THE
HIPPOCAMPUS
AS A COGNITIVE MAP

JOHN O'KEEFE
AND
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TO

E. C. TOLMAN
Who first dreamed of cognitive maps in rats and men

D. O. HEBB
Who taught us to look for those maps in the brain

AND

A. BLACK
Who insisted that we pursue our route with rigour

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Preface

Scientific theories have been likened to maps.* Like maps they provide a means for finding one's way in an unknown domain. And also like maps, they are a social product, the culmination of considerable effort by a large number of people. The cartographer draws on the lessons of past geographers, he builds on the work of the explorer and the surveyor, he relies on the skills of the geometer no less than those of the draughtsman and the parchment-maker. In this preface, we can do little more than pay small tribute to some of those who have contributed to the making of this map.

Our voyage of discovery was launched from the McGill Psychology Department where we were both Ph.D. students in the 1960s. At McGill, Don Hebb had built a department which encouraged students to theorize about the neural bases of perception, motivation, and cognition, and which gave them the freedom and the opportunity to test their ideas. Hebb's theory of the cell assembly was like a model map–it showed what a theory of the brain would look like. Hebb's graduate seminar was particularly influential. He emphasized the extent to which behaviourist notions could handle most of the available data and pointed to those few areas which showed the limitations of this approach: latent learning, sensory preconditioning, the surprise of Tinklepaugh's monkeys. At McGill one learned techniques from post-doctoral fellows and other graduate students. We were particularly fortunate to be able to learn from Ken Casey, now at Michigan, and Herman Bouma, now at the Institute for Perception, Eindhoven. Our research directors Ron Melzack and Dalbir Bindra were generous and tolerant to a fault.

When we first started to explore this terra incognita, we found only a small number of footprints in the sand. Most prominent were those of Helen Mahut, Case Vanderwolf, and Jim Ranck. We were amongst friends: both Helen and Case were McGill graduates and Jim had been a colleague of Ken Casey at Michigan for several years. To be sure others had also sailed past this coast; but, having dimly sighted it from afar, they quickly pushed on towards those ever-beckoning chimera: the Quagmire of Inhibition, the Palisades of Persistence, and the Channel of Attention. Helen Mahut was one who landed, planted her feet and stayed. Her demonstration of a selective deficit in spatial reversal learning after damage

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* This analogy was first suggested by Toulmin (1953). In Chapter 2 we elaborate on this distinction between maps and routes. Our extension of his analogy here to the social aspects of science derives to some extent from conversations with Graham Goddard.

to the hippocampal system was an important pointer to the spatial function of the hippocampus.

Another explorer who signposted the way was Case Vanderwolf. His observation that hippocampal theta in the rat was in some way bound up with a class of movements received early confirmation by Abe Black's impressive analytic experiments. Taken together, these had a strong influence on our thinking about hippocampal function. Not that we ever accepted Vanderwolf's implicit assumption that the function of the hippocampus could be equated with the behavioural or psychological correlate of the hippocampal EEG. Rather we concluded that a theory of hippocampal function had to account for the availability to the hippocampus of information about a class of movements.

Jim Ranck pioneered the recording of single cells from the hippocampus of the freely moving animal. Others had done so before him (most notably Olds (1965) and Vinogradova (1970)) but under very restricted conditions which may have prevented them from seeing the more spatial aspects of these cells. Jim Ranck's rats, like ours, were allowed to move about in an extended environment. The remarkable similarity between his data and ours was a source of great encouragment in those early days. We used different terminology and emphasized different aspects of the behavioural correlates but it was clear that we were seeing the same things. Many of our apparent discrepancies were straightened out during the three months that Jim Ranck spent in our laboratory in 1972 and during our subsequent visits to his laboratories in Michigan and New York. Some of our ideas on the neuroethological approach to single-unit recording are identical to his as set out in an unpublished paper dating from 1972.

More recently Dave Olton and Phil Best have added to our understanding of the spatial function of the hippocampus. They have confirmed the spatial nature of the hippocampal cells. Olton's eight-arm maze task appears to be a particularly sensitive test of the hippocampal mapping system.

Much of the past seven years has been occupied with the details of the map: delimiting the boundaries of the theory, knitting together the disparate pieces and patches, deriving detailed predictions about the behaviour of lesioned animals, designing experiments to test them. During this time we have had tremendous help from Abe Black. His investigations on the behavioural correlates of the hippocampal EEG and the effects of hippocampal lesions are models of experimental design. Some of the more explicit and testable predictions of the theory can be traced back to conversations with Abe Black. He has a complete collection of all of the many versions of this book, most of them heavily annotated and dog-eared. We decided it was time to publish when we found that we could actually read much of the typed text in the most recent one.

Other dog-eared, mind-chewed copies belong to Graham Goddard and Jim Ranck. Graham Goddard wrote an extensive commentary on the original version while patiently waiting in queues to get into the Tutankhamun exhibition at the British Museum. Jim Ranck read several chapters while trapped in the New York subway during a recent power failure. The theory has been tested under a wide range of environmental conditions.

Others who tried to improve the book through their comments on parts or all of it include Per Andersen, Herman Bouma, Hide Ishiguro, Peter Molnar, Eileen O'Keefe, Geoff Raisman, Tim Shallice, Pat Wall, and several anonymous advisers for the Oxford University Press. Peter Molnar read the book in proof with great care and pointed out numerous mistakes. One of the advisers took the trouble to point out many errors in the original version and prompted revisions of several sections. A more recent adviser chided us for our chiaroscuro painting of the data, but we had decided early on to present the boldest black-and-white case for the theory; later will be time enough to etch in the subtle greys.

Over the past seven years, we have been extremely fortunate in our colleagues and students, some of whom have been our strongest critics. Among those whose research is cited in this book are Abe Black, Dulcie Conway, Richard Morris, Liz Somerville, Simon Keightley, Dave Kill, and Barbara Oetliker. Our experiments were possible only because of skilled technical help we have had from Alan Ainsworth and Jitendra Patel. Jane Astafiev drew many of the figures and Julia O'Connor typed and repeatedly retyped the manuscript. Together with Howard O'Connor she also prepared the author index. Financial support has been provided by the Science Research Council, the Medical Research Council, and the Wellcome Trust, all of the United Kingdom.

Perhaps our greatest thanks are due to those who created a safe haven for us to work in during these past seven years. Maps are not made during a state of siege. Most of the book was written while we were honorary research fellows in the Cerebral Functions Group, in the Department of Anatomy, University College London. J. Z. Young, the head of the department until 1974, has had a life-long interest and involvement in the neural basis of animal memory. He advised as, encouraged us, and housed us. His hospitality has been continued by the present head, G. Burnstock.

Pat Wall, head of the Cerebral Functions Group, more than anyone else deserves credit for the existence and present shape of this book. It was he, who, in 1971, originally suggested that we turn a tentative review article into a slim monograph and then had to witness its inexorable growth year after year to reach its present girth. Throughout he has encouraged and cajoled us, defended us, read what we wrote, criticized, gently prodded, pushed, and has patiently given us the time to work things out. In these rapid-transit days of publish or perish, he slowed time to our snail's pace.

L.N. would also like to thank Holger Ursin and Bolek Srebro for their hospitality during the six months he spent at the University of Bergen and Larry Squire for his hospitality during the eleven months he spent in the Department of Psychiatry, University of California, San Diego.

The staff of the Oxford University Press skilfully steered the manuscript through the many phases of publication. We thank them for their patience, help, and good humour.

Friends and family put up with us all these years, successfully keeping us out of the asylum and restraining themselves from committing homicide: Eileen, Kieron, Riley, Maria, Britt, Melissa, Kenny, Mischa, Joao, Diane, Garth, Fiz, Wanda, and Charlie. ‘Thank you’ is small recompense.

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Contents

Introduction

THIS book is concerned with three topics which, at first glance, do not appear to be related: (1) a part of the brain known as the hippocampus; (2) the psychological representation of space; (3) context-dependent memory. We shall argue that the hippocampus is the core of a neural memory system providing an objective spatial framework within which the items and events of an organism's experience are located and interrelated. In this introduction we shall briefly summarize the main points of our argument.

The first chapter begins with an analysis of the history of ideas about space, as developed primarily by philosophers and influenced by physicists and mathematicians. Here, our primary concern will be to show that traditionally there have been two incompatible theories of the nature of physical space. The *absolute* theory views space as a stationary framework which is separate from, but can nevertheless contain, material objects; the *relative,* or relational,* theory views space as nothing more than the relations between material objects themselves. These arguments, which relate purely to an understanding of the physical universe, have had a profound effect on thinking about ways in which organisms represent space. In particular, the advent of non-Euclidean geometries and the theory of relativity have shifted the argument dramatically in the direction of a relative theory of both physical and psychological space, with the consequence that most authors attempt to derive all psychological notions of space from an organism's interaction with objects and their relations. The notion of an absolute spatial framework, if it exists at all, is held by these authors to derive from prior concepts of relative space, built up in the course of an organism's interaction with objects or with sensations correlated with objects.

In contrast to this view, we think that the concept of absolute space is primary and that its elaboration does not depend upon prior notions of relative space. In our view, organisms represent space in several independent, though interrelated, ways. A number of neural mechanisms generate psychological spaces referred to the observer, and these are consistent with a relative theory of space. Amongst these are spaces centred on the eye, the head, and the body, all of which can be subsumed under the heading of *egocentric space.* In addition, there exists at least one neural system which provides the basis for an integrated model of the environment. This system underlies the notion of absolute, unitary space, which is

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* The terms relative and relational have been used interchangeably by most authors. While Lacey (1970) argues that there is a distinction to be drawn between the two, for our purposes this distinction is unimportant.

a non-centred stationary framework through which the organism and its egocentric spaces move.

We shall call the system which generates this absolute space a *cognitive map* and will identify it with the hippocampus.* The term was first used by Tolman (1948), and has recently reappeared in the work of some psychologists and geographers interested in the perception of large-scale environments (e.g. Menzel 1973, Richards 1974). Although this use of the concept of cognitive mapping has descriptive and predictive value, it is too vague for the present book. To serve our purpose adequately it must be specified in sufficient detail to enable us to translate it into a neural model.

Briefly, a cognitive map would consist of two major systems, a *place* system and a *misplace* system. The first is a memory system which contains information about places in the organism's environment, their spatial relations, and the existence of specific objects in specific places.** The second, misplace, system signals changes in a particular place, involving either the presence of a new object or the absence of an old one. The place system permits an animal to locate itself in a familiar environment without reference to any specific sensory input, to go from one place to another independent of particular inputs (cues) or outputs (responses), and to link together conceptually parts of an environment which have never been experienced at the same time. The misplace system is primarily responsible for *exploration,* a species-typical behaviour which functions to build maps of new environments and to incorporate new information into existing maps.

In order to set this cognitive mapping system within the more general context of brain and behaviour we shall find it necessary to describe briefly a model of animal learning which lays strong emphasis on the use of *hypotheses* (cf. Tolman and Krechevsky 1933). Such a model states that, when faced with a problem, animals do not respond randomly, but rather select and test hypotheses, or strategies, concerning the solution. We assume that one form of hypothesis involves approaching or avoiding places in the environment and, further, that this type of hypothesis is based on information contained within the hippocampal cognitive mapping system. Other hypotheses, such as those involving an approach to a specific object or the performance of a particular response, are based on information stored by other neural systems. While our discussion will touch upon the properties of all these forms of hypothesis, our emphasis will remain on place hypotheses and the cognitive map.

On the basis of a review of the anatomy and physiology of the hippocampus a structural model of the cognitive map will be proposed. Amongst other things, this model will draw upon the existence of place-coded neurones in the hippocampus and will also propose specific functions for the rhythmic electrical activity *(theta)* recorded from the hippocampus during certain behaviours. In conjunction with the general behavioural model specified earlier, this structural model will enable us to generate predictions about the changes in behaviour brought about by dysfunction of the hippocampus. Here, we shall attempt to review the available literature from experiments with a variety of species, and show that it is consistent with the theory. The work with infra-humans will be discussed first, followed by an analysis of the relevant human clinical data. It will be suggested that the left hippocampus in humans functions in *semantic mapping,* while the right hippocampus retains the spatial mapping function seen in infra-humans. On this view, species differences in hippocampal function reflect changes in the inputs to the mapping system, rather than major changes in its mode of operation. This proposal offers the hope of unifying animal experimental and clinical approaches to the problem of hippocampal function.

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* Strictly speaking, the hippocampus should be called a cognitive mapping system, and the term cognitive map reserved for the products of that system. For convenience, we shall use the latter term to refer to both the neural structure and the representation of a particular environment; the context should make our meaning clear.

** Simply stated, a place is a part of absolute space and can only be defined in terms of its locus within the neural system mediating that concept. We shall say more about this central concept later (see p. 93).

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Remembrance of places past: a history of theories of space

1.1. Introduction

SPACE plays a role in all our behaviour. We live in it, move through it, explore it, defend it. We find it easy enough to point to bits of it: the room, the mantle of the heavens, the gap between two fingers, the place left behind when the piano finally gets moved. Yet, beyond this ostensive identification we find it extraordinarily difficult to come to grips with space. We could begin by asking a few common-sense questions. Is space simply a container, or receptacle, for the objects of the sensible world? Could these objects exist without space? Conversely, could space exist without objects? Is there really a void between two objects, or would closer inspection reveal tiny particles of air or other matter? These questions call to mind several related ones. Is space a feature of the physical universe, or is it a convenient figment of our minds? If the latter, how did it get there? Do we construct it from spaceless sensations or are we born with it? Of what use is it?

Philosophers, and particularly metaphysicians and epistemologists, have long sought answers to these questions. Aristotle, for example, held that the place of an object was the internal wall of its container. This derived from his view that the universe was a plenum - totally filled with matter - and that the outer surface of any object must be contiguous with some other matter; that is, the inner surface of its surround. This view of space survived for many centuries, in spite of strong criticisms and alternative proposals. In our review we shall not describe these early developments.* Rather, we shall pick up the story at the end of the seventeenth century, by which time the accumulated criticisms against the Aristotelian notion of space had led to its abandonment.

We have chosen to start with Newton, Leibniz, and Berkeley because it was in their writings that the problems of space were initially formulated in a way that is relevant to us. Before we turn to our history we should state our motives and intentions, and warn the reader of our biases. Our primary

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* Jammer (1969) provides an historical survey of theories of space, while Efros (1917) discusses some of the more telling criticisms of Aristotle's views.

interests are historical and intellectual. Put simply, we want to avail ourselves of the ideas and arguments which others had brought to bear on the subject. More importantly we wanted to locate ourselves within this historical tradition. When we first began to entertain the notion that the hippocampus provided organisms with an *a priori* Euclidean spatial framework, we found little support or sympathy in the writings of contemporary psychology or neuroscience. On the contrary, as we shall see, most current writers hold that space as represented by the mind or brain is dependent on the relationship between objects or stimuli. History shows that this was not always the case. Moreover we think it affords some perspective on why the current scientific climate favours relative theories of psychological space.

Our bias, then, is against relative theories and our intention is to emphasize the weakness of this point of view, not so much to harry it out of existence as to undercut its claim to account for all of the phenomena. We aim not for victory but an honourable truce. In our view both the absolute and the relative theories of psychological space are needed and we suggest that the Constructor of Brains hedged his bets and incorporated both systems into his invention. The problem then for the neuroscientist becomes one of describing the properties of these different spatial systems, identifying them with specific areas of the brain, and describing the interactions amongst them.

Recurrent throughout this historical chapter are a series of antitheses: psychological v. physical space, absolute v. relative space, innate v. learned or constructed, Euclidean v. non-Euclidean. The theory of space espoused by a particular author can be viewed (roughly) as a set of choices between these alternatives. While most of the choices are relatively independent of each other, certain groupings seem to go together more easily than others. For example, a theory of relative psychological space is usually conceived as acquired or constructed rather than as an innate property of the mind. It might help the reader if, at this point, we make more precise the sense in which we are using these terms and to adumbrate what we see as the issues surrounding each antithesis.

(i) Psychological v. physical space. We shall use the term *psychological space* to refer to any space which is attributed to the mind[*] either as an intrinsic aspect or as an inevitable or highly probable product of the normal operation of the mind, and which would not exist if minds did not exist. Psychological spaces can take many forms. Included are concepts which the mind constructs on the basis of reflections on experience, abstractions from sensations, organizing principles which impose unified perceptions upon otherwise diverse sensory inputs, organized sensory arrays which derive their structure from the nature of peripheral receptors, or a particular set of sensations transduced by a specialized (spatial) sense organ. The study of psychological spaces falls within the domain of the disciplines of psychology and physiology.

In contrast, *physical space* is any space attributed to the external world independent of the existence of minds. It describes a feature of the universe, even an inanimate, incognitant one. Physics is the discipline which addresses itself to the existence and properties of this space.[*]

Our concern in this book is primarily with psychological space. From our point of view, it little matters what the shape of physical space is or indeed whether space is a property of the physical universe at all. Our interest in physical space derives from two considerations: (a) historically, ideas about physical space have influenced thinking about psychological space, and (b) some theories of psychological space suggest that it is learned from or constructed on the basis of experience with the physical universe. Were this latter suggestion the case one might expect some correspondence between the properties of psychological space and those of physical space, and evidence of discordance between the two is *prima facie* evidence against such theories.

(ii) Absolute v. relative space. This distinction can be applied to psychological as well as to physical spaces. In our usage *absolute space* embodies the notion of a framework or container within which material objects can be located but which is conceived as existing 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. Movement of a body (including the observer) changes its position within the framework but does not alter the framework or the relationship of other objects to the framework. In contrast, *relative space* designates a set of relations amongst objects or sensory inputs which in themselves are inherently non-spatial. Objects are located relative to other objects and relative space does not exist independent of the existence of objects. In order to ease the reader into the absolute/relative distinction in the realm of psychological space, let us do a little introspective experiment. Let us close our eyes and think about space. Several different types of space come readily to mind. To us, these spaces divide rather naturally into those which are felt to emanate from the observer and those which are independent of the observer. In this latter notion there is a sense of position within a framework

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[*] Throughout this chapter we shall freely use the term mind without precisely defining it. In our usage, it is not identical with consciousness nor does it necessarily imply consciousness. Nor does it simply mean the operation of the whole brain. Readers who feel uncomfortable with this term and prefer an unknown set of well-defined entities or processes to a well-defined set of unknown entities or processes can systematically substitute the clumsy phrase 'some operations or activities of some (unspecified) parts of the brain' for 'mind'.

[*] Only mathematics is not tainted with sectarianism and tries to describe the properties of spaces irrespective of whether they are physical or psychological, or indeed exist at all in the sense we are using the term.

which also contains other objects. The framework may be local, such as a room or a building, or it may be more extensive, such as the city, the continent, or the universe. Persons and objects are located within these spaces but do not define them in any fundamental sense - remove any object and space is left behind. The space itself is immovable and continuous - there are no gaps in it. Although one can think of small parts of it, these are always recognized as portions of a whole. This notion of a connected, total space is what is meant by absolute space. Everything occurs within it, and it serves as the basic framework tying the universe together into a coherent whole.

In addition to this unitary environment, other notions of space come to mind. These are usually more restricted in scope and usually relate directly or indirectly to features of the observer's body. Thus an object is located to the left of me now, but to my right after I make an about-face. Higher order relative spaces can also be imagined. Object A is to the left of B, behind B, or between B and C. In these higher-order spatial relationships, objects are located with respect to each other in terms of their relation to the observer. Take a stroll and A ends up to the right of B or in front of it. In general, this type of relative space moves with the observer. Consequently changes in his position can alter the relations of objects in the schema.

A different type of relative spatial relation appears less bound to the observer: A is near to, around, in the neighbourhood of B. While these topologic relations are independent of the position of the observer, they are rather dependent on the context. Is Edinburgh near London? Yes, in a global context; no, in a British one. We suspect that examination of this context will reveal a full-blown concept of space and that seemingly simple topologic concepts covertly presuppose such spatial frameworks.

