky word: in what sense can it be said that we experience infinity, or an irrational number? We also fail to understand how a cognitive developmental psychologist of Piagetian inclination can assert that children who represent the level of water in a tilted beaker as forming a 90° angle with the sides of the beaker "remain" purely Euclidean in their thinking. VONÈCHE seems to imply that Euclidean geometry, being the most elementary form, comes first in the developing child, just as it did in the history of human thought. This inverts the progression of geometrical thought ascribed to children by Piaget, though not in fact by us (see pp. 41–45, and especially the footnote on p. 45).

PINKER provides useful criticism by outlining a scheme within which three different kinds of spatial representations reside. The elegant work of Kosslyn, Pinker, and their coworkers (Kosslyn et al., this issue) has established the existence both of a visual imagery system akin to a pictorial surface and an intrinsic three-dimensional shape system which permits the recognition of objects according to this shape. Pinker suggests, and we agree, that the mapping system is an abstract framework containing markers which "stand for" the objects occupying specific locations. This tripartite arrangement makes sense not only of the data from normal subjects engaged in cognitive mapping tasks, but of amnesic subjects as well. The latter are capable of generating images and have no trouble recognizing three-dimensional objects in various perspectives (a parietal cortex function?), but fail at map formation (see pp. 422–425, 434). On this note we can turn to what was surely the least acceptable part of the book – the extension of the model to humans.

Mapping in humans

One of the major motivations behind our extension of the model to humans was our conviction that a theory of hippocampal function would be worth pursuing only if it managed to extend beyond rats, cats, and monkeys to include humans. Several assumptions guided

our attempt to span the species. First, we concluded on the basis of our anatomical review that there was little evidence suggesting a major alteration in the hippocampal machinery in humans, as compared to other species: electrophysiological data were consistent with this view (Babb 1973). What do seem to change, even between closely related species, are input and output patterns. Here, we are in agreement with HIRSH & KRAJDEN that the hippocampus performs certain operations upon a new set of inputs, in this case leading to "spatial" maps for language, what we have called semantic maps. Second, we felt that, as in the rat, the hippocampus in humans had to be a memory system. We supposed that there are many kinds of memory systems, each storing the results of its operations, and that the hippocampus was a special, spatial, kind of memory system. This was simply an elaboration of the position taken by the theory on infrahumans. Third, we assumed that the hippocampus in the left hemisphere, concerned as it was with language, must be constructing something like a spatial map for "verbal" entities. We were encouraged in this line by the work of those linguists who saw important connections between cognition and semantics (Jackendoff 1976, Miller and Johnson-Laird 1976). Finally, fourth, we accepted the dogma that damage to the hippocampus resulted in amnesia (Milner 1968a) with only minor, and perhaps insufficient, contact with amnesia patients. In so doing, we relied heavily on (then) currently available data about the specific features of the amnesic syndrome. All these considerations led us to the view, expressed in the book, that the hippocampus in humans was a memory system concerned with mapping aspects of both space and language.

The detailed specifications of the model for humans, as for the neural nuts and bolts of the map itself, were our best guesses, and would, we hoped, serve as useful starting points from which to elaborate better versions of the model. While we saw no reason to change the major tenets of the mapping model based on rats, it was clear to us that human maps are probably more sophisticated than those of rats. Thus, for example, we supposed that in the human the ability to store, and keep separate, individual maps of related experiences would allow for a more pronounced temporal dimension in memory (as noted above), though we were aware of how inaccurate such temporal memory appears to be, at least in comparison to spatial memory. This led us to the view that the mapping system might serve as the substrate for what Tulving has called episodic memory.

We find little reason to alter most of these assumptions. However, the comments of HOREL, SQUIRE, and JACKENDOFF seem to require at least some modifications of our position. In his extensive and well-documented article Horel (1978) suggests that the amnesia consequent upon mesial temporal lobe resection (as in patient H.M.) has more to do with damage to the temporal stem than with damage to the hippocampus. Horel is quite right when he asserts that our theory adapts to, rather than predicts, the global amnesic syndrome. He goes too far, however, when he tries to rob the hippocampus of all its mnestic functions. It is undoubtedly the case that some aspects of the memory loss in amnesia stem from damage to taxon structures such as the amygdala and temporal neocortex. However, we maintain that those aspects of the memory disorder relating to space (see HÉCAEN) relate to hippocampal damage. Additionally, and in contrast to Horel's comments, we do insist on placing the source of exploratory behavior in the hippocampus; the compulsive behavior of monkeys with Kluver-Bucy syndrome hardly constitutes exploration. This being said, the problem of specifying where the role of a spatial memory system ends, and that of nonspatial memory begins, is quite difficult.