Finally there is the space occupied by things, the *extension* of a body or an object. This is clearly related to that body and travels with it.

All of these non-absolute spaces are logically separable, although they may be interrelated. They are all set within the unitary spatial framework we have called absolute space, and it is within this framework that they move relative to each other. These spaces all fall under the rubric of relative psychological space.

(iii) Innate v. learned or constructed. This obviously only applies to psychological space. By innate or *a priori* is meant that the structure and function of those parts of the brain involved in generating the space are specified by information in the organism's genes. The alternative is that their structure and function is determined by the specific experiences of the organism. We include in this class of *a posteriori* theories both those which postulate a direct influence of experience in organizing neural spatial structures and those which view space as a concept generated by non-spatial cognitive operations of the brain. It should be pointed out that there are intermediate positions between the two extremes. One possibility close to the *a posteriori* side is that some parts of the structure are genetically determined, and others are dependent upon experience. This may be merely a restatement of the antithesis at the molecular level. Another possibility somewhere near the middle is the notion that genetic instructions might specify several alternative structural wiring diagrams, the one to survive being selected by the experience of the particular organism. Finally, it might be that the structure and function of those neural systems concerned with space are genetically specified but that particular experiences determine such things as the efficiency of the systems, how much reliance the organism will place on the information gathered in and generated by them, or how ready it is to act upon that information. These intermediate positions make differing claims about the extent and nature of the genetic endowment concerning psychological space, as we shall see later when we discuss particular theories representing some of these possibilities. Of course, all theories, *a priori* and *a posteriori* alike, accept that the specific contents of representations gathered within these spatial systems are derived from experience. The difference lies in what they say about how these representations are structured, and from whence this form of structuring, or framework, derives.

An *a priori* theory makes no claims about the nature of physical space but instead directs attention to questions of how psychological spaces evolved, what survival advantages they offer organisms, and how genetic instructions programme the development of the involved neural structures. Some *a posteriori* theories are interested in the nature of physical space while others are not. Consider a version of *a posteriori* psychological space which holds that it was learned on the basis of experience with physical space. This naive realist view would predict a close correspondence between the two and, therefore, as we have pointed out above, evidence of discrepancies will count against this model. A different theory is that psychological space could be constructed on the basis of experience with non-spatial aspects of the physical world such as objects or stimuli. As this position has considerable influence in the history of psychology we shall examine it carefully in this chapter to see how tenable it is. We shall be particularly interested in two of its assumptions: first that organisms can identify and re-identify particular objects or stimulus configurations without recourse to a pre-existing spatial framework, and second that organisms can select and group together those movements and only those movements which are successful in re-establishing an original sensory array which has changed. One, or perhaps both, of these abilities are the minimal requirements for the generation of a psychological space from spaceless data; the first because it is supposed to give a reliable set of items upon which one can begin to build a set of (spatial) relationships, and the second because it allows such spatial notions as distance to be extracted from non-spatial information about the animal's movements. Since it is the contention of those who support the theory of an innate psychological space that these abilities are not possible in the absence of such a space or of some equally powerful set of innate ideas, we shall probe deeply to test their credibility.

(iv) Euclidean v. non-Euclidean metric. While the question of metric can be applied to either physical or psychological space, we are primarily concerned with the latter. The demonstration in the middle of the nineteenth century that non-Euclidean geometries and spaces of more than three dimensions were mathematically coherent and comprehensible, if not necessarily visualizable, led to questions as to whether physical space had a true metric or whether it was a matter of convenience which metric was chosen, or even whether it had a metric at all. These doubts were naturally reflected by similar questions about the metric of psychological space. In this book we shall be sticking our necks out and taking the strong position that the metric of the cognitive map is Euclidean, although psychological spaces associated with other neural areas may not be so. In the final analysis the evidence on the metric of the map must come from behavioural studies on such spatial abilities of animals as homing and triangulation and from studies on the physiology of the spatial nuclei such as the hippocampus. In this chapter we shall content ourselves with pointing out that any evidence which suggests that physical space does not have a Euclidean metric or that physical space can be described equally well by a Euclidean or non-Euclidean metric, far from counting against a Euclidean metric for psychological space, leaves the latter as one of the only possible sources of Euclidean notions and suggests that the mind (or a part of it) has a strong affinity for the laws of Euclid.

In this review we start with Berkeley, Newton, and Leibniz, in whose writings we can discern the original formulations of several lines of thought which persist to the present. Equally importantly, these writers serve as a good introduction to the work of Kant whose theory of psychological space, with suitable modification, will be adopted in this book. While Newton, Leibniz, and Berkeley differed on the nature of physical space, they agreed that psychological space was relative and derived from experience. Kant, on the other hand, asserted that psychological space was absolute and *a priori.* In the rest of the chapter we trace the development of the different theories of psychological space along empiricist, nativist, and Kantian branches down to their current formulation by modern psychologists such as Hull, Gibson, and Piaget.

1.2. Newton, Leibniz, and Berkeley

1.2.1. NEWTON'S UNIVERSE

In a famous series of letters Clarke and Leibniz (cf. Alexander 1956) debated the merits of two radically opposed conceptions of the nature of space: Clarke defended the validity of Newton's concept of absolute space, while Leibniz argued that only relative space existed. In the *Principia* Newton had spoken of these two types of space:

> 'Absolute space, in its own nature, without relation to anything external, remains always similar and immovable. Relative space is some movable dimension or measure of the absolute spaces; which our senses determine by its position to bodies' (Scholium to Definition VIII, Number II, Cajori 1934).

In conjunction with absolute time, absolute space provided the basis for the omnipresence of the deity. Perhaps more important, absolute space was thought by Newton to be central to his physical theories. Though it described the structure of the universe, absolute space was not immediately accessible to the senses. Thus,

> 'because the parts of space cannot be seen, or distinguished from one another by our senses, therefore in their stead we use sensible measures of them. For from positions and distances of things from any body considered as immovable, we define all places; and then with respect to such places, we estimate all motions, considering bodies as transferred from some of those places into others. And so, instead of absolute places and motions, we use relative ones; and that without any inconvenience in common affairs' (Scholium to Definition VIII, Number IV).

Notwithstanding its inaccessibility to the senses, Newton felt that the existence of absolute space could be inferred from its effects upon matter, in particular from certain forms of motion.

The theory of absolute space, as conceived by Newton, relied upon a particular notion of the universe, which saw it composed of matter and the absolute space containing that matter. God created matter in the form of solid, impenetrable particles, or atoms, and these atoms were embedded in space. Though small, atoms had both mass and extension and should, with suitable advances in optical techniques, eventually be visible to the eye. Atoms were separated from one another by void or empty space. In combination with each other they formed the objects or bodies of the sensible world. The movement of bodies and their interactions with one another constituted, for Newton, the science of dynamics, and it was within this context that the notion of absolute space became critical. Newton distinguished between absolute and relative motions in terms of the forces acting on bodies that cause motion. Thus,

> 'True motion is neither generated nor altered, but by some force impressed upon the body moved; but relative motion may be generated or altered without any force impressed upon the body. For it is sufficient only to impress some force on other bodies with which the former is compared, that by their giving way, the relation may be changed, in which the relative rest or motion of this other body did consist' (Scholium to Definition VIII, Number IV).

True, or absolute, motion could not be referred to other bodies, but rather must be referenced to some absolute framework. The classic case, discussed in detail by Newton, concerned centrifugal motion, as in the rotation of a pail of water. Here, the centrifugal force which pushes water up the sides of the rotating pail occurs independently of the relative motions of pail and water. Newton argued that this force can only be comprehended through the assumption of absolute motion, and that absolute motion demanded the notion of absolute space. Thus, we could not sense these absolute entities, but we could validate their existence through their effects.

1.2.2. LEIBNIZ'S UNIVERSE

Leibniz was a philosopher who, while accepting Newtonian physics, categorically rejected its metaphysical presuppositions. He could not accept Newton's atoms as the fundamental building blocks of the universe; he could not accept the notion of the void in which these atoms were supposed to float; he could not accept the mysterious forces through which one body reached across the void to influence another. Instead, he viewed all these as secondary manifestations derived from a more fundamental metaphysical system. Here there was no matter, no causal interaction, no time, no space; all that existed was an infinitude of simple non-corporeal substances living out their destiny in total isolation from one another. This deep metaphysical level Leibniz called the monadic realm, and its inhabitants he called *monads.*

In order to see how Leibniz derived the concept of space from the monads it will be necessary to discuss some of their properties. Here, we can give only the flavour of his ideas; the interested reader should refer either to Leibniz's own writings (cf. Parkinson (1973), or to the books by Russell (1937) and Rescher (1967). Unlike Newton's atoms, the monads had no mass nor did they have extension. They were idealized mathematical points possessing a rudimentary form of consciousness, which Leibniz likened to that of a deep dreamless sleep or coma. These monads existed in infinite numbers and taken together they constituted the universe. If it made any sense to ask the question: what is there between two monads?, the answer would be another monad. The defining characteristic of each monad was its continuously changing internal state. The successive states of a monad, past, present, and future, were predetermined by God at the creation of the universe. Each monad had its own built-in programme which, once set in motion, carried on inexorably throughout eternity. Leibniz specifically denied that there could be interactions of any sort between monads. The order exhibited by the world was due not to such interactions, but rather to the harmonious relationship amongst the programmes of the monads established at creation. At any given time the state of a single monad reflected the simultaneous states of all the other monads. To understand fully any one monad

it would be necessary to understand the entire universe. This notion of the relationship between the different monads can be put in another way, and compared with Newton's idea of causal interactions. Newton's cosmological musicians achieved their symphonic unity by listening to and playing along with each other. Leibniz's harmony arose out of the isolated efforts of his insensate musicians, each in command of a degraded version of the symphony, each playing his part to the music in his head.[*]

Each monad was unique in the sense that its internal programme reflected the whole from a particular point of view. None the less, there was some similarity between all of the monads, and some were more alike than others. Leibniz couched this relationship between monads in perceptual terms: a monad 'perceived' another more clearly if it had a similar programme, while dissimilar monads were 'fuzzy'. Thus, although in theory a monad reflected all the other monads, in practice its knowledge of some was better than of others. Furthermore, this 'perception' of one monad by another was not reciprocated by an equally clear perception in the opposite direction. The importance of this asymmetry will become apparent as we turn to Leibniz's theory of space.

How did Leibniz derive such concepts as extension, object, and space from this elegant but uncomfortably strange universe? He did so at two separate levels. In much of his writing he stuck to the metaphysical level and showed how certain properties of the monads and their relations could be equated more or less arbitrarily with extension, object, and space. In the correspondence with Clarke he ventured into the phenomenological domain and tried to give an account of the genesis of psychological space.

In the realm of the monads Leibniz viewed the emergence of extension as involving the same process by which mathematical points make up a line. Certain clusters of monads would have programmes which were so similar that from the point of view of most other monads they would be indiscernible. They would then appear as the repetition of the same monad; this defined extension. More complicated clusters of linked monads were 'perceived' as objects. Space was also derived from the 'perceptions' of monads. From the point of view of any particular monad, all of the other monads could be ordered in terms of their similarity to the perceiving monad. This set of ordered relations was the basis for space in the monadic realm. Leibniz equated 'A is a clearer monad than B' with 'A is closer than B'. The space derived in this way is relative to the point of view of the 'percipient', and because of the asymmetrical nature of intermonadic perception the distance between two monads would be viewed as different by each monad. In this way it was possible to maintain that every

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[*] This analogy can be extended in the following way, to incorporate one of the major theological differences between Newton and Leibniz: for Newton the composer of the symphony had occasional recourse to altering the course of the music, making minor adjustments here and there; for Leibniz the composer was simply an observer, capable of seeing the symphony in its entirety, but not influencing it.

monad 'perceived' the universe in its unique fashion. In theory, this meant that every monad could be identified by its perception, and there would be no need of any spatio-temporal framework to assist in identifying or locating monads.

At a psychological level a similar process was assumed to be in operation. This is hardly surprising, since Leibniz viewed minds as a single monad dominating a complicated aggregate of monads. But minds did not directly perceive individual monads; rather, they were sensitive to monad clusters in the form of extended objects. The process by which space of the monadic realm was reflected in the mind was summarized by Leibniz:

> 'I will here show, how men come to form to themselves the notion of space. They consider that many things exist at once and they observe in them a certain order of coexistence, according to which the relation of one thing to another is more or less simple. This order, is their *situation* or distance. When it happens that one of those coexistent things changes its relation to a multitude of others, which do not change their relation among themselves; and that another thing, newly come, acquires the same relation to the others, as the former had; we then say, it is come into the place of the former . . . then we may say, that those which have such a relation to those fixed existents, as others had to them before, have now the *same place* which those others had. And that which comprehends all those places, is called *space.* Which shows, that in order to have an idea of place, and consequently of space, it is sufficient to consider these relations, and the rules of their changes, without needing to fancy any absolute reality out of the things whose situation we consider' *(Leibniz's Fifth Paper,* in Alexander 1956, p. 69).

For Leibniz, space was read off the relations between objects, but only after an intermediate step involving the construction of a network of places. Thus, places were dependent upon the presence of extended objects. This put Leibniz in the position of claiming the ontological priority of extension over space, a view common to all relative theories. It is in this sense that such theories hold that space cannot exist in the absence of objects.

1.2.3. BERKELEY'S UNIVERSE

Whereas Leibniz sought to explain the existence of bodies and absolute space on the basis of monads, Berkeley denied that they existed independent of minds and their contents, ideas. There were two types of ideas: sensations which were impressed on the mind from outside (in the final analysis by God), and images of these sensations which were conjured up by the mind itself. Typical sensations were the colour red, a round shape, a sharp edge, a loud hum. The difference between an externally impressed sensation and an internally generated image lay in the greater strength, orderliness, and coherence of the former. Other notions, such as the idea that solid, impenetrable bodies existed in an external world and gave rise to sensations in the mind, were denied by Berkeley. Instead, such bodies were constructed by abstraction from sensations and their combinations and sequences. For example, when the mind repeatedly experienced one bundle of sensations followed by another sensation or sensations it conjured up the notion of bodies and causality, attributing power to the first sensations in producing the second:

> 'Thus, for example, having observed that when we perceive by sight, a certain round, luminous figure, we at the same time perceive by touch the idea or sensation called heat, we do from thence conclude the sun to be the cause of heat, and in like manner perceiving the motion and collision of bodies to be attended with sound, we are inclined to think the latter an effect of the former' *(Human Knowledge*, in Turbayne 1957, p. 38).

But neither causality nor bodies nor any of these derived ideas actually existed aside from our thoughts about them. Stop thinking of them and they disappear (unless, of course, someone else continues to think of them).* Our primary concern here is with space and related concepts such as distance, so we shall turn directly to Berkeley's account of how the mind builds up these secondary notions.

Berkeley first drew a distinction between the notion of space derived from tactile information (which for him included proprioceptive feedback during movement) and that derived from visual information. His notion of the primacy of tactile information, and hence *haptic space*, is important historically and recurs in later writers. It rested on observations that the radical changes in visual and auditory sensation produced by changes in distance were ruled out for tactile sensation. According to Berkeley the primary notions of bodies and space which we form on the basis of tactile sensations were due to the differential resistance produced by the same movements at different times:

> 'When I excite a motion in some part of my body, if it be free or without resistance, I say there is *space*; but if I find a resistance, then I say there is body; and in proportion as the resistance to motion is lesser or greater, I say the space is more or less *pure.* So that when I speak of pure or empty space, it is not to be supposed that the word 'space' stands for an idea distinct from or conceivable without body and motion - though indeed we are apt to think every noun substantive stands for a distinct idea that may be separated from all others; which has occasioned infinite mistakes. When, therefore, supposing all the world to be annihilated besides my own body, I say there still remains pure space, thereby nothing else is meant but only that I conceive it possible for the limbs of my body to be moved on all sides without the least resistance; but if that, too, were annihilated, then there could be no motion, and consequently no space' *(Human Knowledge,* in Turbayne 1957, p. 81).

These notions of proximal tactile space and tangible bodies served as the foundation onto which was grafted our notion of visual space. Prior to

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* Sometimes Berkeley trivializes the problems generated by this difficulty by asserting that bodies do not disappear when we stop thinking about them since God perceives them. Here of course God begins covertly to take on some of the properties of external absolute space.

Berkeley it was thought that knowledge of the distance of an object depended on an innate connection to such phenomena as the amount of convergence of the eyes, the disparity of the rays entering the eye from an object, the number of intervening objects, and the distinctness of the object. As we shall see, this point of view was rehabilitated in the nineteenth century. Berkeley, however, showed how all of these were only contingently related to distance and thus must have been learned by association with tangible space:

> 'and I believe whoever will look narrowly into his own thoughts and examine what he means by saying he sees this or that thing at a distance, will agree with me that what he sees only suggests to his understanding that after having passed a certain distance, to be measured by the motion of his body, which is perceivable by touch, he shall come to perceive such and such tangible ideas which have usually been connected with such and such visible ideas' (*A New Theory of Vision*, in Luce and Jessop 1948, p. 188).

The role of visual space was predictive; it informed the mind beforehand what tactile sensations were contingent on which movements and what visual sensations to approach or avoid.

Thus, for Berkeley there were several types of relative space, depending on different modalities. These were connected to one another through associations derived from experience, with touch serving as the basis. In his early writings Berkeley suggested that the tactile sense actually gave one access to the physical universe. Later, he rejected this notion, claiming that he had only adopted it in order to facilitate acceptance of his radical ideas. A rigorous application of these ideas meant that no contact with an external universe, if one existed, was possible. These ideas naturally led Berkeley to reject completely the Newtonian concepts of rigid body and absolute space. Physics was the study, not of material bodies, but rather of the succession of sensations programmed in the mind by God. Berkeley's rejection of Newton's absolute inertial framework led him to search for alternative solutions to Newton's problems. Thus, for example, he concluded that centrifugal motion, which Newton cited as evidence for absolute motion and absolute space, was really movement relative to the outer shell of the universe, the stars.

1.2.4. A COMPARISON OF THE THEORIES

It would be useful at this point to pause and summarize the answers given by these three authors to the questions posed at the start of this chapter. We can also discuss the strong points of each of the theories, as this will serve to introduce the work of Kant, the writer who effectively combined these strong points into a single powerful theory of space.

(1) *Does physical space exist and what is its form?*
The answers given by our authors cover the entire spectrum. At one extreme stood Newton, who believed that physical space existed external to, and independent of, any and all conscious beings, that its form was absolute, and that, although it could not be experienced directly, its influence on physical bodies could be demonstrated. At the other extreme, Berkeley and Leibniz both denied the existence of physical space, but for different reasons: Berkeley as a corollary to his thesis that nothing existed outside of our thoughts, Leibniz because there was no role for space in the realm of his non-corporeal monads.

(2) *Does psychological space exist and what is its form and origin?*
Here, in contrast to their opposed positions on physical space, there is virtual agreement amongst our authors that psychological space exists and that its form is relative. Furthermore, they all felt that this relative psychological space was manufactured by the mind as a consequence of its experience with bodies or sensations. Three possible recipes for constructing a relative psychological space were suggested: (a) map out the regions of least resistance to bodily movements, call that space; calculate the amount of movement between sensations, call that distance (Berkeley); (b) notice which bodies do not change position relative to each other and use these as a reference frame to define places, and thence space (Leibniz); (c) pick some reference body which is assumed to be stable relative to absolute space, then calculate the positions and motions of other bodies relative to this *inertial* frame (Newton). The first two recipes stressed the primacy of the concept of body or sensation over that of space, while the last did not. This central disagreement between the models reflects the opposed order in which they derived the concepts of space, object, and extension; this opposition is inherent in the absolute/relative space dichotomy. For both Berkeley and Leibniz, extension had logical priority, leading to objects which then defined space. For Newton absolute physical space existed without objects or their extension, and relative psychological space was an approximation to this. Newton was left with a curious position on absolute space; it existed in the physical world, but not directly in the psychological realm. Yet,

> 'in philosophical disquisitions we ought to abstract from our senses, and consider things themselves, distinct from what are only sensible measures of them' (Scholium to Definition VIII, Number IV).

The ultimate validity of absolute space was physical rather than psychological, and Newton offered no simple formula for the translation from one realm to the other. The problem raised by this conception of absolute space formed part of the motivation behind Kant's work, as we shall see shortly.

The consequences of the different way in which psychological space was derived by these writers can be seen in later approaches to this problem.

In particular, Berkeley and Leibniz spawned psychological models persisting to the present day. Though they agreed on the primacy of extension and the secondary nature of space, their derivation of extension differed. Berkeley derived it from movement, and his emphasis upon the role of movement remains a constant feature of *empiricist* theories. Leibniz, on the other hand, derived extension from a kind of monadic 'perception', and can thus be viewed as a forerunner of the *nativist* position, which assumes that spatial information is somehow extracted from sensations.

(3) *What is the relation between physical and psychological space?*

Berkeley excused himself on this one through denying the existence of physical space, but both Newton and Leibniz provided answers, albeit vaguely formulated ones. Newton simply asserted that relative psychological space, with its dependence on relative frameworks, was an approximation to absolute physical space. Leibniz's account of the relation between the two spaces was also vague, but more interesting in one of its implications. For him, psychological space was a translation into the phenomenal world of the underlying relationships between monads, based on their mutual perceptions in accordance with the pre-established harmony. An implication of this is that the contents of the mind can reflect the external world in spite of an absence of communication between the two.

(4) *What is the function of space?*

The general answer is that absolute space performs work, while relative space is little more than an epiphenomenon. The absolute space of Newton was an integral part of his universe and added something to our knowledge of that universe. In contrast, relative spaces of all kinds were derived from other knowledge and served as shorthand for, or generalizations from, that knowledge.

Each of the three theories we have discussed had at least one strong point in its favour. Newton's advocacy of absolute physical space rested on the role this concept played in his physics, and the empirical success of that physics. Berkeley's epistemological analysis cast grave doubts on the ability of conscious beings to know anything about the existence or nature of the external world. Finally, Leibniz provided one example of how notions applicable to the external world, such as space and objects, could be manufactured by conscious entities totally isolated from that world. It was these three ideas that Kant synthesized into his theory of space.

1.3 Kant

Throughout his life Kant grappled with the problem of the metaphysical foundations of Newtonian physics and Euclidean geometry, both of which seemed necessarily true of the physical world, but neither of which, he felt, could be grounded in sensation. Kant could not accept the gap left in the Newtonian system between the notion of absolute space underpinning the physical laws, and the notion of relative space which organisms generate from their interaction with physical bodies. Induction seemed too weak a basis to sustain the notion of infinite, everlasting, absolute space. Furthermore, Kant was deeply impressed by the arguments of Berkeley and Hume that conscious minds could have no access whatever to the physical world (if it existed). His resolution of these problems, reached in the *Dissertation* of 1770 (cf. Handyside 1928) and the *Critique of pure reason* (1787),* was that space was indeed absolute, but that it was not a property of the physical world. Rather, it was an innate organizing principle of the mind, by which the sensations derived from the physical world were constructed into a conscious manifold. Space was a *way* of perceiving, not a thing to be perceived.

The steps by which Kant arrived at this solution are not crucial to our argument, and we shall only sketch in their outlines. The interested reader is referred to Handyside (1928) for some of Kant's earlier writings on space and to Garnett's (1939) discussion of these writings. Briefly, Kant's initial position on the nature of space was a compromise between Leibniz and Newton. From Leibniz he took a relative notion of space, but unlike Leibniz he derived this space from the interaction of substances through Newtonian forces.

> 'It is easily proved that there would be no space and no extension if substances had no force whereby they can act outside themselves. For without a force of this kind there is no connection, without this connection no order, and without this order no space' (*Thoughts on the true estimation of living forces,* in Handyside 1928, p. 10).

Note that while accepting Leibniz's relative space, Kant firmly rejected his notion of pre-established harmony. Only through interactions could relative space be derived. However, Kant's synthesis was essentially circular and clearly inadequate. Space was derived from the interaction of bodies through forces, but the very existence of bodies and forces seemed to presuppose space. Further, a reliance on forces as the wellspring of space failed to explain all of the latter's properties; in particular, it could not account for the three-dimensionality of space. At this time the only evidence that Kant could adduce for this property was the inability of the imagination to visualize spaces of other than three dimensions; here, he pointed towards one of the arguments he was to use later.

Following several other unsuccessful attempts to provide a relativist account of space, Kant shifted to a Newtonian position in which he accepted the necessity of a concept of absolute space:

> 'absolute space has a reality of its own, independent of the existence of all matter, and indeed as the first ground of the possibility of the compositeness of matter' *(On the first ground of the distinction of regions in space,* in Handyside 1928, p. 20).

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* All references to the Critique are to the 1787 version, as translated and edited by N. Kemp Smith.

The major evidence Kant used in support of the necessity for a notion of absolute space concerned objects which were similar but incongruent, such as left and right hands, left and right screws. The parts of these objects and their internal relations had exactly the same description and yet they could not be superimposed on each other because the three-dimensional space they occupied was different. In other words, part of the description of a left hand involved a reference to the space in which it was set. If there were only one hand in the universe, it would be impossible to say whether it was a left or right hand without recourse to an absolute spatial framework. At this point Kant was still thinking about absolute space in the Newtonian way, as a property of the physical world. His shift to a psychological interpretation of absolute space was due to Leonhard Euler's influence. Euler suggested that space was purely psychological but that it was not derived, as previous writers had thought, from sensations originating in the external world, nor from minds reflecting on these sensations. Using this insight of Euler, together with his earlier arguments in favour of the necessity of absolute space, Kant formulated his mature theory of space.

In the *Dissertation* of 1770 and the *Critique of pure reason,* Kant developed a theory of knowledge in which the psychological notion of space played a fundamental part. It is necessary first to consider the general theory of knowledge before turning to the specific role of space. Kant began by classifying propositions in two different ways: the first in accordance with their origins, the second in accordance with the means available to validate them. In addition to knowledge which originated from the external world and could therefore be called *empirical,* or *a posteriori,* Kant identified a category of *a priori* knowledge, defining it as

> 'knowledge that is ... independent of experience and even of all impressions of the senses' (*Critique*, p. 42).

This knowledge took the form of

> 'rules which I must presuppose as being in me prior to objects being given to me'

and

> 'concepts to which all objects of experience necessarily conform, and with which they must agree' (*Critique*, p. 23).

The other way that Kant classified knowledge was based on the means by which the validity of propositions about the world could be verified. The first type of logical proposition, which he called *analytic,* consisted of a subject and a predicate, where the predicate was contained in the meaning of the subject. Definitions are the paradigm analytic propositions. The truth of analytic propositions is tested by unpacking the meaning of the terms in the subject and checking these against the predicate. In contrast, *synthetic* propositions were ones which conjoin a subject and a predicate, neither of which implies the other; these can be verified with reference to the external world.

The obvious combinations of these two classes are *analytic a priori* and *synthetic a posteriori.* In the first, the predicate is contained in the meaning of the subject and thus can be validated by the mental process of unpacking the meaning of the subject; in the second, the validity of the relationship between subject and predicate must be sought in the external world. However, in addition to these two, Kant proposed a third combination, *synthetic a priori.* These were propositions in which the meaning of the subject did not imply the predicate but which could not be validated empirically; instead, this had to be done with reference to purely mental processes. One example Kant gave was the geometrical axiom that 'a straight line is the shortest distance between two points'.

Kant maintained that our notion of space is a *synthetic a priori* which, though not derived by the individual from the world, gave certain knowledge of that world. A little background on Kant's ideas of how the mind worked would help in understanding what he was saying here, for the concept of *synthetic a priori* knowledge is a highly controversial and important one. Kant took a moderate Berkeleyan position on the relation between minds and the external world. He did not follow the Berkeleyan road all the way to its solipsistic conclusion that nothing existed except the contents of consciousness, but settled with the position that, although the external world existed outside of our minds, we could never know anything about it as it was in itself. Knowledge was necessarily derived from consciousness, and the world which existed apart from consciousness was ultimately unknowable.

According to Kant the mind represented the external world in two different ways, each derived from a different mental faculty. The faculty of sensibility constructed representations (called intuitions) which consisted of particular events and objects set in their spatio-temporal context; the faculty of understanding listed each object under the abstract concepts of which it was an instance. Thus, the sensibility would represent a red ball as that particular ball next to the blue chair. In the understanding, it would be listed under redness, balls, etc.

Within the faculty of sensibility, Kant distinguished between two types of intuition: empirical intuitions, such as impenetrability, hardness, and colour which derived from the unknowable outer world, and pure intuitions such as extension which were wholly due to the structure of the sensibility itself. The former constituted the matter of the representation while the latter gave it its form. It was Kant's contention that space was a pure intuition in the sensibility as opposed to an empirical intuition or a concept in the understanding.

Following Euler's reasoning, he denied that space was a concept; it was not abstracted from several different instances of space, since these more limited patches of space were always conceived as parts of a single, infinite space. On the other hand, space was not an empirical intuition either, since it was a necessary precondition for objects to be perceived in the first place:

> 'Space is nothing but the form of all appearances of outer sense. It is the subjective condition of sensibility, under which alone outer intuition is possible for us. Since, then, the receptivity of the subject, its capacity to be affected by objects, must necessarily precede all intuitions of these objects, it can readily be understood how the form of all appearances can be given prior to all actual perceptions, and so exist in the mind *a priori,* and how, in a pure intuition, in which all objects must be determined, it can contain, prior to all experience, principles which determine the relations of these objects' *(Critique*, p. 71).

Particular objects in an intuition, or concepts in the understanding associated with these objects, did not have the same necessity about them as did space. A ball did not have to be red, nor did it have to be located in a particular place. On the other hand, a part of the house containing the room, a space for the room, a place of the ball, all these were necessary and could not be thought away:

> 'If we remove from our empirical concept of a body, one by one, every feature in it which is (merely) empirical, the colour, the hardness or softness, the weight, even the impenetrability, there still remains the space which the body (now entirely vanished) occupied and this cannot be removed' *(Critique,* p. 45).

The fact that space could not be annihilated and that it had to take a certain form constituted strong arguments for Kant's view. Further, his notion of space provided a solid epistemological basis for the postulates of Euclidean geometry and Newtonian physics, showing that these could be immediately and innately available to the mind and yet provide knowledge applicable to the external world.

Unfortunately, Kant did not provide an analysis of how this innate knowledge arose. He postulated that we can have no knowledge of the external world as it is in itself, and that the ultimate validity of the notion of absolute space derived solely from the fact that it was part of our mode of perceiving, an intuition. Yet, this space synthesized by the mind, this mode of perception, must have some correspondence to the physical world if it is to be useful to the organism. How could this come about? How, in other words, do *synthetic a priori* intuitions arise? Had Kant been a Darwinian he might have developed something like an evolutionary version of Leibniz's pre-established harmony; in fact, present-day writers concerned with the origins of psychological knowledge (e.g. Piaget 1971b) interpret Kant in this way. Let us briefly spell out what this might mean.

The external world has a structure which cannot be directly and totally perceived by any particular organism. Let us assume (with some modern physicists) that it is a sort of n-dimensional energy soup. Animals on all levels of the evolutionary scale develop perceptual and classificatory systems which are sensitive to various aspects of this soup; these become their version of reality, or their *ambient.* One evolutionary development led to a set of systems which divided the soup sharply into discrete objects and provided a spatial framework for containing these objects. Ancillary notions such as causality and force were also developed to account for connected bits of the soup which could not be clustered into a single object.*

Kant emphasized the point that it is difficult for us to imagine how the world would appear in the absence of these particular intuitions. Yet, it might be argued, as Berkeley did, that this way of perceiving the world is not given *a priori* but must be learned anew by each member of the species. One major difference, then, between a Kantian theory and an empiricist one resides in the level at which learning about space occurs. Kant only makes sense if we assume that a species has 'learned' to partition the world into absolute space and the objects it contains, and that individual members of that species need not acquire this knowledge *de novo.* Empiricist theories, on the other hand, must assume that each individual learns about space and objects in its own way. While this confers the advantage of flexibility on the individual members of the species, it conflicts with the notion that concepts of space must obtain a necessary form.

There are three possible non-Kantian ways of circumventing this problem, and all of these have been tried at one time or another. First, one might deny that there are necessary ways of conceptualizing space; we shall see later that this claim has been made by the so-called *cultural relativists.* Second, one might suggest that we acquire notions of Euclidean space entirely from experience because we live in a roughly Euclidean world. If we lived in another type of universe we would have different notions about space. This, of course, is a strict empiricist model. Finally, one could assume that the concept of absolute space is not given *a priori,* but that perceptual and response systems are pre-structured in such a way as to determine that experience will lead to the formulation of this concept in each individual. A variety of such models have been proposed, differing in the nature and extent of this pre-structuring. These models are not empiricist, but neither are they strictly Kantian, in that they deny the pre-formed status of three-dimensional Euclidean space. We shall be discussing various versions of all these non-Kantian models in the remainder of this chapter.

Leaving these alternative views aside for the moment, we can see that, by allowing for evolution, an innate spatial mode of perception could be developed which would confer upon the perceiver accurate knowledge of certain aspects of the external world. As it is this neo-Kantian position

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* As to the question of why this particular set of classificatory systems developed, rather than some other, we can provide no answer. For instance, it is not obvious why we perceive space as three-dimensional, though it is clear that we do.

which we shall be adopting in this book, it is worth restating two main features of the argument:

1. Three-dimensional Euclidean space is a form imposed on experience by the mind.
2. This unitary framework, conveying the notion of an all-embracing, continuous space, is a prerequisite to the experiencing of objects and their motions.

The history of attempts to explain space perception after Kant revolves around theories which denied one or both of these tenets. It is our contention that no theory denying these principles can succeed.

Viewed in the way described above, Kant can be construed, albeit in the face of his own vigorous denials, as providing the basis for dividing the study of the natural world into physics and psychology. Unforeseen by Kant, physics has been able to develop techniques and languages for the description of the external world itself. The role of psychology, then, is to describe the innate features of the minds of different organisms which have evolved to match certain aspects of that physical external universe, and the way in which the physical universe interacts with the mind to produce the phenomenal world.

1.4. After Kant: nativism versus empiricism

By concentrating upon absolute space, and ascribing it to the innate machinery of the mind, Kant had the effect of largely removing the evaluation of his theory of space perception from the experimental arena. It is not surprising, then, that over the course of the next century, during which time experimental approaches to psychological problems began to gather impetus, the influence of Kant's model waned. There were, of course, other reasons for this and for the concomitant return to an emphasis upon relative space; in particular there were radical changes in first mathematics, and then physics, which undercut Kant's arguments based on Euclidean geometry and Newtonian physics. Whatever the reasons, the shift in emphasis from an absolute to a relative theory of space has had the most profound effect upon research in psychology.

Kant's postulation of an *a priori* absolute framework did not, by itself, provide a complete explanation for all aspects of space perception. He was virtually unconcerned with a series of more mundane issues such as the nature of depth and distance perception, localization within the spatial framework, and so on. In attempting to come to grips with these problems, those that followed Kant opened up a set of new difficulties and the investigation of these eventually led to the formulation of entirely new models for space perception, the so-called *nativist* theories. Common to all these models was the assumption that some portion of our spatial knowledge is independent of specific experience.

On the other side, the empiricists were equally interested in these problems. Their basic assumption, of course, was that all forms of knowledge about space, including its very existence, must be acquired through experience. For some time after Kant empiricists laboured at refining the position set out by Berkeley. In the course of these refinements one can detect a shift away from any interest in the notion of unitary space, a shift which matched the one occurring within nativist theory. Thus, the nineteenth century was characterized by a major change in the emphases of theories of space perception; in effect it was decided that absolute space was not important and that relative space held the key to a complete understanding of the problem.

1.4.1. EARLY VIEWS

The perception of space includes knowledge about the size, location, and distribution of things in a unitary three-dimensional environment. Kant concentrated on the last problem, leaving the first few problems to subsequent writers. There are only two possible classes of explanation for these: either there is information in the mosaic of stimulation which inherently specifies certain features of space, or these features are contingently derived from experience.

We have already discussed Berkeley's model, which includes assertions about the derivation of distance, location, and the idea of space itself, from experience. This formed the basis for future empiricist models, some of which we shall discuss later. The major problem faced by these models is simply stated: how can an organism learn about space, in all its aspects, from scratch? It is encumbent upon empiricist theories to demonstrate two things: (1) no knowledge of space is available prior to experience; (2) there are empirically verifiable means by which such knowledge can be derived from experience.

The other class of theories, termed nativist, assumes that some spatial knowledge is available prior to specific experience. Kant fits into this category, as do several who followed him and accepted the idea that the framework of unitary space was innate. There was another class of nativists, however, who accepted innate spatial knowledge without crediting the idea of an intuition of unitary space. This constituted the major shift away from Kant noted already, and led to a totally different type of theory. It is unfortunate that Boring (1942), in his classical review, failed to recognize this schism, for he has obscured the important differences between models which posit an innate framework and those which do not.* The requirements of a successful nativist theory vary with the features of space perception it takes as given. In general, it must provide evidence that there are mechanisms capable of automatically extracting the required information

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*Piaget, for instance, lumps Kant together with the later nativists and dismisses both for the same reasons (see p. 42n). Pastore (1974) has commented on this misinterpretation in Boring.

and that these do not depend upon specific experiences of the organism. In addition, if the theory leaves any aspects of space perception outside this innate umbrella, it must provide an explanation for how these are built up from what was innately available.

Both the early nativists and the empiricists working at the start of the nineteenth century were as yet unaware of a whole set of problems concerning space perception; these unfolded partly as experimental research burgeoned, in particular that concerned with visual space perception in humans.* Another factor contributing to the elaboration of new issues was the solution offered to the first problem attacked by the post-Kantian nativists, that of localization in three-dimensional space.

It will be recalled that Kant could offer no strong argument for the necessity of three-dimensional spatial intuition, nor did he discuss the problem of localization within that space. Lotze (1886) described the issue in this way:

> 'Let it be assumed that the soul once for all lies under the necessity of mentally presenting a certain manifold as in juxtaposition in space; How does it come to localize every individual impression at a definite place in the space intuited by it?' (pp. 51-2)

1.4.1(a). The nativist solution. Writing shortly after Kant, Muller offered a solution to the problem of three-dimensional localization, set within his essentially Kantian 'doctrine of specific nerve energies'. This doctrine held that when a nerve was stimulated the sensation elicited was a property of the nerve itself (or its termination in the central nervous system) and not of the physical nature of the stimulus. Thus, for example, a mechanical blow to the eye resulted in a visual sensation. In addition, however, nerves could signal some aspects of the physical world as well owing to their sensitivity to vibrations, chemicals, heat, and electricity. One of these qualities of the world in itself was extension, and the visual and tactile modalities were best suited, by virtue of their structure, to signal it:

> '. . . inasmuch as the nerves of the senses are material bodies, and therefore participate in the properties of matter generally occupying space . . . they make known to the sensorium, by virtue of the changes thus produced in them by external causes, not merely their own condition, but also properties and changes of condition of external bodies ... All the senses are not equally adapted to impart the idea of 'extension' to the sensorium. The nerve of vision and the nerve of touch, being capable of an exact perception of this property in themselves, make us acquainted with it in external bodies ... The retina of the optic nerve has a structure especially adapted for this perception' (Muller in Herrnstein and Boring 1966, p. 33).

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* Our emphasis upon human visual space perception is necessitated by this historical fact. Where possible we shall introduce evidence from other sources, as our argument is meant to apply across all species and modalities.