Consider the mechanisms by which we retrieve information from long-term memory. In a recent thesis Williams (1977) has probed the strategies used by subjects in retrieving the names of their fellow high-school graduates. Among the strategies used were "general associations," "locations," "name generation," "picture scanning," and "activities." What stands out in these data is the extent to which spatial locations (contexts) were used in retrieving from very long-term memory, even when ostensibly nonspatial strategies such as "activities" were being used. For example, in a protocol demonstrating the use of this strategy, specifically "playing in the band," the subject started by imagining herself in the band room. This indicates that recall might depend importantly on the ability of subjects to find an appropriate context "containing" the desired information. As Williams points out, the problem of *finding* a context only exists in recall paradigms which, it seems, can involve largely reconstructive processes. Given an appropriate context, recall of the entities within it becomes relatively easy (see Chapter 14 for our expression of similar ideas). In recognition paradigms, the problem is one of *choosing* the appropriate context. These data demonstrate not only the importance of contexts (typically specific spatial locations) in long-term memory assessed by either recall or recognition techniques, but also the way in which access to information available in taxon systems can be crucially dependent upon the locale system.

Such considerations led us to speculate that there would be extensive loss from premorbid memory of those forms of information stored in the locale system, as well as a marked inability to use contexts as retrieval aids. As SQUIRE points out, however, the recent data gathered in long-term memory studies suggest a marked discontinuity between premorbid and postmorbid memory loss. As important as these data are, they do not directly address the above position, because in these tasks the subjects were presented with the retrieval contexts, either in the form of faces to be recognized, or the names of the old TV shows for which details were to be recalled. Given these contexts, the ability to reconstruct details should be relatively unimpaired in amnesic patients, as Squire and others have indeed shown. (The story is more complicated in Korsakoff patients, who were included in our analysis with some reluctance, see pp. 414–416.) In the Marslen-Wilson and Teuber (1975) study, for example, H.M.'s performance was vastly improved by certain kinds of cues (as controls' might have been were it not for a ceiling effect), but he fared rather poorly with cues of a circumstantial nature. We would predict that in the Williams task of recalling high-school graduates amnesic patients would be selectively deficient in their ability to generate spatial strategies, but unaffected in their use of such things as initial letters as aids to retrieval.

The problem of why amnesic patients seemingly have difficulty forming new taxon memories remains. H.M. can learn some new faces, so the defect is not complete even in this most severe case, and HOREL is surely right in asserting that some of the memory problem relates to extrahippocampal damage. We assume that if the locale system is important for getting things out of taxon stores (by providing retrieval contexts), it must play an important role in getting them in. SQUIRE emphasizes this point in his discussion of the possible role of the hippocampus in a "restructuring" process by virtue of which memory is reformed and strengthened. Here, he alludes to what appears to be a shift, over time, in the form of normal memory from the specific to the general. This shift seems to be associated with the point at which the hippocampus is no longer needed for accurate remembering, and has occasioned considerable research and speculation (e.g., Wickelgren 1979). We think Squire is right in pointing out that the hippocampus becomes less important in recall as the memories one seeks fade into the past. In our view, this results from the way in which events are stored within the hippocampus. As the unit work shows, the same neuron takes part in many maps. If specific memories are being laid on top of one another within the mapping system, at a certain point access to the older ones should become exceedingly difficult, to be accomplished perhaps by unusual events such as brain stimulation (Penfield 1958), or seizures, or the usual event of aging, which is accompanied by changes in synaptic function and plasticity within the hippocampus (Barnes 1979), as well as by selective losses in place learning (Barnes, Nadel, and Honig, in press).

In the last analysis, our view of the amnesic syndrome, and its relation to hippocampal damage, will be judged by how well the theory accounts for the facts of normal memory. Present evidence, and the commentaries of HÉCAEN, DOWNS, and PINKER, encourage us to feel that, at least as far as specifically spatial information is concerned, our views are reasonable. When one considers the role of the left hippocampus in memory, however, things become less clear.