According to Muller both the three-dimensional structure of space and the distribution of objects in that space were specified by the spatial positions of retinal elements activated by visual inputs. That is, the peripheral visual apparatus was itself extended in three dimensions; perception of both absolute and relative space was a simple matter of 'reading' the set of activated nerves.

Lotze expanded on this notion of the 'spatial' information transmitted by nerves in addition to the quality of the sensation. He called this extra information the local sign of the stimulus and noted that without some such information the mind would merge two identical or similar sensations arising from different areas of the world.*

Both Lotze and Muller argued for the innate basis of the intuition of space. In this they agreed with Kant and differed from those nativists who followed; these latter rejected both this claim and the unitary space it presupposed. Given that positional localization was being ascribed to certain sensations, it seemed sensible to drop the idea of an *a priori* spatial framework and rely solely upon spatial sensation. At least this would return the study of space perception to the laboratory. This new type of nativism, as espoused by Hering (cf. Hurvich and Jameson 1964) and James (1890), for example, differed from both the *a priorism* of Kant and the empiricism of Berkeley. It assumed that our notions of space were no different from those of colour: simply features to be extracted from the ambient stimulating environment. What remained to be determined was the nature of the stimuli involved and the means by which information was derived from them.

Hering's version of the theory of local signs supposed that each retinal element, when stimulated, provided information about its location. There were three types of local sign, one pertaining to each dimension of space. The stimulation derived from an object, for instance, automatically conveyed information about the spatial attributes of that object. Similarly, relations between objects could be read off the pattern of retinal stimulation.** All nativists agreed that there were sensations corresponding to, and evoking feelings of, space. However, there was no concentrated effort to explain why or how these sensations came to elicit spatial feelings.*** A related but, from our point of view, more important failing of most nativist models concerned their lack of interest in the means by which a unitary spatial framework was generated out of these sensations. Following Hering, it was usually assumed that simply specifying the basic spatial sensations

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* It is not clear exactly what form Lotze thought these local signs would take. He may have had in mind something like the topographic sensory maps of the body surface, retina, etc., which have subsequently been described.

** There is in all nativist models a strong strain of naive realism. Perceptions more or less mirror the physical world, which is directly perceived.

*** James admitted this failing of nativist theory when he stated that 'who calls a thing a first sensation he has no theory of its production' (James 1890, vol. 2, p. 280).

was all a theory of space perception had to do. This lack of concern reached its logical end-point with James (1890), who more or less denied the existence of any concept of unitary space:

> '... if any known thing bears on its front the *appearance* of piecemeal construction and abstraction, it is this very notion of the infinite unitary space of the world. It is a *notion*, if ever there was one; and no intuition. Most of us apprehend it in the barest symbolic abridgement: and if perchance we ever do try to make it more adequate, we just add one image of sensible extension to another until we are tired. Most of us are obliged to turn around and drop the thought of space in front of us when we think of that behind' (vol. 2, p. 275).

This attitude towards unitary space shows how far nativism had travelled from its Kantian beginnings. Extended objects automatically elicited the perception of a three-dimensional universe. The unitary space which dominated Kant's thinking was an unnecessary notion.

Mach (1897) was an important exception to this trend; while agreeing that sensations conveyed spatial information he felt that these could only produce positional localization for the single *field* perceived in one instant, or fixation. Mach suggested that what can be given in immediate sensation is limited to information pertaining to relative, or *egocentric,* localization, the position of things relative to the observer, and to each other, at that moment in time. In order to appreciate the world as a coherent, interconnected universe he felt it necessary to consider the information provided by movements of the eye, head, and body. The individual *frames* of experience provided by sensation could be spatially ordered quite easily. However, how were these individual frames to be combined into a connected series? Mach was the first nativist to realize the importance of the fact that we do not experience the world as a set of frames loosely interwoven with the seams still showing. Rather, these are effortlessly integrated into an overall conceptual space. According to Mach, this problem is solved through the intervention of motor feedback processes which guarantee, for instance, that

> 'the whole optical space appears to us a continuity and not an aggregate of fields of vision . . . at the same time, the optical objects remain stationary . . . Thus we arrive at the practically valuable conception of our body as in motion in a fixed space' (pp. 63-4).

In attending to these questions Mach showed that specifying the means for a static three-dimensional localization would not be sufficient in accounting for space perception *in toto*. He was also the only early nativist to pay attention to the conception of a fixed space.[*] None the less, he did not specify how motor processes brought about the integration of separate visual fields and the concomitant conception of fixed space. Was this an

unlearned mechanism or was it acquired? If the latter, and Mach seemed to lean in this direction, how was this learning accomplished? Finally, what form did the motor information take, and from where did it emanate? These questions, ignored for some time after Mach, still require answers, as we shall see later.

No further resolution of these issues was to come from the traditional nativist approach; further progress involved radical shifts in the theory, and these were not undertaken until the Gestaltists presented their new model. The temporary failure of nativism at the time was matched by similar problems in the empiricist camp, whose evolution after Kant we can now briefly review.

1.4.1(b) The empiricist solution. An emphasis upon objects and their localization, combined with a relative lack of concern with the concept of unitary space, also characterized empiricist theory in the nineteenth century. Empiricists disagreed with a fundamental assumption of nativist theory: that sensations contained inherently spatial information. Here, Kant and the empiricists found common ground: sensations were spaceless in content. Berkeley's empirical model laid the basis for a number of similar ones, involving refinements of the mechanisms by which associations formed between a succession of sensations could generate the concepts of extension and space.

After Kant empiricist theory developed quite slowly, perhaps because of the support derived by the Kantian position from the success of Euclidean geometry and Newtonian physics. Reid (1785) modified Berkeley's position while accepting his main argument that space is an empirically derived concept. He held that Berkeley's emphasis on tangible space was overstated, and attempted to show how tangible and visible space must be independent but necessarily interrelated. Thus

> 'The correspondence between them is not arbitrary, like that between words and the thing they signify, as Berkeley thought; but it results necessarily from the nature of the two senses' (p. 286).

That is, Berkeley held the strong empiricist position that any correspondence between visual and tactile space was a function of experience in matching the two: Reid, on the other hand, held that the two necessarily reflect one another. However, he did not specify what lay at the basis of this fixed correspondence, nor how these two types of space gave rise to the concept of unitary space. Much the same position was adopted by a number of his contemporaries, including Stewart (1818).

Some decades later a number of British Associationists continued this empiricist tradition. Spencer (1855), for example, argued rather forcefully against the Kantian model, raising objections to be encountered subsequently in the work of Poincaré (1913). Spencer first argued that the fact that

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[*] It should be noted that Lotze (1886) also recognized the importance of information from motor processes in articulating the meaning of local signs, but he did nothing more than mention this fact.

we cannot contemplate the annihilation of space does not confer upon space the unique status claimed for it by Kant. Instead, he suggested that, if we view space as 'an ability to contain bodies', then it is perfectly logical that we have acquired the knowledge that this ability cannot be annihilated. He went on to argue that this notion accounts for the fact 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' (p. 54).

That is, we cannot consciously hold an image of space which goes beyond the powers of our sensing apparatus. We cannot see both in front of and behind ourselves; thus we cannot conceive of a completely unified space.

As for how space concepts do arise, Spencer continued where Berkeley left off.

> '... whether visual or tactual, every perception of the space-attributes of body is decomposable into perceptions of relative position . . . all perceptions of relative position are decomposable into perceptions of the relative position of subject and object; and ... these relations of position are knowable only through motion' (p. 233)

This is as direct a statement of the rigidly egocentric concerns of empiricist theory as one can find, and it accurately sums up the situation which produced a total neglect of the notion of absolute space. Spencer attempted to show how the concept of position devoid of body, and thence the concept of space, were developed through experience and the processes of association. This was similar to the mechanism suggested by Berkeley, and we shall not repeat it here.

At about this time radical developments in geometry provided new impetus for empiricist theory. As we have seen from our study of Kant, geometry held a special place amongst the sciences. It was the paradigm science, a purely relational system which nevertheless faithfully and necessarily reflected the structure of the physical world. All of this changed in the middle of the nineteenth century with the discovery (invention) of non-Euclidean geometries by Riemann, Lobachevski, Bolyai, and others. These geometries were based on modifications in one of Euclid's original postulates, the so-called parallel postulate. This stated that through a point outside a line only one other line could be drawn parallel to the original line. This postulate had always been viewed with suspicion; it lacked the intuitive 'self-evidentness' of the other postulates and axioms and relied on such unobservables as the relationship between lines at infinity. During attempts to derive it from the other more intuitively obvious postulates, or to dispense with it altogether, it was noticed that other geometries, with total internal consistency, were possible. In these there were no parallel lines or even an infinitude of parallel lines. This discovery called into question the *a priori* status of Euclidean geometry and its assumed relationship to the physical world. No longer could it be maintained, as Kant had, that Euclidean geometry

> '... carries with it apodictic certainty; that is to say, absolute necessity, not based upon experience, and consequently a pure product of reason' (Prolegomena, Section 6).

Helmholtz was among the first to incorporate directly the new findings of mathematics into his theory of psychological space. Thus, he entitled one of his papers on the subject *Space can be transcendental without the axioms being transcendental* (cf. Warren and Warren 1968). While accepting the idea that the psychological spatial framework was innately given,* Helmholtz denied that the metric of this framework was inherently Euclidean. If, following Kant, one continued to insist upon the Euclidean nature of this framework, then one was in danger of being at variance with the physical world, should that world be best described, for example, by Riemannian geometry.

Helmholtz felt that the particular metric to be applied to our concepts of space had to be acquired through experience, though he did not specify how this would occur. Similarly, the meaning of local signs was also learned. Thus, Helmholtz was placed in the empiricist camp. He categorically rejected the nativist views of his contemporary, Hering, because such

> 'theories are always obliged to assume that actually existing *sensations* can be squelched by an experience showing them to be unfounded' (1910 p. 556).

That is, Helmholtz felt that if it was necessary to invoke experience to explain some spatial perceptions it was most parsimonious to explain all perceptions in this way; there was no need to talk about innately spatial sensations.** There was, of course, a fatal flaw in Helmholtz's theory, from

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* It is worth emphasizing the recurrence of the idea that our concepts of space are completely dependent upon sense-data limits. As we have seen, James stressed this point as well, showing that there was little disagreement here between empiricists and nativists. Much the same position was also adopted by Poincaré. We would agree that organisms do not 'see' absolute space; cognitive maps are not pictures of the universe, they are schemata from which any portion of space can be constructed. The fact that we cannot *perceive* unified space does not mean we cannot *conceive* it; the latter potentiality derives from the possession of a structure which can be used to construct spaces that stretch endlessly in all dimensions.

* Helmholtz did not discuss this issue at any length; at one point, however, he suggested that the psychological concept of unitary space is innately derived from the sense of touch. James (1890), taking note of this, dismissed Helmholtz in the following way: 'Of course the eye-man has a right to fall back on the skin-man for help at a pinch. But doesn't this mean that he is a mere eye-man and not a complete psychologist' (p. 279).

** A corollary of this view was also promulgated by Helmholtz when he stated that whatever 'can be overcome by factors of experience, we must consider as being itself the product of experience and training' (p. 13) This view led empiricists to concentrate upon demonstrating the ways in which different experiences could influence various perceptual functions. The demonstration of the effects of wearing prisms, begun with Stratton's (1897) work, is a prime example of this. It was held by empiricists that the study of adaptation would tell us something about the origins of space perception, by recreating the 'naive' state. This entire methodology, of course, suffers from a confusion between modification and generation, as Pastore (1960) and others have pointed out. The fact that certain experiences can affect space perception does not tell us anything about the origins of space perception itself. For this reason we shall not have much to say about studies exploiting this type of reasoning.

the empiricist point of view, in that he allowed an innate spatial intuition to creep in by the back door of touch.

A somewhat stronger empirical position was adopted by Wundt (1894). He argued that neither the sensations of touch and movement, nor those of vision were sufficient in themselves to account for space. Thus, he described the basic empiricist and nativist models as follows:

> 'It has often been thought possible to set up a theory in terms of movement and movement-sensations alone, and either entirely to neglect the local sensation qualities of the retina and skin, or to regard them as functioning in entire independence of movement, and as being, like the latter, sufficient in themselves for an adequate explanation of the facts . . . Experience shows that *these* two influences are so intimately connected that neither of them is operative without the other' (p. 168).

Instead, Wundt held what he described as a theory of 'complex local signs'. This theory

> '. . . regards the extensive idea as the mental resultant of intensively graduated local signs of the movement-sensation and qualitatively graduated local signs of the sensory surface. Space perception depends on the uniform association of these two sensation series' (p. 169).

These sensation series were developed through experience, and the perception of space, therefore, was a function neither of the innate structure of the mind, nor of particular properties of the sensations themselves. The catch in this, of course, is the phrase 'uniform association'. Wundt did not explain where this came from, nor how it functioned. If this association was automatic, then Wundt imported non-empirical notions into his model without openly saying so. He provides a good example of how difficult it is to be a thorough-going empiricist.*

1.4.1(c). Early views reviewed. In the course of elaborating these early versions of nativism and empiricism, investigators uncovered a host of new problems. Most of these revolved around the distinction between sensation and perception. Within both theoretical approaches the perception of space was seen as dependent upon some transformation of the former into the latter. Both demanded some measure of uniformity in this transition; unfortunately, this uniformity was lacking in a wide variety of situations.

Nativism suffered most from this discrepancy. Assuming, as it did, some fixed relationship between sensation and perception, this approach ran aground when confronted with the so-called *perceptual constancies.* This category of phenomena demonstrated that sensation (as received at the sensory surface, the proximal stimulus) could vary without any change in perception. The simplest of such phenomena, that of retinal localization invariance, shows that a form looks the same regardless of where on the retina it falls. This, in itself, can be handled by certain versions of early nativist theory. However, perceptual size invariance presents an insuperable problem. The distance of an object can vary (and thus the proximal stimulation must vary) without any change in perceived size. In order to account for this, and similar findings, nativism had to continually resort to the role of experience in explaining perception. This, as Helmholtz pointed out, was both uneconomical and unsatisfactory, and it led to the abandonment of the Hering-type nativist model.

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* James (1890) had the following to say about Wundt's theory: 'Wundt's theory is the flimsiest thing in the world. It starts by an untrue assumption and then corrects it by an unmeaning phrase. Retinal sensations are spatial: and were they not, no amount of 'synthesis' with equally spaceless motor sensations could intelligibly make them so' (p. 277-8).

Empiricism, on the other hand, could at least hope to explain these phenomena in terms of learning; it is required only that it be demonstrated that such constancies are acquired rather than innate. Thus, since size constancy is intimately connected with the perception of distance, it is necessary to show how the perception of distance develops through experience. However, there was another class of phenomena which embarrassed empiricists: the illusions. Here, perceptions did not correlate with sensations in the expected way, according to everything experience ought to have taught the organism. In attempting to explain both the constancies and illusions empiricism was forced to rely on such things as unobservable sensations and 'unconscious inferences' from these. This was not a particularly satisfying state of affairs.

At the start of the twentieth century there were, as James (1890) pointed out, only three possible theories of space perception:

> 'Either (1) there is no spatial *quality* of sensation at all, and space is a mere symbol of succession; or (2) there is an *extensive quality* given immediately in certain particular sensations; or, finally (3) there is a *quality produced* out of the inward resources of the mind, to envelop sensations which, as given originally, are not spatial, but which, on being cast into the spatial form, become united and orderly' (p. 271-2).

However, James dismissed the Kantian position,* while neither the empiricists nor the nativists had provided anything like a solution to the problems of space perception.

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* James called the Kantian view 'mythological' because he was 'conscious of no such Kantian machine-shop in my mind' and he had 'no introspective experience of mentally- producing or creating space' (p. 275). It is interesting that McDougall (1923), reviewing the same material, came to a rather different conclusion: 'spatial perception, as enjoyed by ourselves and the higher animals, is an extremely complex function, the capacity for which is not built up by each of us *de novo*, but is laid down in our innate constitution' (p. 245). In direct response to James he stated that 'the processes that go on in our heads can never be revealed in all their vast complexity to the introspective glance of any psychologist - not even of a William James' (p. 248). Pylyshyn (1973) and others have commented on the importance of representations which cannot be open to conscious experience.

1.4.2. LATER VIEWS

The difficulties faced by early empiricist and nativist models indicated that new departures were needed. These were provided on the one hand by Poincaré and on the other by Gestalt theory. The former attempted a thorough going empiricist model which would eschew such things as unconscious inferences and which would provide explanations for the development of space perception through experience. The latter reformulated the basis of nativist theory, denying the elementarism of Hering and re-incorporating the idea of a spatial framework, though in a limited way. Both of these new approaches owed something to the contemporary developments in mathematics and physics, in particular the new ideas concerning the relationship of geometry to the physical world and the concept of the *field* developed in relativity physics.

1.4.2(a). Poincaré's model. Henri Poincaré (1913) proposed a model for the development of space perception, within a broadly empiricist context, which has served as a point of departure for all subsequent empiricist approaches, including those applied to humans and infra-humans. Writing from the standpoint of a mathematician and philosopher of science, he represented an almost total return to the Berkeleyan view of space. Poincaré agreed with Kant that the notion of geometrical space was not part of the objects of sense themselves; he held it to be a matter of convenience which geometry one chose. However, the space of Kant's introspection was continuous, infinite, three-dimensional, homogeneous, and isotropic, while the space of Poincaré's introspection, which he labelled *representative* space, lacked all of these qualities. Discussing examples from visual, tactile, and motor space, Poincaré suggested that the space given in perceptual experience was much more restricted and egocentric than that suggested by the Kantian model. Thus, according to Poincaré

> 'absolute space is nonsense, and it is necessary for us to begin by referring space to a system of axes invariably bound to our body' (p. 257).

Many of Poincaré's ideas about space rested on notions of the active role of the motor system in the development of perception: an organism incapable of movement could not perceive space. Our minds constructed the space of Euclidean geometry as a convenient fit to experience 'by studying the laws by which . . . sensations succeed one another' (p. 71). These sensations were derived from either real or imagined movements, relative to things in the external world.

Poincaré distinguished between two types of external changes in things: those of *position* and those of *state*. While the former could be 'corrected' by some internal change, the latter could not.* These internal changes were the source of the sensations underlying the notion of space. Thus, to localize an object in space 'simply means that we represent to ourselves the movements that must take place to reach that object' (p. 70). Similarly, and with direct reference to the internal correction of an external change, the displacement of an object could be assessed by the organism through such movements as were necessary to bring the perception of that object back to its former state; that is, by reinstating the original view of the object. Directions of movement were not given, but

> 'the sensations which correspond to movements in the same direction are connected in my mind by a simple association of ideas' (p. 69).

Poincaré was aware that this association might be

> 'extremely complex, for the contraction of the same muscle may correspond, according to the position of the limbs, to very different movements of direction' (p. 69).

It was largely through the agency of the *displacement group* that Poincaré envisioned these associations as occurring. Derived from the mathematical theory of groups, a displacement group was defined as that set of internal changes capable of correcting for the same external change. Each of the movements (real or merely represented in thought) in such a group would be practically indistinguishable in its effects from all the others. The essential feature of the displacement group was its property of associating those movement patterns which were equivalent in their ability to reinstate a previous situation. By virtue of this association quite different spatial displacements would be connected in the mind, providing the basis for spatial concepts.**

It is essential to ask what Poincaré meant by 'correcting for an external change', as this ability, simply assumed in his model, is central to the displacement group and the construction of space. Poincaré suggested that displacements are considered as equivalent when they enable the observer to *regain the original view of an object*. Thus, correction involves reestablishing a subject-object relationship exactly equivalent to that which existed prior to the external change. If this is not possible through any displacement then the observer judges that the object has undergone a change of state. If it is possible, one can calculate the movement required

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* Poincaré used this concept to separate object motion from form change. But, as Johansson (1964) pointed out, this left him unable to account for the simultaneity of the two, e.g. the spatial qualities of a fire or a moving animal.

** This theoretical position was adopted by Hull (1934a) in his discussion of the development of spatial behaviour in rats, as we shall see shortly (pp. 50-2). Its central feature is that many paths from any place A to any other place B will be connected together because the organism has had considerable experience with the equivalence of these paths in its exploratory movements. According to both Poincaré and Hull, this connection among equivalent paths occurs automatically, while Piaget (see pp. 41-5) held that it had to be learned.

to achieve it and thereby assess the movement of the object. However, the ability to regain the original view seems to presuppose some primitive means of definitively identifying objects. Poincaré provides no explanation for this capacity, nor any hint that it is learned rather than innate. This being the case, the construction of displacement groups rests on *a prioristic* foundations, though the *a priori*s are not the ones Kant chose. For instance, when a movement (real or represented*) which is assumed to lead to the reinstatement of an original situation does not do so, we do not conclude that the displacement group, and the notion of space it embodies, have been incorrectly constructed. Thus

> 'If experience succeeds, we say that it teaches us about space; if it does not succeed, we hie to exterior objects which we accuse of having moved' (p. 271).

Success, of course, is measured in terms of re-identifying the original view of the object. What this means, basically, is that Poincaré cannot distinguish between objective and subjective motion, nor construct space, without recourse to something like innate ideas, an uncomfortable position for any empiricist.

When Poincaré moved to a discussion of the necessity of three dimensional space he further undercut his empiricist position. Thus

> '... when it is said that our sensations are "extended" only one thing can be meant, that is that they are always associated with the idea of certain muscular sensations, corresponding to the movements which enable us to reach the object which causes them ...' (p. 274)

and these associations

> '... are the fruit of a long personal experience and of the *still longer experience of the race*' (p. 274, our italics).

Poincaré admits that such associations as the latter would constitute *a priori* intuitions, but argues that the presence of these associations does not mean that it would be impossible for us to conceptualize space in other ways, only that it would be difficult to do so.

We must admit that we find Poincaré rather confusing by this stage. While calling himself an empiricist (and being so labelled by most others) he makes use of several *a priori* notions. While seemingly concerned with the problem of the necessity of certain forms of spatial perception he constantly argues for the conventionality not only of geometry but also of psychological concepts; we represent space in the way we do because it is convenient, not because it is right. None the less, Poincaré represents an important benchmark in empiricist theory. With the notable exception of Piaget (see pp. 41-5), subsequent writers were rarely concerned with the epistemological questions that had traditionally formed part of any theory of space. He provided not only the nucleus of all future empiricist approaches, but also the set of problems to which such solutions would be applied. His assumptions concerning the development of the concept of metrical three-dimensional space have had a particular impact. Starting with empirical data from the physical world of sensations and movements this concept was constructed through a series of transformations, beginning with the simplest, and least specified, of geometries and leading to the most rigid. This entire process was fuelled by the mathematical properties of the *group of displacements,* which were a necessary *a priori* system. According to Poincaré this sequence was demanded by the facts; there could be no metrical information directly available in the stimulating array because there was no intrinsic metric in the physical world. Sense data present only heterogeneous spaces; experience must construct the rigid, metric framework of connected space. These assumptions, and particularly the one concerning the sequence of acquisitions leading to metrized spatial constructs, have formed part of virtually every model of space perception in this century, be it empiricist or nativist.

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* By equating real and intended movements Poincaré was giving an implicit answer to the question of the nature of the information provided by motor processes in the elaboration of space; it comes not from the muscles themselves, but from some higher centre which programmes movements. We shall have more to say on this later.

1.4.2(b). Gestalt theory. As espoused by Hering, James, and others nativism was a flawed theory, as we have seen. Sensations were inherently spatial, but they were also fixed in some way to the structure of the receptors, or their termination sites in the central nervous system. This combination of sense elements and fixed connections fell apart in the face of the perceptual constancies and led empiricists to deny the validity of the entire nativist approach. However, Gestalt theory, by making a few basic alterations in the nativist position, managed to salvage a reasonable theory which could at least cope with some of the previously unacceptable facts. Gestalt theory is associated with several writers, in particular Wertheimer, Kohler, and Koffka; though they did not agree on everything, there was a basic consensus on most of the fundamental issues. Gestaltists could not accept the tendency to reduce psychological processes to discrete and localizable physiological mechanisms; this formulation had torpedoed earlier nativists. On the other hand, the Gestaltists did not wish to return to the dualistic notion that mind could never be accounted for in physical terms. Gestalt theory derived its basic concepts from the new ideas generated in physics. Relativity physics, and the notion of the *field,* offered a potential means of conceptualizing perceptual mechanisms that would be both physiological and non-atomistic.

The central nervous system is extended in space and can give rise to patterns of simultaneous activity which have a spatial quality. According to Kohler (1947):

> 'Experienced order in space is always structurally identical with a functional order in the distribution of underlying brain processes' (p. 39).

Such a distribution of neural processes was labelled a *physiological field.* The part activities comprising such a field had no phenomenal meaning in isolation. Rather, the entire pattern of neural activity served as an isomorph for some molar psychological entity such as a thought or a percept. Further, there was no exact localization of these fields within the brain; what was important was the pattern of the activity, not its location.

In order to understand how physiological fields mediated psychological processes, it is necessary to provide a few further details of the basic Gestalt model. In his monumental treatise summarizing the theory, Koffka (1935) described several different 'fields'. The *geographical field* was the external physical world, that which provides the stimulating environment for all organisms. The *behavioural field,* on the other hand, was the environment as the organism presumably perceived it, a world full of things with meanings, exerting forces, and capable of eliciting certain responses. The former was objective, value free, and the same for all organisms located in the same place. The latter was subjective and unique to each organism.

The idea that all behaviour could be accounted for in terms of the behavioural field was rejected, for this would deny the role of the structure of the organism itself. Rather, the proper level at which to explain behaviour was the physiological field, that system of isomorphs which both represents the behavioural field and takes into account the biological structure. Here the central concept of *organization* was introduced. The nervous system constrains and organizes the possible physiological fields according to its properties, while at the same time the simultaneous inputs interact with one another to produce an integral whole. A number of organizational rules were adduced to explain why a physiological field segmented as it did, and why forms took the shape that they did. To a large extent these rules have become part of the accepted lore of form perception.

However, the concentration upon the physiological field, and the interactions which organized its distribution, compelled Gestalt theory to concentrate entirely upon form perception. Organization led to the segregation of any field into discrete 'things', the nature of which were determined by such features as articulation, continuation, good shape, and so on. These 'things' did depend, for their constitution, upon the existence of a framework, but here the Gestalt model was not using framework in the same three-dimensional sense as did Kant. Rather, Gestalt theory utilized a framework provided by the notions of horizontal and vertical. Koffka's book devoted only a few pages to the entire problem of the organization of three-dimensional space and said nothing at all about the concept of unitary space. In fact, it is possible that Gestalt theory would deny the validity of psychological unitary space in its entirety, much as James did. According to Koffka 'behavioural space is not Euclidean' (Koffka 1935, p. 275).

This accords with the data provided by relativity physics, but Koffka did not go on to deal with whatever geometrical properties behavioural space does have.

These considerations, by themselves, should not have prevented Gestalt theory from developing a physiological mechanism for objective space. There are, however, several other reasons why this might have happened. First, Gestalt theory was, as we have seen, strongly influenced by the prevailing emphasis upon relativity and the concomitant notion of the conventionality of geometry. There seemed no need to account for any particular metric in perceived space. Instead, the emphasis was placed upon the anisotropy of both the geographical and behavioural environments. Second, the basic structural form of Gestalt theory might have itself precluded any successful analysis of unitary space. Having denied the concept of an *a priori* objective space, and juxtaposed this with the idea of physiological fields as integral wholes, there seems no way that Gestalt theory could generate unitary space from individual fields. To do so would be to contravene the basic whole-part principles embodied in the theory. Either unitary space was there or it was not, it could not be constructed. This, of course, is a corollary of the often noted inability of Gestalt theory to cope with learning.

The outcome of all this was seen most clearly in Lewin's (1936) book on *Topological psychology,* where he attempted to deal with behaviour in space according to Gestalt principles. Thus

> 'psychology does not deal with one single connected space which represents the totality of its world' (p. 68).

Instead, Lewin concentrated on the dynamic properties of *life space,* equivalent to what Koffka called the behavioural field, and what most writers now term *personal space.* This space is described most adequately, according to Lewin, in terms of topological geometry, which is a system for defining spaces and spatial relations without measurement. It deals with connectedness, whole-part relations, boundaries, and so on. By itself, topology could not incorporate notions of distance or direction; Lewin therefore added some components of vector geometry which enabled him to discuss directional forces within the life space.* This conceptualization does allow for a discussion of some of the dynamic forces constituting a behavioural field, and it is these motivational variables that Lewin concentrated upon.**

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* Lewin termed the space generated by this combination of topological space and vectorial geometry *hodological space*; this derives from the Greek word *hodos,* which means path. Thus, Lewin was interested in specific paths, or routes, through behavioural space.

** Lewin never actually formalized the mathematics he was to use. According to Kaplan: 'The actual application of topology to psychology remained for Lewin a program and a hope' (1956, p. 1307).

Lewin's insistence that psychological space is concerned with, and organized by, the meaning of things within the behavioural field has carried over directly to contemporary approaches to mental mapping, accounting for its bias in this direction (see pp. 75-80).

As useful as such an approach might be for exploring certain features of spatial behaviour, it is clear that its usefulness is limited to egocentric space. There seems to be no way that Gestalt theory can provide a basis for objective spatial representation. Added to these difficulties in coping with unitary space, we should briefly mention the problems associated with the physiological field and the idea of non-localization. The so-called 'law of mass action' assumed that the debilitating effects of brain damage depended more on the extent than on the locus of the damage; this was a necessary adjunct to the field theory. Though given some early support in Lashley's work (see e.g. Beach *et al.* 1960), it now seems clear that some form of discrete localization of function must be allowed (see Hebb 1949, pp. 12-16, for some discussion of this issue). The Gestalt programme, then, could not survive intact. Its emphasis on organizational properties was important, however, because it showed one way in which perception could be detached from the elements of sensation. Its total inability to handle metrical three-dimensional space meant that it could never provide a complete accounting for space perception.

1.4.2(c). Later views reviewed-briefly. Neither Poincaré nor the Gestaltists, in their reformulations of empiricism and nativism respectively, provided satisfactory theories, but they did lay the groundwork for further progress. Poincaré identified the starting point (the notion of the displacement group) from which successively more subtle concepts of the geometry of space could potentially be elaborated. His failure to show how this could be done opened the door for a more thoroughly empiricist approach, and that has been provided by Piaget and his co-workers.

The failure of the Gestalt programme was of a different order. In the course of its progress numerous experimental facts of perception were generated, and these constituted new problems for any future theory. The particular solutions offered by Gestalt theory could not work, but they did indicate that, within any nativist theory, inherently spatial sensations had to be extensively transformed before they could account for perception. This notion of transformations, combined with an entirely new idea concerning the source of spatial information from the environment, characterized J. J. Gibson's (1950, 1966) influential model.*

1.4.3. PRESENT VIEWS

We have seen that Poincaré's model failed to provide a resolutely empirical explanation for the generation of spatial concepts. Further, it appears that any nativist model which denies the *a priority* of metric unitary space must incorporate an empirical explanation for the development of this concept, beginning with the inherent space provided by the structure of the organism and the automatic transformations worked on the sense data. Both Poincaré and, as we shall see, Gibson, starting from rather different theoretical foundations, settled on the agency of the mathematical group as crucial to the elaboration of space perception. For Poincaré this group consisted in the sets of movements which effected the same displacement in space. Their automatic association was seemingly inherent. For Gibson the group consisted in the set of transformations worked on sensory inputs, such that invariants were extracted from these data. Again, these processes were inherent.

Beginning where Poincaré left off, Piaget and his colleagues have proposed an elaborate scheme which attempts a thoroughgoing empiricist analysis of the ontogeny of space perception (Piaget 1955, Piaget and Inhelder 1956, Piaget, Inhelder, and Szeminska 1960). Theirs is a theory dealing with the epistemology of space *representation*, rather than perception. It is neither empiricist nor nativist; rather, it is best described as *structuralist.* Yet Piaget insists that spatial representations are acquired from experience and presents a scheme for this development which would, if correct, fulfil the programme of generating spatial concepts without *a priori* knowledge of unitary space. As such, it represents a view of psychological space antithetical to the position we adopt here, and must be closely considered.

While Piaget has concentrated upon the development of human spatial representation, the empirical approach of Poincaré has also influenced experimental psychologists working with infra-humans. In particular, Hull adopted this approach in his attempts to account for maze learning in rats. By briefly reviewing his model we can introduce the general problem of the study of space perception in infra-humans, with which much of the remainder of this book is concerned. Here, we can consider the early work on the nature of the rat's spatial abilities, and the kinds of explanatory models it elicited.

1.4.3(a). Piaget. Piaget's commitment to an essentially empiricist position is clear:

> 'it would be a complete mistake to imagine that human beings have some innate or psychologically precocious knowledge of the spatial surround organised in a two- or three-dimensional reference frame . . . Far from constituting the starting point of spatial awareness, the frame of reference is in fact the culminating point of the entire psychological development of Euclidean space' (Piaget and Inhelder 1956, p. 416).

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* With Piaget and Gibson we shall bring our story up to the present. There have been other approaches, but these two are representative, embodying the empiricist or nativist presuppositions of all approaches. Thus, models such as transactionalism (e.g. Awes 1953, Ittelson 1960) or the sensory-tonic, organismic approach (cf. Wapner, Cirillo, and Baker 1971, for a review and some early references) place egocentric space before unitary space and are variations on the themes we have been exploring in this chapter. Though interesting in their own right, such approaches need not be considered separately in the context of our review.

On the other hand, Piaget is by no means the type of empiricist who considers organisms as merely passive receivers of information from the external world. Rather, Piaget holds that the experience which is essential to the development of intellectual concepts must itself be acted upon and organized by the subject. Thus, his is a constructivist model within which the structure of the organism interacts with the structure of the external world in a constant dialogue, the end-product being an increasingly refined version of the real world. Underlying the dialogue between organism and environment are two fundamental processes: assimilation and accommodation. The former refers to the way in which some specific action, in the course of reproducing itself, incorporates new external objects to its scheme. Thus, it is a form of generalization. The latter refers to the complementary process of modification of assimilation schemes in the course of incorporating a variety of new objects.

Piaget is concerned with the basic epistemological questions that we have already focused upon in our discussion of Kant. Acknowledging that there appears to be a certain predetermined aspect to the structures of intelligence, including those concerning space, he inquires into the seeming necessity of these structures. Either they are preformed in the sense described by Kant, or they are in some way constructed. Piaget opts for the latter position, and his theory is then largely concerned with showing how necessary mental structures might be constructed in the course of experience.*

Piaget starts by pointing out that mental structures cannot 'arise out of nothing' (1971a, p. 62). There must be

> '... certain givens from which the construction of logical structures takes off, but these "data" are not primordial in any absolute sense ... nor do they "contain" what is, in the course of construction, "derived" from and "based" on them' (1971a, p. 62).

That is, the very first assimilations and accommodations can only take place against the background of some prior structures, and these must be inherent in the organism. For Piaget, what is given by heredity is a set of sensori-motor co-ordinations, or movement patterns, such as sucking and grasping in the human infant. These form the starting point in the endless chain of assimilation and accommodation, leading eventually to mature views of reality.

It is important to remember that for Piaget these earliest inborn structures contain only the seeds of later structures, and nothing more, for it is this distinction which separates his model from one of totally pre-formed structures.

How, then, does Piaget envision the construction of spatial concepts, starting only with a limited set of sensori-motor schemes? According to him

> '... at birth there is no concept of space except the perception of light and the accommodation inherent in that perception. All the rest- perception of shapes, of sizes, distances, positions,etc.- is elaborated little by little at the same time as the objects themselves. Space, therefore, is not at all perceived as a container but rather as that which it contains, that is, objects themselves; and, if space becomes in a sense a container, it is to the extent that the relationships which constitute the objectification of bodies succeed in becoming intercoordinated until they form a coherent whole. The concept of space is understood only as a function of the construction of objects . . . only the degree of objectification that the child attributes to things informs us of the degree of externality he accords to space' (1955, p. 98-99).

Here, Piaget asserts his commitment to a relative theory of space, derived from the relations between objects. But, the development of spatial concepts from object relations does not happen automatically, as Leibniz might have asserted. Thus,

> 'The "intuition" of space is not a "reading" or apprehension of the properties of objects, but from the very beginning, an action performed on them ... Action is first manifest in the form of sensori-motor activity regulating perception ... Poincaré had some inkling of this when he envisaged movements as the source of basic spatial concepts' (Piaget and Inhelder 1956, p. 449).

The very first concepts concerning space, then, are generated through actions directed at objects. Central to Piaget's argument, as it was to Poincaré's, is the concept of the *group*, about which Piaget has the following to say:

> 'A group is a closed circle of operations that return to the point of departure through an operation of the group as a whole . . . Like Poincaré, we shall not hesitate to speak of groups to designate the child's behavior patterns to the extent that they can be reversed or corrected to bring them back to the initial point. The only objection to Poincaré's description is that he considered such groups as capable of being immediately extended in adequate perceptions or images, whereas in fact they remain in the practical state for a long time before giving rise to mental constructions' (Piaget 1955, p. 105-6).

As with Poincaré, the group of displacements confers upon the organism the ability to return to its starting point. But, whereas Poincaré assumed implicitly that this ability rested upon some innate mechanism guaranteeing object identification and explicitly that the reversibility it implied was intellectually apprehended by the organism, Piaget asserts that reversible

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* Piaget rejects the strict Kantian model for a number of related reasons. At one level he seems to feel that construction is a much more powerful tool than pre-formation, in the sense that the former opens up 'the possibility of individual acquisitions' (1971b, p. 226). Of course, such individual acquisitions are quite beside the point with regard to structures which must obtain a necessary form in the end. At another level he rightly points out that pre-formation merely shifts the explanatory load from the individual to the species. It still remains a problem to explain how pre-formed structures came into being. Finally, Piaget rejects Kantian *a priorism* by lumping it with Gestalt ideas on the innate basis of forms, a theory which, he points out, cannot assimilate external experience at all. However, there is no justification for tarring Kant with this brush, just as Piaget is wrong in assuming that the later nativists, such as Hering, were avowed Kantians.

groups must themselves be constructed during development and that, once developed, they remain solely practical for some time. In so doing, Piaget avoids several of the problems associated with Poincaré's model. By assuming the *a priority* of groups, in the absense of an *a priori* spatial intuition, Poincaré was forced to assume some primitive ability to identify objects. Piaget can deny the *a priority* of both groups and space, and therefore allow for the development of processes generating the object concept and object identification upon which spatial concepts can be based. The consequence of this position, of course, is that the development of any secure ideas about space must be preceded by the concept of the permanent object, as stressed by Piaget himself. This is something which can be tested experimentally; we shall see shortly that the evidence on this point remains equivocal.

In denying that the earliest spatial notions are anything more than practical Piaget draws a distinction between spatial *behaviour* and spatial *representation.* An organism might behave as though it were in possession of certain spatial principles without being aware of the concepts inherent in these principles. For instance, reversibility in action would not imply the intellectual mastering of the *idea* of reversibility. This is a notion with which we concur, and to which we can only add that organisms might well be in possession of practical Euclidean schemes and a unitary spatial framework without being consciously aware of them. Here again, experimental evidence can be brought to bear on the question of just what kind of practical spatial knowledge is inherent in the sensori-motor schemes given to the neonate upon maturation of its nervous system.

Piaget, of course, specifically denies that the various practical spaces, at the service of reversible groups, can be taken as an ensemble adding up to something like unitary space. Rather, these spaces are separate and

> '... there is no specifically geometric and kinematic representation that would make it possible to place them in a common environment' (1955. p. 113).