Semantic maps

Our section on semantic maps was the most speculative and understandably elicited the most resistance from the commentators. Here our main goal was to see how far one would have to stretch the basic attributes of the mapping system to produce a system which could explain the specific loss of linguistic memories following left hippocampal damage. We strongly concur with JACKENDOFF that word lists and the like are hardly the sort of thing one wants to study here. Unfortunately, the data available to us were largely of this "neuropsychological" form. We hoped to provoke the linguists into considering the thesis that a psychological cognitive map could serve as the deepest level of representation underlying sentences. Jackendoff has certainly been provoked, although often in directions unsympathetic to our ideas. Aside from a semantic quibble over the use of the term "deep structure," Jackendoff makes four criticisms of our ideas. First, he argues that our syntactic rules for generating surface sentences from semantic maps are too primitive and would not be accepted by most linguists. Second, that our distinction between absolute and relative spatial strategies is not reflected in the grammatical structure of sentences about these strategies. Third, that all of long-term memory cannot be located in the locale system. And, fourth, that the distinction between a right hippocampal map devoted to nonverbal spatial functions and a left hippocampal map devoted to semantic functions is untenable.

We did not set out to provide a complete linguistic system and it is somewhat unfair of JACKENDOFF (and VONÈCHE) to berate us for this failure. Nevertheless, his query as to why our system would not generate ungrammatical sentences like the examples he cites is a reasonable one, and we will try to answer it. Unfortunately, his first example – *To the ground, the rock fell from the roof* – although stylistically awkward, does not strike our ears as egregiously ungrammatical, especially if said with a Yiddish accent and a certain degree of chutzpah (see NONNEMAN). The second sentence – *The roof, the rock fell from to the ground* – is clearly ungrammatical, but would not have been generated from our map. The relationship between a place (the roof) and its contents (the rock) is identified by (subject) *has* (object) (see p. 404). So, entering the place first and its contents second yields the kernel sentence – *The roof has the rock (on it)* – or some suitable transform of this. *Subject, object* is not a legitimate reading of place and its contents. Similarly, prepositional phrases are readings of places in the map, or motions in or out of places, and cannot be arbitrarily separated from the names of those places. It does not seem difficult to generate the rules by which legitimate separation could be achieved. For example; *The ground is where the rock fell to and the roof is where it fell from* suggests that the reading of a movement from or to a place must be completed before another can begin.

JACKENDOFF's second point is that the difference between absolute and relative spaces is not reflected in grammatical distinctions. But this is a misreading of the relationship between the left and right hippocampus. The left hippocampus operates on principles similar to those of the nonverbal locale system, but it does not contain the same information. There is no reason to believe that the left hippocampus contains only information about absolute space. Following Jackendoff's example, we have already shown how it could contain information about nonspatial entities such as maps of possession, so we see no reason to restrict its spatial content to absolute spatial information. The analogy is with the principles of operation of the nonverbal locale system, not with the content. Such interesting spatial concepts as along, around, through, and back can be captured in this system. For example, around can mean either near or encircling. As we pointed out in the book (p. 8), such concepts as near or neighboring usually depend on the overall context in which they occur. Encircling can be represented by a place circumscribed by another place.

JACKENDOFF's third point, that there must be some long-term memory located outside the locale system, seems to derive from a simple misunderstanding. The attribution of long-term memory to both locale and taxon systems is a central feature of the theory, but the properties of these memory systems and their contents are assumed to differ.

Finally, JACKENDOFF doubts whether the split between the verbal and nonverbal systems is as large as we seem to indicate, since many sentences depend on an interaction between verbal and nonverbal entities. We agree with him, but plead not guilty. The large hippocampal commissural connections would seem to indicate that the two hippocampi, as with the two hemispheres in general, must speak enough of a common language to keep each other informed of activities of mutual interest. And where there is a common language there is at least hope for understanding.

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NOTE

1. Some of GRAY's scepticism about single unit data seems to rest on a misunderstanding of the extent and nature of the information provided by this approach. Firstly, extracellular recording, as opposed to intracellular recording, tells us what cells do with information, not what information they get. Secondly, unit recording provides data not only about these transformations, but also about their temporal relations to other events of interest, such as the animal's behavior. To follow Gray's analogy, we know not only who possess the train timetable, but also when they use it relative to the movements of the train itself. The train driver (hypothalamus?) consults it throughout the journey; the porter (ventral horn?) checks to see when passengers needing assistance will arrive; and the station chief (frontal cortex?) checks it once a fortnight against the actual performance. We will refrain from extending the analogy to include the obvious conclusion that the locations of the timetables in the station (hippocampus?) provide important clues to function.

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