It is this common environment which must be generated through experience and which underlies any representations of multi-modal unitary space we subsequently possess.

To summarize: Piaget attributes to the organism certain forms of innate knowledge, embodied in action systems such as those controlling the sucking and grasping reflexes. Such knowledge is purely practical and not represented as yet in thought. None the less, it controls the organism's access to the external world. By 'watching' its own behaviour the young child derives certain fundamental principles, such as the reversibility of groups; these are built up through the matched processes of assimilation and accommodation. Using these as the foundation, other concepts, including that of the object and the spatial field, are established, and these in turn lead to the elaboration of more refined spatial concepts.

Piaget's model makes a variety of predictions about the course of the development of spatial representation. Two classes of predictions are of particular interest to us: (1) those concerned with the relationship between the development of the spatial field and the concept of object; (2) those concerned with the particular sequence of acquisitions of spatial notions, beginning with topological concepts and leading through projective ones to metrical Euclidean space.* Of course, in general, the statements Piaget makes about the time of onset of these various concepts are of interest in the evaluation of his theory. Evidence concerning the first category comes from studies of such things as the ontogeny of the object concept, the ontogeny of shape and size constancy, and the use of spatial frameworks. Evidence concerning the second category comes primarily from studies of the apprehension of the different geometrical features of objects. Here, Piaget's object-oriented bias has clearly restricted his investigations.

In all these cases Piaget's model, for its verification, requires evidence that concepts develop slowly and in a particular sequence. Rather than review the evidence immediately we shall drop our discussion of Piaget momentarily and move on to Gibson. As we shall see, his model ultimately makes some of the same predictions as does Piaget's, in particular concerning the primacy of topological notions of space. We shall return to the question of the ontogeny of spatial perception and representation shortly (pp. 52-5).

1.4.3(b). Gibson. J.J.Gibson (1950), in his landmark book *The perception of the visual world,* provided a new approach to the study of space perception. He attempted to reformulate the basic problems of space perception so as to furnish some explanation for the concept of unitary space, which he considered central to any understanding of space.

His fundamental commitment to a nativist, rather than empiricist, position is clear:

> 'there is always some variable in stimulation (however difficult it may be to discover and isolate) which corresponds to a property of the spatial world' (p. 8).

However, Gibson's brand of nativism is a far cry from that suggested by previous writers. First, he made it clear that the standard 'sensation yields perception' story was totally inadequate, noting again that while sensations are continuously varying, perceptions can remain remarkably constant. His rough equivalents to these time-(dis)honoured concepts involved the reintroduction of the distinction between the visual field and the visual world previously suggested by Mach. According to Gibson, the visual field 'is the

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* Piaget makes much of the fact that this sequence reverses the historical order of their discovery by mathematicians, implying that the most primitive (child-like) was the hardest to find. Science had to peel off more and more layers of culturally imposed onionskin before reaching the basic mathematical concepts underlying all the others.

experience on which the doctrine of visual sensations is based' (1950, p. 27), while the visual world 'is the familiar, ordinary scene of daily life' (1950, p. 26). Further

> 'The field is bounded; the world is unbounded. The field is unstable; the world is stable. The field is composed of adjacent areas, or figures; the world is composed of surfaces, edges, and depths, or solid objects and interspaces. The field is fluid in size and shape; the world is rigid in size and shape' (1963, p. 3).

Thus, Gibson distinguished between the visual field which formed the central focus for most previous nativists and which was clearly non-Euclidean, and the visual world of unitary, stable, metrical space. Further, he took the radical stance that 'perception' pertains to the visual world. There is no other way, he suggested, to account for the facts of perceptual invariance. Objects remain the same size and shape though they move relative to the observer (and the stimulation derived from them changes), while the phenomenal world retains its stability and unity throughout.

How, according to Gibson, does the observer extract a stable visual world from the unstable visual fields, or retinal images, gathered from the ambient environment? Gibson's solution focused on the transformations the retinal image undergoes from moment to moment. These transformations form continuous series* which leave certain features of the retinal image unchanged. That is, there are portions of the field which do not vary from one fixation of the eye to the next. These higher-order invariant features 'may be the mediators of a stable visual world' (1950, p. 154). On the other hand, those features which do vary would mediate motion perception. It is important to identify the features of the ambient environment which might provide the necessary invariance. Gibson based his model upon the recognition that contours and texture gradients, features of surfaces rather than objects *per se,* formed the appropriate distal sources for a continuous series of transformations. The visual world, then, was based not on a succession of discrete images of a set of objects, as in a moving-picture sequence, but rather on a 'continuous but changing image' (1950, p. 158). The contours and textures in that image, with the transformations which could hold certain features invariant, remained constant, while the overall image could incorporate change. This invariant framework then embodied the objective space to which Gibson applied the term visual world. It was stable, boundless, and rigid. But, what about the geometrical properties of this world?

The transformations worked upon the retinal image could not hold any putative Euclidean properties of the physical world invariant, for the image itself was non-Euclidean. According to Gibson, only the topological properties of the image retain invariance, and it is this invariance which must form the basis for whatever objective world we perceive and its dimensions. Thus, while Gibson seems to have provided a workable model for the integration of a coherent space, he accomplished this at the price of starting with a non-metrical, or topological, space. His model, if it is to be complete, demands some mechanism for generating from this continuous topological world a metrically specifiable world.

Gibson rejects the traditional (empiricist) distinction between exteroceptive and proprioceptive sources of information. This distinction, as many have noted (cf. Mace and Pittenger 1975), is raised in the service of models which argue that some separate access to motor plans is needed by the organism if it is to distinguish between objective and subjective movement. How else to tell, these models imply, whether the observer or the world has moved? But Gibson argues that this is rarely required because there is an invariant framework against which motion can be assessed. If the observer moves, the entire field should move in register. The fact that, under this condition, in a motionless world the environment appears stable indicates that there must be a series of adjustments accounting for the effects of eye, head, and/or body movements. On the other hand, the objective movement of anything in the visual field would be detected by its independent change (variance) relative to the remainder of the field. It is easy to derive from all of these factors the basis for various perceptual constancies.

In two recent articles Gyr (1972, 1975) has questioned the adequacy of this formulation. His objection is that Gibson has not successfully integrated exteroceptive and proprioceptive information, which would provide a surer basis for distinguishing objective from subjective movement as well as account for the fact that organisms do use motor feedback (or reafferent) information. While Gibson agrees that there are no passive observers, in that eye, head, and body movements are essential to perception, he does not accept that proprioceptive inputs are necessary to veridical perception.*

Keeping the two sources of input separate possibly enables the Gibsons to account for a stable continuous world where the source of motion can be identified. But this world is not metricized, and no suggestions are made as to how it could be done through development, though it is clearly a requirement of the model. One way out of this dilemma is to concede that

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* This series of transformations, of course has all the properties of a mathematical group. Thus, the concept so important to the empiricism of Poincaré and to the Piagetian model takes up a central role in Gibson's version of nativism.

* Among the reasons for keeping exteroception and proprioception separate might be the problem raised by the need to co-ordinate the transformation of the two types of input such that they specify the same geometrical information. This problem becomes acute when one turns to the development of metric space during the growth of an organism; proprioceptive feed-back could vary with body growth, and these variations would have to be continuously re-mapped onto the visual transformations all the while that topological space is slowly giving rise to metrical space. A daunting problem. E. Gibson (1969) has alluded to this difficulty when she raised the possibility that the dimensions of objective space might vary with age.

movement processes automatically transform and metricize the visual fields, yielding the stable visual world. As this would be tantamount to accepting Kant's *a priori* intuition Gibson cannot make this step.

Little formalization of the mathematics involved in the transformations required by the model has been provided. In particular, one would like to see a formal treatment of the transition from topological to metrical space, as this would bear on Piaget's model as well. While this particular aspect of the model has been largely ignored, several investigators have explored Gibson's claim that visual input alone can specify the visual world.

In one of the first of these investigations Johansson (1964) attempted to resolve the problem of size-distance invariance. It is clear that, taken in isolation, the retinal image is ambiguous with regard to size and distance: two objects at different distances could produce the same retinal image. Yet our perceptions somehow are veridical with respect to these features, as they must be if motion is to be perceived. Johansson showed that

> 'Perception of motion in depth from a proximal pattern changing in two dimensions has ... proved to be rather inevitable, and thus a spontaneous consequence of stimulation in most cases' (p. 202).

He concluded that a change in two dimensions of the proximal (retinal) image automatically specifies three-dimensionality, and that this is basic to the accurate perception of size and distance. The model he proposed for this, involving vector subtraction, has been superseded by later work showing it to be insufficient. Eriksson (1973) pointed out that, while certain features of three-dimensionality might be obtained from changes in the optic array, these are not sufficient to disentangle the size-distance problem. His analysis applied both to the case of the stationary observer and the moving observer, and took into account changes in the texture gradients emphasized by Gibson.

In a subsequent paper Eriksson (1974) attempted to disentangle the contributions to perception provided by optical and movement information. He suggested that

> 'the visual system is able to provide the organism with distance information which is relative, and slant information which is absolute' (p. 233). *

However, information about absolute distances cannot be obtained solely from the visual input. Here, some contribution concerning the movements of the body, in terms of amplitude, direction, and acceleration, is required. Such information would allow the organism to calculate absolute distances; only then could the size-distance problems be resolved. Eriksson concluded that

> 'a living organism in a normal, redundant environment obtains veridical space information from the interaction of two systems: (a) the optical system generating object constancy and relative distance information, (b) the body-state system generating veridical distance and size information on the basis of movement parallax' (pp. 234-5).

While Eriksson seems to imply that these mechanisms are innate, he allows that experience might generate other means of accomplishing the same function as movement parallax. However, he rightly points out that experience could not lead the organism to these alternative sources of information without the prior action of a system capable of performing the necessary processes. Thus, he denies that veridical perception can be acquired entirely through experience, even with innately meaningful sensations. That would be putting the chicken before the egg.

From the point of view of Gibson's model, these considerations suggest that Gyr's criticisms are well founded. Without some necessary contribution from both visual and motor processes veridical perception would seem to be impossible. Such a fixed linkage between the two would take one a long way down the path to a pre-existent framework, and it remains only to determine the exact nature and metrical properties of this framework. Eriksson implies that it must be Euclidean, but offers no proof for this. We shall not comment on this problem here, but shall formulate some reasons for postulating a Euclidean framework later. Thus, at the very least, Gibson's model must be modified to account for a necessary interaction between optical and motor information. This does not, of course, invalidate many of the important points Gibson has made; his recognition of the role of texture gradients is particularly important. It enables one to separate out those features of the environment specifying objects from those specifying the ground. From what we know of the processes involved in figure-ground separation this must surely be innate and amongst the first of the transformations worked on the optical data. In conjunction with recent speculation about 'two visual systems' (cf. Trevarthen 1968, E. Gibson 1970) it offers a possible explanation for shape constancy that is separate from that accounting for the veridicality of unitary space. Thus, the specification of the form and features of objects might take place independently of, and parallel to, the specification of that object's movements and location in absolute space. Of course, though shape might be treated in this way, size constancy would require the latter information. Only after data concerned with textures and contours had been integrated with information about movements could the veridical perception of absolute space, and its contents, be achieved.

1.4.3(c). Space perception in animals. Before turning to a brief discussion of the development of spatial concepts we should examine the ways in which the problem has been treated in the animal literature. To some extent this will involve a return to models we have already discarded, but it is a necessary one; much of the evidence pertinent to our theory comes from

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* Eriksson points out that this means that visual input alone can account for shape constancy.

work with rats and we need to demonstrate that the principles deduced from work with humans apply to this species as well.

Hull (1943) utilized the empirical model established by Poincaré in his attempts to provide an explanation for spatial behaviour in rats. The need to say something about such behaviour arose out of the study of maze learning. Early investigations into the nature of maze learning were motivated primarily by an interest in the way in which such habits were acquired and maintained: which sensory inputs were essential, which not.* Notwithstanding this emphasis, these studies provided important insights into the basis of the rat's spatial abilities. The initial experiments (e.g. Small 1901, Watson 1907) were misinterpreted to suggest that maze habits were learned as a chain of proprioceptively guided responses; that is, as a stimulus-response (S-R) routine leading automatically to the goalbox. However, subsequent research demonstrated that complex mazes could not be learned on the basis of proprioception alone (e.g. Dashiell and Helms 1925, Dennis 1931, Honzik 1936), though simple repetitive sequences such as LRLRLR might be (Hunter 1940). Most workers rapidly agreed that vision was particularly important, expecially in those mazes which were open to a heterogeneous environment (Honzik 1936). Extra-maze cues were shown to be important by rotating the maze relative to the environment; such rotation invariably disrupted the maze habit (e.g. Watson 1907). However, there was no specification of how these extramaze cues exerted their powerful influence.

All this work was done on *spatial* mazes, that is, mazes in which different responses were required in different parts of space, as well as in different segments of time. One of the attempts to test the ability of rats to learn mazes proprioceptively involved the use of the *temporal* maze; here, the rat runs around the same parts of space and must distribute his differential responses solely in the temporal domain. Thus, after turning left on the first circuit through the maze the rat must turn right at the same point on a subsequent circuit. Hunter (1920) found that rats were incapable of learning a RRLLRRLL sequence in the temporal maze and concluded that:

> 'Work on the temporal maze indicates that it is all but impossible to set up a mere temporal sequence of kinesthetic processes with the rat. Running the spatial maze therefore must require cues which have space location as well as temporal position. In other words, the rat must recognize in terms of space where he is in the maze' (p. 16).

Results such as these suggested to most investigators that the rat had some sort of place orientation or directional set. Thus, Dashiell (1930), on the basis of work done in a maze containing many paths to the same goal, suggested that:

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* Munn (1950) reviews these studies in detail. Here, we concentrate on the contribution this work has made to an understanding of space perception (see also pp. 286-90).

> 'learning consists in the establishing not of a definite *pattern of specific* turns, but of some more *general orientation* function. This general function enables it to pursue new pathways from time to time, the while remaining successfully oriented in the direction of the objective' (p. 19).

The notion that rats develop a direction orientation referring to the location of the goal is central to any analysis of maze learning; different psychologists approached this phenomenon in vastly different ways, adding to a fundamental split in psychological theory which remains today. Tolman (1932, 1948) proposed that rats form *cognitive maps* of environments:

> 'we believe that in the course of learning something like a field map of the environment gets established in the rat's brain ... and it is this tentative map, indicating routes and paths and environmental relationships, which finally determines what responses, if any, the animal will finally release' (1948, p. 192).

These cognitive maps were essential to the ability to respond flexibly in the maze situation, the animal emitting whatever responses were necessary to get to the goal. Implicit in Tolman's model is the assumption that the animal does not learn about space through the association of movements, but rather that the animal's spatial abilities rest on the construction of maps which represent the spatial relationships between the various things experienced in the environment.*

Faced with the same evidence, and well on the way to developing his general theory, Hull (1934a,b) proposed the concept of the *habit-family hierarchy.* He stated that:

> 'a family of habits may be defined in general as a group of two or more habit sequences, all of which may be initiated by a particular stimulus and terminated by a particular reaction' (1934a p. 39).

Thus, alternative solutions to the same maze problem, accounted for in terms of cognitive maps by Tolman, were dealt with in a strict S-R fashion by Hull. The rat learns not only about those routes which he actually traversed, but also those which, though different, start from the same point and end up at the same goal. According to Hull, this is accomplished because:

> 'hierarchies based primarily on locomotion are set up early in life, presumably as the result of locomotion in free space, with the result that the animal at the beginning of a maze experiment is already in possession of a vast repertoire of equivalent but fairly distinct locomotor habits, any one of which, in free space, would mediate a transition of his body from the starting point to the goal' (1934a, p. 41).

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* It is unlikely that Tolman ever felt that such maps might be more than good metaphors, able to account for certain interesting forms of behaviour, but surely not existing within the brain, as we propose here.

The similarity of this explanation to that proposed by Poincaré is clear; organisms learn about space through the association of movements which, though different, all take the animal from one place to another. Hull's habit-family hierarchy is yet another application of the idea of mathematical groups and carries with it the same problems associated with Poincaré's model.

The weight of evidence concerning maze learning favoured Tolman's view that animals learn something about places in the environment. Woodworth (1938), writing during this period, summarized the situation as follows:

> 'Since neither chain reflex nor motor pattern accounts for the rat's behaviour in the maze, we ask once more what it is that the animal learns. The most obvious answer, which has been given repeatedly by investigators in describing the rat's concrete behavior, though avoided in their theories, is simply that the rat learns the place' (p. 135).

Thus, the investigation of maze behaviour in rats suggested that spatial behaviour could not be explained through the invocation of simple S-R habits. Something like a cognitive map, providing an overall framework and specifying places in the environment, is needed. However, Tolman said very little about the properties of such maps, or about the way in which they might have developed. Do rats learn to build maps? Or, is this ability given prior to experience?

1.4.4. THE ONTOGENY OF NOTIONS CONCERNING SPACE

We have seen, in work with both humans and rats, a clear need to discuss space perception in terms of a unitary framework stabilizing a three-dimensional world. We have argued that this framework is part of the innate machinery of the organism, and that it could not be developed entirely through experience, nor from experience with inputs specifying only relative spaces. This implies that the framework must be present at birth, or as soon as the necessary neural structures achieve mature function. While there has been considerable work on the ontogeny of space perception, virtually all of it has proceeded within the context of relative theories of space perception. Consequently, there is little evidence which speaks directly to the question of the ontogeny of the notion of absolute space. The nature-nurture issue, as applied to this field, has been concerned with the role of experience in the development of capacities such as localization which are functions of egocentric, rather than non-egocentric, space. Similarly, most of the work with children has concentrated upon such things as the development of left-right or up-down concepts, the use of arbitrary reference systems (e.g. the North-South axis), and the coordination of perspectives. None of this work can provide a clear answer to the question of whether or not an innate spatial framework specifying metric three-dimensional space exists. While it has traditionally been assumed that this latter capacity only arises after the acquisition of a variety of relative spatial notions, there is, in fact, no evidence to compel this view.

Work with infra-humans (cf. Zuckerman and Rock 1957, Pastore 1960, Epstein 1964, Fantz 1965, for reviews) has generally confirmed that localization and depth perception are innately determined, though learning can provide alternative means for achieving these functions. Thus, Fantz concluded that 'perception is innate in the neonate but largely learned in the adult' (p. 400). He assumed that such learning involves finding out which dimensions of the environment to attend to, and that this principle holds for all species. Such learning of selective attention processes can only occur, as Eriksson (1974, see pp. 48-9) pointed out, if there is an innate mechanism specifying the appropriate information base in the first instance. Unfortunately, there is no body of evidence directed at the problem of 'place-orientation' abilities or other capacities dependent upon cognitive maps.

Curiously enough, though the methodological problems of working with humans are usually greater than those encountered with infra-humans, more progress has been made in determining the complex spatial abilities of children. Whereas James (1890) assumed that the infant confronted a world of 'buzzing confusion', work within the past decade has demonstrated that there is considerable structure to the infant's world.

The prime proponent of the view that unitary space only develops after long experience is, as we have seen, Piaget. He assumes that notions of space are dependent upon those of the object, and that something like a unitary space develops only after a series of acquisitions concerning heterogeneous relative spaces. Further, metric space is assumed to develop only after the mastery of topological space, perhaps as late as 11 years after birth in humans. It is important to stress Piaget's distinction between perception and representation; the former refers to practical knowledge, demonstrable only in actions; the latter refers to cognitive structures. Piaget is interested primarily in the latter, and thus concentrates upon the verbalizations of his (necessarily) older subjects. From our point of view it is unnecessary to insist upon innate representations in the sense defined by Piaget. It matters not whether the infant is aware of its capacities; their presence will organize its behaviour and, more important, its potential for representations. It is clear that there is no particular correlation between the ability to talk about space and the capacity to behave appropriately in it (e.g. Asso and Wyke 1970); thus any approach which relies upon verbalizations is bound to be seriously misleading.

In contrast to Piaget's views, most other contemporary workers have concluded that infants rapidly come to perceive a world of permanent objects embedded in three-dimensional continuous space (cf. Bower 1971, 1975, Hershenson 1967, Bond 1972, Harris, Cassel, and Bamborough 1974, Yonas and Pick 1975). Bower, for instance, suggests that young infants (up to 16 weeks)

> 'ignore features to such an extent that I would suggest they respond not to objects but to movements. Similarly, I would suggest that they respond not to stationary objects but to places' (1971, p. 37).

Thus, Bower is asserting that places precede objects, in direct opposition to Piaget's view.

In what is primarily a theoretical and methodological review, Yonas and Pick (1975) come to the conclusion that infants possess something like a three-dimensional mapping system providing them with

> 'a knowledge of spatial layout in three dimensions which is something like an adult's knowledge of spatial layout of his nearby environment' (p. 24).

Their review of the available data from studies of space perception in infants supports this conclusion. However, as they point out, the crucial experiments, involving such things as detour behaviour, have not yet been done. We agree with their suggestion that experiments designed to distinguish between 'place' and 'response' learning, similar to those done in rats (see pp. 71-4), should provide conclusive evidence on this question.*

While permitting the conclusion that an innate framework exists, most investigators do not take the additional step of ascribing to this framework any particular metric. It will be recalled that both Piaget and Gibson hold that spatial knowedge advances from topological (non-metrical) to Euclidean notions.

Piaget's strongest evidence consists of those studies showing that the topological features of objects are responded to before metrical ones (cf. Piaget and Inhelder 1956). However, most attempts at systematic confirmation of this sequence have failed (e.g. Laurendeau and Pinard 1970, Lovell 1959, Fisher 1965, Cousins and Abravanel 1971, Dodwell 1963). Reviewing these data, Dodwell concluded that there is 'no clear-cut progression from one type of thinking about space to another' (p.161). Thus, the topology-to-Euclidean development demanded by Piaget's theory seems not to occur, at least as far as the perception of the spatial properties

of objects is concerned. When we move to space, as distinct from objects, we run out of data. Aside from evidence concerning depth perception and localization, little information is available. Several recent studies have shown that quite young children (4 years old) can read and interpret maps accurately without prior exposure or training (Muir and Blaut 1969-70, Blaut, McCleary, and Blaut 1970, see p. 79). These data are inexplicable within Piaget's scheme, but they do not prove the innateness of a Euclidean metric.

Our brief examination of the developmental literature is consistent with the conclusion that the ability to organize perceptions in a rigid, three dimensional space is available quite early, probably prior to any specific learning experiences. This is not to say that infants are 'aware' of it, as we have pointed out above. The representation in conscious thought of such notions of space might well follow the time course suggested by Piaget, or other similar models. In that case, the observed sequence of acquisitions would reflect the ease with which the child can symbolize its own perceptions and behaviour, not the order in which these become available as organizing frameworks for experience.

1.4.5. CONCLUSIONS

A unitary spatial framework, considered essential to the interpretation of a wide variety of abilities, seems to be present innately and available to organisms of numerous species as soon as they can move. Neither a strictly empiricist approach, nor a nativist one denying the *a priority* of such a unitary space, is consistent with both data and logic. This conclusion has several implications: (1) it ought to be possible to discover the neural correlates of unitary space perception, in as much as this 'concept' is not built up from others but rather is built into the brain; (2) the philosophical position suggesting that the psychological notion of absolute space must be derived from notions of relative space, or from experience with a world of relative space, is wrong. The adoption of this position within mainstream psychology seriously hindered the advance of understanding concerning space perception. It focused the attention of most investigators upon objects and subject-object relations (cf. Ittelson 1973). This, in turn, totally emphasized egocentric space at the expense of objective, non-egocentric space, a position which is only recently being reversed in the surge of interest in environmental perception. It is clear that whatever the status of relative space in physics and mathematics, it cannot be given ontological priority within psychology.

We shall conclude this chapter with a return to the epistemological issues with which we began. We ask the reader's forbearance in this, but we simply cannot leave the questions raised earlier unanswered. There are two related issues worth discussing here: the status of extension, and the means by which things are identified and re-identified. In the course of this dis-

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* Three recent studies provide some data on this question. Bremner and Bryant (1976) tested infants on place v. response performance and found that behaviour in 9-month-olds was primarily based on responses. Acredolo (1977), on the other hand, demonstrated that place behaviour was as frequent as response behaviour in 3-year-olds, and dominated in the 4- and 5-year-olds, becoming progressively more independent of particular landmarks. Finally, Fishbein (1976) has placed into opposition the possibilities that children use a spatial framework emphasizing the relations between objects or one which emphasizes the potential locations which objects might occupy. This study provides a direct assessment of the use of 'relative' or 'absolute' frameworks. Fishbein found that all of his children, aged between 6 and 11, were using an absolute framework, and that there was no change in this ability over the time period studied. These data contradict Piaget's assumption that the absolute framework cannot develop until about 10 years of age. See pp. 9l-2 for some further discussion of this problem.

cussion we think that the inevitable failure of relative theories in psychology will be understood not as a function of any lack of ingenuity on the part of their adherents, but more as a function of the basic flaws in the notion of relative space.

1.4.5(a) The concept of extension. One of the implications of any model denying an *a priori* concept of unitary space is that the notion of extension must precede that of space, which can only be constructed out of the relations between extended objects. For nativists extension derived inherently from sensations, and it was assumed that the extended fields so perceived could be integrated into a three-dimensional world. For empiricists the concept of extension itself had to be constructed from experience, to be followed by that of space.

The assumption that extension precedes space raises several serious problems. Russell (1901) noted that the relativist falls into a vicious circle because he can define space only in terms of objects assumed to exist in space. In a related manner, Hooker (1971) points out that, according to the relative theory,

> 'Material objects are spatially extended. Space cannot therefore consist solely in external spatial relations among material objects, for then material objects could not be regarded as spatially extended-their spatial exteriors could not be constructed' (p. 99).

Hooker concludes that this problem can be circumvented only by positing the existence of basic material objects which have no parts, that is, things which are not extended. Leibniz clearly foresaw this problem when he postulated the existence of monads.

In order to construct a space from a plenitude of partless 'things' it is essential that each of these 'things' be qualitatively unique. Identical objects can only be distinguished by their spatio-temporal position; it is not possible to construct space from them. Thus, one is caught in the dilemma of requiring certain relata to constitute space, but then finding that these relata can only be distinguished in certain cases if space already exists. Leibniz tried to avoid this trap by specifying that every monad was qualitatively unique; thus, space in the monadic realm could be easily constructed. This uniqueness required the assumption that intermonadic 'perceptions' be asymmetrical; the distance between two monads, or points, differed depending on where one stood. This non-commutativity of distance means that the space of one monad could not be transposed onto the space of another.*

This curious notion of asymmetrical space was essential to Leibniz's

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* This model serves as a useful approach to the personal spaces described by Lewin and others, where distances are often non-commutative, as we shall see. However, this property appears to be a function of experience and the learned meanings of places, rather than of the organising system itself (see pp. 37-40).

theory, for without it certain identification of monads would be impossible, an issue discussed at some length by Strawson (1959). The extent to which Leibniz went to solve the problem of identification attests to the fundamental nature of the issue. We have seen, for instance, that the ability to identify objects was taken as an *a priori* by Poincaré, in one of the major failings of his model.* This failure, we would submit, is not accidental, for the issue of identification lies at the root of all theories working with relative space.

1.4.5(b). The problem of identification. The theory proposed by Poincaré attempted to specify the means by which localization and a reference framework can be established solely through experience, and can be used as a starting point in our discussion. Poincaré provides some details of the required mechanisms when he discusses the way in which an organism localizes an object in space. Let us follow the gist of his argument.

The problem is simply stated: one is at A and the object to be localized is at B. According to Poincaré, one localizes the object by representing to oneself the movements required to reach it. It is clear, as Poincaré recognized, that a variety of movements will take one from A to B and, conversely, that the same set of movements, under different environmental conditions, might lead to different places. It is thus necessary to build up, by successive sets of movements, an 'association of ideas'. This was the basis for the group of displacements. However, one should only associate those movements which one took from A to B; it is crucial to start each set of movements from exactly the same place. But, how is this place to be identified? This difficulty can be overcome by postulating two objects, one at A and the other at B, and allowing the organism to shuttle back and forth between the two. However, this only works when one is certain that neither object has moved in the interim. Poincaré's solution to the problem of determining the immobility of the reference objects has been noted already; it involves identifying objects. But, what if there are identical objects in the world, differing only in terms of spatial location? This brings us right back to the necessity for a world of qualitatively distinguishable objects, so we can return to Leibniz for a further look at how his model fares.

Leibniz specified that his monads were unique, and within this notion it would seem as though space could be articulated. However, the spaces of

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* The reader should not assume that the problem of identification is limited solely to those versions of relative theory we have chosen to discuss. On the contrary, it is central to any relative theory. For example, Bohm (1965), in a book on relativity theory, falls into the same dilemma when he attempts to show how organisms can learn about relative space. Even before the young child has secure concepts of space Bohm assures us that it 'can go from one place A to another B by many different paths, and that all these paths lead him to the same place (or alternatively that if he goes from A to B by any one path, he can "undo" this and return to A by a large number of alternative paths)'. Thus, the child can apparently re-identify places before it has a fixed notion of space. Bohm does not specify how this is managed.

each monad could not be superimposed. Without the panoramic 'vision' of the monad how could a continuous stable world be articulated? The asymmetry built into the monadic realm means that distance relations cannot be constructed. Cox (1975) has suggested that an ordered space within the monadic realm could be achieved if one drops asymmetric perceptions, but here she falls prey to Strawson's argument that this would make re-identification theoretically impossible. Her added assumption that two monads with the same set of perceptions could never exist co-temporally in the infinite complexity of our universe is special pleading.

Leibniz's difficulties increase when he moves to the phenomenal world of objects and extension. In order to construct objects Leibniz clumps similar monads together as aggregates on the assumption they can be viewed as identical by distant observers. These objects in turn form the relata for psychological space. But, the monads forming them no longer incorporate the essential property of uniqueness. In negotiating the gap between monad and object Leibniz has been forced to abandon the very feature of monads which enabled them to be perceived as occupying different parts of space. *Leibniz' objects can not be extended in space,* and therefore at the phenomenal level his system falls victim to the Russell-Hooker criticisms set out above.

We must return from Leibniz to the problem of identifying objects, then, without a solution. There would seem to be a limited number of alternatives available. One might suggest that it is not necessary that all objects be distinguishable, merely that there be some set of objects which contains only unique members, assumed not to move relative to one another. This 'reference set' could be specified by innately available information or it could be learned through experience. The former possibility leads ultimately to the doctrine of innate ideas. The latter demands some mechanism for determining when objects move. But here the argument doubles back on itself, for only by identifying objects in the first place can one distinguish between objective and subjective movements. It is hard to avoid the conclusion that experience alone cannot specify the objects to be chosen as a reference framework. Either one accepts the notion that there are innate ideas, or innate dispositions to choose certain objects, or one is left without a means of object identification.

Gibson's model comes closest to satisfying the second of these procedures. The invariance inherent in textures during movements could, as he suggests, form the basis for the disambiguation of objective and subjective movement. As we have argued, additional information about the organism's movement seems crucial to the objectification of perception; absolute distances cannot be obtained without it. Once we accept the idea that there is a fixed input from movement processes which objectifies the three-dimensional space articulated by sense data, we have adopted Kant's solution. Strawson (1959), in his discussion of the problem of identification, concludes that it rests 'ultimately on location in a unitary spatio-temporal framework' (p. 39). We agree with his conclusion: there must be a pre-existent spatial framework in order for organisms to experience the world coherently. Objects could not be identified, nor localized, nor even seen as extended in the absence of this framework.

1.4.5(c). Final Views. We have organized this chapter around the dichotomy between absolute and relative space in the hope of showing how the choice of either of these as prior crucially influences the ability of any theory to cope with space perception. Most contemporary models start with relative space and attempt to build first a framework and then metrical space. These models rarely get off the ground, stumbling over the problems of re-identification and movement.

It seems reasonable to conclude, first, that there is a clear need for the concept of unitary space. Further, it appears that this framework cannot be acquired through experience; it must be available soon after birth, for the processes of localization, identification and the coherent organization of experience depend on it. Finally, the weakness of Gibson's model indicates that not only must the framework be built in, but that its metric must be specified as well. To reinforce this conclusion we can consider briefly the implications of assuming that the metric of objective space is acquired through experience, a position adopted even by those who would accept the idea that the framework is itself innate (e.g. Haber and Hershenson 1973). This particular argument has been voiced most emphatically in the course of cross-cultural investigations concerned with the theory of cultural relativity. Whorf (1956) stated this position clearly:

> 'Newtonian space, time and matter are no intuitions. They are recepts from culture and language' (p. 153).

More recently, Bertalanffy (1971) has expanded upon this position. Briefly, he argues as follows: (1) because of the existence of non-Euclidean geometries and relativity physics it is impossible to accept Kant's *a priori* intuitions; (2) the space of our perceptions, particularly visual space, is also non-Euclidean (see, for instance, Blank 1959); (3) different cultures have different basic ideas about space; thus (4) culture must be responsible for the fact that we use Euclidean and Newtonian concepts.

Bertalanffy also discusses the concept of the *ambient,* that slice of reality to which each species is attuned by virtue of its sensory apparatus. He admits that primates live in an ambient which is accurately mapped by Euclidean and Newtonian concepts. That is, we neither perceive, nor act relative to, the spaces of astrophysics or quantum mechanics. But, Bertalanffy does not concede that, if our concepts are to be Euclidean and Newtonian, a problem arises in acquiring them from experience with non-Euclidean visual space. If we dismiss the notion that different cultures conceptualize space in different ways,[*] we are left with explaining how Euclidean space is abstracted from a non-Euclidean world in a uniform fashion by all members of the species, and we have already seen the difficulties inherent in this.

The ubiquity and adaptiveness of Euclidean and Newtonian concepts in the ambient in which we live indicates that there must be mechanisms guaranteeing that these concepts are used by everyone, even without their being aware of it. Given that practical experience might not, by itself, lead us to them, we must assume that they are inherent in our structure. We *must* perceive the world in these terms, though we *can* conceptualize it in others. Paradoxically, the demonstration that physical space is non-Euclidean, rather than invalidating Kant's theory, seems to necessitate it. Grunbaum (1964), approaching this problem from a rather different angle, arrives at the same conclusion, stated in the form of a series of questions, worth paraphrasing here:

(1) How do we arrive at an appropriate Euclidean metric through the use of a visual system working with non-Euclidean data?
(2) If we have been seeing non-Euclidean space all along, why has it taken 2000 years to conceive of these geometries?
(3) Why was it necessary for Helmholtz and Poincaré totally to retrain their intuition in order to pictorialize non-Euclidean space? [**]
(4) Why is Euclidean geometry easier to learn than non-Euclidean geometry (if it is)?

Our attempt to answer the problems posed by Grunbaum's questions provides the basis for this book. While we readily accept the existence of the types of relative space described by Poincaré and others, we think there is substantial evidence for the independent existence of another spatial system, one which generates a Kantian unitary space.

We shall postulate that, in most of the brain, space is represented in the relative manner; that is, referenced to the organism and built up through experience. We shall refer to these egocentric spatial systems as *taxon* systems, and this type of space will be called *taxon space.* Included within this category are all the sensory and motor systems. In Kantian, or *locale,* space, representations are located within a Euclidean system, yielding a space which does not depend for its existence on particular objects but which serves as a framework for relating these objects to each other independent of the observer. The location of an object in this space is, as Poincaré suggested, a function (although only indirectly) of the organism's

movements in space relative to that object. The details of the mechanism accomplishing this will be discussed later (see Chapter 4). Locale space is non-egocentric. We agree with Kant that the relations embodied in this space are the content of what is usually called knowledge, the knowing *that* as opposed to the knowing *how*, to use Ryle's (1949) terms.

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[*] The evidence is very spotty on this question, and is completely confounded with language usage. There are differences in certain perceptual functions across cultures, but no good evidence that the basic intuitions of space differ. As we shall see, there is strong evidence that members of so-called primitive cultures are particularly good at mapping and navigational abilities, both of which would seem to presuppose a Euclidean framework. Again, the difference between perception and (linguistic) representation must be stressed.

[**] If in fact they actually did so, which we doubt.

2

Spatial behaviour

IN the first chapter we concluded that the ability to represent objectively the world in three-dimensional space, that is, to build cognitive maps, must be innate. In this chapter we shall concentrate on specifying those behaviours dependent on cognitive maps. It is quite a recent development that one could argue whether or not cognitive maps were innate; for much of this century the concept in its entirety was rejected by mainstream psychology. Mental maps, mentioned at the start of the century by Gulliver (1908), were banished, along with introspective data, during the dark night of the ascendancy of behaviourist theory in America.

Notwithstanding this ostracism, we can call upon a varied literature in specifying behaviours that would seem to require cognitive mapping. Much of this has been generated in the past 10 years in the course of a dramatic upsurge of interest in *environmental psychology,* which concerns itself with the perception and utilization of large-scale, or macro-, environments (e.g. Wohlwill 1970). Before turning to this new evidence, however, we can briefly look at two other areas of research which have contributed to the current popularity of the idea of cognitive mapping: (1) studies of navigation in various species, but mostly birds; (2) the work of Tolman and others on place learning in simple and complex mazes.

Though these data provide a justification for thinking about cognitive maps in our theories of behaviour, they fail to provide an appropriate formalization of the concept, and certainly not one that would be useful for translation into neural terms (see Olds (1954) for an early attempt at such a translation). Consequently, we devote the second section of this chapter to a more formal treatment of maps. Here, we contrast *maps* to *routes,* as we shall suggest that spatial behaviours based on the taxon systems can be viewed as connected series of specific behaviours. The term route, with its implication of landmarks and specific responses guided by these, serves as a useful metaphor in describing taxon behaviours. Similarly, the use of the term map implies the availability of an aggregate of interrelated information with no necessary specification of guides. We intend in this section to determine those features of maps which must be incorporated in any theory of cognitive mapping.

In the final section of this chapter we translate these ideas about maps and routes into more familiar psychological terminology, providing a means for evaluating behaviours based on the two. Here, we outline the properties of the locale (map) and taxon (route) systems.

2.1. Some examples of mapping

2.1.1. STUDIES OF NAVIGATION

The ability of many animals to find their way back to their nests over large distances would appear to be based on some type of mapping system. We shall discuss two examples from the vast literature on this subject: *homing* and *migration* in birds, and open-sea navigation in humans. In the introduction to their well-known classic study of homing in the noddy and sooty tern Watson and Lashley (1915) summarized previous studies of migration and homing and the hypotheses generated to account for them. It was already known that many species of birds migrated hundreds and even thousands of miles every year to reach their breeding grounds. The most extreme example of this was the arctic tern, which appeared to spend 14 weeks of the year in its arctic home, slightly longer in its antarctic home, and the rest of the year making the 22,000 mile round-trip flight between the two. Similarly, homing pigeons could be released from new locations hundreds of miles from their nest and find their way home in remarkably short times.

In their own experiments, Watson and Lashley studied both the long-distance homing of the tern and its short-distance ability to locate its nest on the island. Although they were able to demonstrate that the birds could return to Key Island, Florida when released at distances of up to 1000 miles away, they were unable to provide evidence in support of any particular hypothesis as to how the birds accomplished this feat. Hypotheses current at that time included suggestions as to what stimuli were directing the animals and which sense organs were sensitive to these (magnetism, wind currents, direction of the sun or light, direct visual perception of the goal, special nasal senses), in addition to vaguer suggestions about hereditary topographical memory. They found no effect of occluding the nares and calculated that the island could not be visible from release points greater than l00 miles.

In contrast to these essentially negative conclusions on long-distance homing, their studies on the proximal nest-finding ability of the birds were more successful and led them to conclude that

> 'coming in from the sea, they direct their flight by the more conspicuous features of the island, the buildings, prominent bushes, etc. From these the direction is taken along the shore-line, or the edge of the cleared nesting area to the alighting place, which also offers prominent visual stimuli. From this the path to the nest is followed, either by a series of visual-motor habits built up around other nests, debris, etc., or by a series of kinaesthetic-motor habits irrespective of external stimuli' (p. 75).*

* As we shall see, this is a good description of what we call a route (pp. 83-6).

Furthermore, they were impressed by the relative autonomy of the individual components of the route:

> 'the same lack of coordination in reactions to complex situations is seen in the choice between two nests, in the re-orientation in the path to the nest, and in the reaction to changes in the appearance of the path. The reactions are to separate groups of stimuli and there is clearly no analysis of the situation as a whole. The birds do what a man would only consider doing under like circumstances; that is, they carry out in overt activity many of the same processes which in man are restricted to the language mechanism. In subjective terms, they show little or no evidence of "ideational processes" in their activities' (p. 83).

Although they recognized that this analysis could not be extended to include long-range homing abilities, they failed to consider whether these latter might be connected with the 'ideational processes' seemingly absent in the more proximal orientation abilities. Instead, they both abandoned field studies for the laboratory with Watson in particular, concentrating on experiments more similar to the habit-based proximal orientations, to the neglect of the more cognitive map-based distant homing.

Until recently, studies of homing and migration have concentrated on testing hypotheses directed towards the identification of the sensory information which the bird used to locate itself and guide it towards the goal (see, e.g., reviews by Matthews 1955, 1968). In spite of much research no single stimulus has been isolated, and work has turned towards the testing of more complicated 'cognitive' models. The most successful of these is the map-and-compass hypothesis of Kramer (1953). Griffin (1955) has pointed out that animals could find their way to a goal in several different ways: (1) piloting, which is steering by familiar landmarks; (2) compass steering, which is heading in a constant compass direction; (3) true navigation, which is heading towards a specific goal regardless of the original starting place and the direction necessary to achieve the goal. These are roughly equivalent to what we shall call *guidances, orientations,* and *map following* in the next section. It now seems clear that birds can use both compass steering and navigation in addition to piloting, as Watson and Lashley had shown. For example, if migratory birds are captured and transported in a direction perpendicular to that in which they were flying, naïve birds on their maiden flight will continue to fly in the same direction in which they had been headed and will thus miss the goal by the amount transported, while experienced birds which have made the trip before will correct for the distance they have been displaced and eventually attain their destination. The naïve birds are following a compass direction while the experienced birds are flying towards a goal.

The most adequate hypothesis to explain true homing behaviour is, as noted already, the map-and-compass hypothesis. This postulates that the bird has a map by which it can locate its present position, that of the goal, and the direction between the two, and a compass which will enable it to calculate that direction. Recent work has concentrated on the features of the compass (e.g. Keeton 1974). Thus far there are three different mechanisms by which birds have been shown to calculate compass direction; these involve the use of the sun, the stars, and magnetic fields.

Birds use the sun to calculate compass direction by measuring its azimuth* and calculating where the sun is located at that time of day according to their internal biological clock. Thus, for example, if their internal clock tells them it is noon and that the sun is in the south at noon, the bird will fly 90° counter-clockwise to the sun if its map requires it to go east to get home. The best evidence for this mechanism comes from studies in which the animal's internal diurnal clock has been artificially shifted by altering its day-night cycle. In such studies the birds do not fly in the homeward direction when released at a distant site, but head off in a direction consistent with the notion that they are calculating the compass direction of the sun on the basis of their internal clock.

A second source of compass information is geomagnetism. Again, Keeton (1974) is our source. Earlier attempts to demonstrate the use of magnetism for orientation by birds failed because the birds seemed lost when required to fly when the sky was overcast. The conclusion seems unwarranted since Keeton and his colleagues have now shown that the difficulty was an unwillingness on the part of the birds (pigeons in this case) to fly in bad weather and not an inability to orient towards home. Pigeons trained to fly in bad weather orient very well. Furthermore, this orientation appears to be independent of the bird's biological clock, since clock-shift studies do not alter the direction in which the animals fly when released. Birds flying under overcast skies are severely disrupted if magnets are fixed to their heads, and can even be made to fly in the wrong direction by the appropriate applied magnetic field. Neither of the latter two conditions has any effect on pigeons flying in unclouded skies, which might explain why the use of geomagnetism went undetected until recently.

Thus, in the adult pigeon the orientational ability is an over-determined one, and the animal can use either the sun or magnetic fields. Interestingly, in the young inexperienced bird both sources of information are required. Thus, young pigeons cannot home with magnets on their heads even in clear skies.

The third source of compass information arises from the stars. This does not appear to be used by pigeons, who are reluctant to fly at night, but is used by migratory birds. Emlen (1975) has studied the ability of indigo buntings to use this information. These birds are not born with an innately wired star map, but are born with the ability to construct map-like representations on the basis of their observations of the heavens. They need a view of the stars before they set off on their migration. During this

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* The azimuth is the arc which the sun makes with the horizon.

exposure period they 'learn' how to orient themselves by establishing a fixed reference framework consisting of the pole-star (the North star, whose position in the sky remains constant through the night) and the set of rotational movements of the surrounding stars. Thus, in addition to acquiring the ability to use the immobile pole-star, the birds can also use any of the surrounding clusters of stars which move during the night. Clock-shift experiments show that this ability, like the sun compass system, relies on the animal's use of its internal diurnal cycle to calculate the direction of the star pattern at any given time of night. Planetarium experiments, in which the 'sky' can be rotated around any star, show that the immobility of the pole-star is the crucial feature determining its choice by birds.

There would appear, then, to be considerable information about the mechanisms by which birds determine compass direction once they have located themselves and their goal on the map. However, what about the map itself? Unfortunately, little is known here, aside from some preliminary findings that pigeons systematically head off in the wrong direction at certain release sites and that these errors are probably related to local distortions in the map; the animals act as though they were some place else in the world (Keeton 1974). One thing seems clear: the birds are not using landmarks of any sort, nor are they following routes. Airplane tracking of individual birds shows that they seldom, if ever, return home by the same route from the same release site and that even when, on the later returns of a series, they come across landmarks associated with previous return flights they do not change flight plans and follow the previously used route.

Gladwin (1970) has described how South Sea islanders (Puluwatans) navigate across large stretches of open sea without the use of technical aids in his fascinating book *East is a big bird*. Very much in the same way as we have just described for birds, they seem to employ all three of the theoretical navigational devices listed by Griffin: piloting by landmarks near the end of the journey, orienting relative to the stars and sun to remain on course, and, most importantly, cognitive mapping. The major device is a spatial map of the region which locates all of the islands relative to each other, together with a large panoply of natural seamarks such as reefs and whirlpools, and mythical ones such as identifiable sea birds, fish, etc.; these latter are never observed in the places where they are supposed to be located but serve mainly to fill up the large gaps in the map which would otherwise remain undifferentiated and might suffer compression. The map also contains a superimposed star map which consists of 32 of the most prominent stars in the sky situated around the periphery of the map according to the points on the horizon at which they rise or set. The stars are chosen to cover roughly the 360° of the compass. It should be noted that in this area of the world, near to the equator, stars rise and set horizontally, so that a knowledge of the time of night and the azimuth of the star will locate it on the star map. It is clear, however, that the location of individual stars is not important but that, in a fashion similar to birds, it is the pattern of stars which provides the necessary information:

> 'it is not necessary to have a discrete point on which to set a course. Instead, to borrow an expressive image from the Mississippi River pilots of Mark Twain's day, you steer by the shape of the sky. You are sailing into a part of the heavens, not towards a dot of light' (p. 152).

In addition to memorizing the position of the islands and the stars, the Puluwatans remember all of the star courses which must be followed while navigating between any two islands.

Thus, when a boat wishes to go from one island to another, it sets off in the direction designated by the appropriate star or star pattern and maintains that direction. In addition to the stars at night and the sun in the day, the human navigator, like the bird, uses all available cues to maintain the appropriate orientation. These include a back-sight along two landmarks on the island which is being left behind to get a precise initial orientation and the use of characteristic wave patterns to maintain orientation. In addition to direction, a navigation system needs some information about distance travelled. We shall suggest in a later chapter that in animals locomoting over land this is provided in part by a collateral output from the movement-generating circuits to the map. For birds a similar output would provide less reliable information because of the large effect of wind currents. For humans incarcerated in a self-propelled vehicle no such information about speed is available. The Puluwatans have solved this problem in an ingenious way. Instead of picturing the boat moving through a stationary map they view the boat as stationary and the world as moving past them. Within this scheme each course between a pair of islands has a reference (or *Etak)* island associated with it, usually one 50 miles or so off to one side. During the voyage the navigator mentally calculates the movement of this (unseen) island relative to the star map and divides the journey into segments of time corresponding to the movement of the Etak island from one star to the next. This device, together with a rough knowledge of speed under different ocean currents, seems to predict accurately the distance and time of travel.

This brief summary of the navigational abilities of birds and humans points to the existence in both of a sophisticated mapping device together with a host of associated devices for operating on that system, providing information by which the organism can locate itself and other places within the map and determining the appropriate direction between these places. In the human system all of the information in the map has to be painstakingly learned and even to some degree the mode of using the map must be acquired. In the migratory bunting the star map which the animal uses to orient itself would appear to be pre-programmed to accept only information about star configurations and their patterns of movement during the night. Notice that a genetically pre-wired map of the stars would be disadvantageous, since the pattern of the stars slowly shifts in the sky over several thousands of years. Little is known of the map in the homing pigeon, but it would seem reasonable to assume that, by analogy with the compass in these birds, it is over-determined and receives information from several different types of stimuli distributed across the surface of the earth, such as magnetic fields and other types of radiation. In that case, it is likely to be completely innate.

Before leaving the topic of navigation there are several other studies we should consider for they all agree in concluding that something like a cognitive map must underlie complex behaviour in space. Zanforlin and Poli (1970) studied the digging activity of rats, focusing on their ability to move accurately through space while burrowing. Rats were allowed to run through an opaque tube placed in a sand-box, leading from start (A) to goal (B). Following this, they were required to burrow from A to B under either of two conditions: with or without sight of the visual landmarks associated with start and goal at the initiation of a trial. Under both conditions rats succeeded in getting to the goal through the sand, though visual cues facilitated performance noticeably. The authors excluded the possibility that magnetic, olfactory, or visual cues were used during the actual burrowing. Further, the routes taken by the rats did not match the shape of the routes used in the training tubes, eliminating the possibility that fixed sequences of responses were involved. Thus, they concluded that

> 'the burrowing rats use a complex system of kinesthetic-vestibular cues for orientation coupled with a previously acquired mental map' (p. 667).

The fact that accurate burrowing could be accomplished without visual cues indicates that localization 'in the map' can be achieved by other means, but these remain unspecified and are clearly less accurate than is vision.

Peters (1973) has described hunting behaviour in wolf-packs which, he feels, necessitates the use of the cognitive mapping concept. He cites three pieces of evidence: (1) wolves can take intentional short cuts, or detours; (2) packs can split up and re-group at some distant point, beyond the effective range of howling, such that some idea of distance and direction is required; (3) wolves can return to a rendezvous point where pups have been left *from any direction.* All this strongly implies a map-like organization of their psychological space. Peters stresses that these maps are likely to be heavily dependent upon smells, an important point as it extends the notion of mapping to other modalities. We assume, in fact, that all cognitive maps are at least potentially multi-modal.*

Menzel (1973) has described the way in which chimpanzees move about space in similar terms. A young chimp was carried (in a cage) around a field and allowed to see an experimenter hide a piece of fruit in 18 designated locations about the field. The chimps could do nothing but watch. After all the food was hidden the chimp was returned to the start and, some minutes later, set free. Five control chimps who had not been able to observe the placements of the food were also set free at the same time. The results were clear cut; the chimp who had observed the food being hidden got virtually all of it, and the others almost nothing. The detailed behaviour of these animals was noteworthy; the successful observer chimp typically ran directly to the food, ate it, then ran directly to another food site, and so on. Menzel provided routes of the animals' performances which indicate that they were using something like a least distance principle; they never behaved as though engaged in a general search strategy. Further, they rarely returned to a place from which the food had already been obtained.

In a further set of tests Menzel showed (1) that the chimps were not simply retracing routes taken while the food was being hidden, (2) that the task could be solved even when the chimp was not carried along, but instead could observe where the experimenter placed the food (it was not hidden in this study, merely placed on the grass), and (3) that the choice of where to go first was dependent upon the overall clustering of food in a given area. Note that the information used by the chimp did not depend on their own movements.

In an important set of experiments Olton and his collaborators have studied the ability of rats to collect food efficiently from several locations (Olton and Samuelson 1976, Olton 1977, Olton, Collison, and Werz 1977). They used a more formal testing situation than Menzel, a maze with eight or seventeen arms radiating from a central platform. Each arm was baited with food once at the beginning of a trial and the problem for the rat was to visit all of the baited arms without returning to a previously visited (and therefore empty) arm. Rats quickly learned to visit seven or eight arms before making a 'mistake' in the eight-arm maze and fourteen or more arms in the seventeen-arm maze. They did not use obvious strategies such as entering the arm next to the one from which they had just emerged, nor did they avoid the smells or other proximal properties of the arms they had previously entered. They appeared to be visiting spatial locations as defined by extra-maze cues. In other experiments it was shown that the memory for locations visited was not a short-lasting one since confining rats to the centre platform for several minutes after the third choice or increasing the overall time to make all choices had no effect on accuracy.

Experiments such as those of Menzel and Olton suggest that animals have a memory system for spatial locations and what occupies them, and

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* Peters also discusses why maps would provide a firmer basis for behaviour than routes. We shall be discussing this in the next section.

that this memory has a high information capacity, can be permanently modified by a single experience, and can be used in a flexible and efficient manner so that, for example, the order in which information is retrieved from the system is not necessarily the order in which it was stored. In reference to the various facts about place learning which his experiments on chimpanzees had shown, Menzel concluded that

> 'Mentalistic terms such as "cognitive mapping" do not necessarily explain the above facts, but they predict them accurately and describe them successfully' (p. 945).

It is in the hope of providing a neural explanation for such facts that the present theory has been constructed.

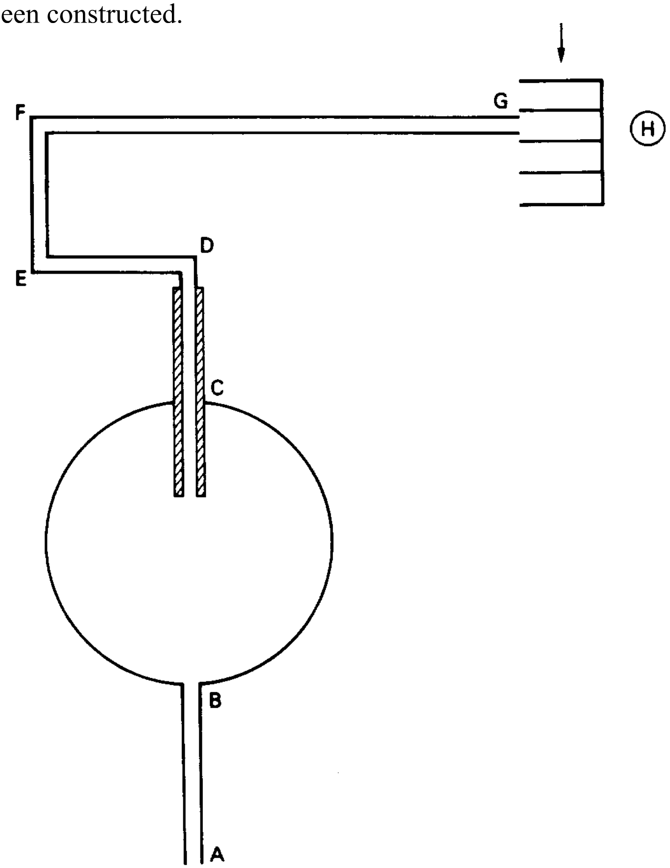

FIG. 1(*a*). Apparatus used in the preliminary training in the Tolman *et al.* (1946) study. A-B and D-G were elevated pathways while C had 18-in. high walls which obstructed the rat's vision. H was a 5-W lamp oriented to project along the alley F-G. The goal at G consisted of a series of enclosed feeding stalls so that a new rat could be tested the minute the previous rat entered the goal.

2.1.2. PLACE LEARNING

In his paper *Cognitive maps in rats and men* Tolman (1948) outlined the evidence upon which he based his theory that rats use field maps of their environment in getting from one place to another. It is worth describing one of the most well known of these findings, as we shall have cause to refer to the task again. The relevant study (Tolman, Ritchie, and Kalish 1946) used the so-called sun-burst maze (see Fig. 1(*a*)). Rats were trained to run from A along the path BCDEF to the goal G.[*] Following training, the sunburst (Fig. 1(*b*)) was introduced and the rats allowed to choose. The greatest number chose the arm pointing directly towards the goal; this is classic detour behaviour, the animal going to a place rather than making a particular response.

Hebb (1949) has described similar place-learning tendencies. For instance

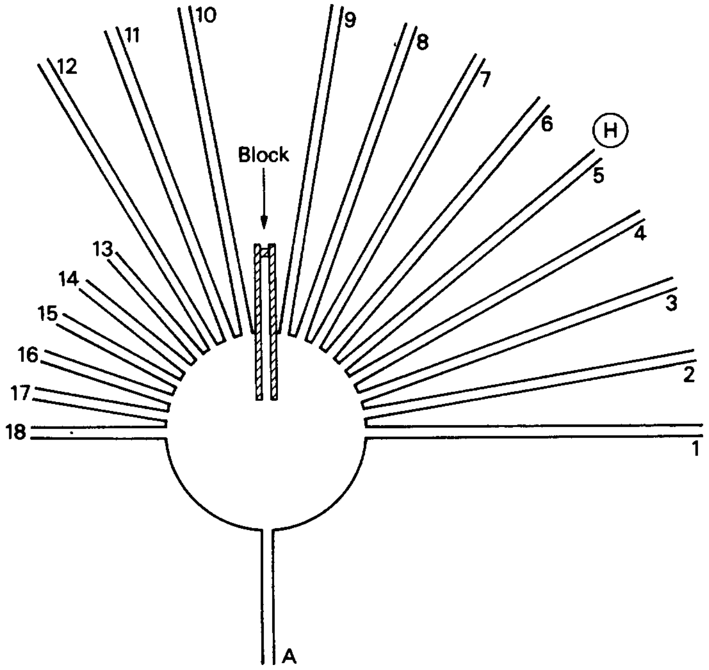

FIG. 1(*b*) Apparatus used to test the directional sense of the rats after the preliminary training was completed. The exit from the walled alley C was blocked and pathway D-G removed. Radial arms gave the animal a choice of direction over the full 180° including one (6) which pointed directly to the mouth of the goal box. In the Tolman *et al.* study 36 per cent of the rats chose this arm, while 17 per cent chose arm (*c*) which pointed in the correct direction. (Copyright 1948 by the American Psychological Association. Reprinted by permission.)

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[*] Unfortunately, a light H was located at the goal, making a strong place-learning interpretation of these data impossible.

rats were taught to run to a food dish located at the edge of an open table. Following this the table (and dish) were rotated 90°. The rats ran at least once to where the dish was previously located relative to the room. Similarly, rats will choose to approach goals which are located in the correct place, but contain the wrong cues, rather than the reverse (Hebb 1938a,b). Hebb also quotes an observation of Lashley's where rats literally jumped off into space towards the place they had previously jumped after the platform that had been there was moved.

We have recently observed the same phenomenon in an avoidance situation (Nadel, O'Keefe, and Somerville, unpublished observations, Black, Nadel, and O'Keefe 1976). Rats were trained to avoid shock in a 5 x 5 ft gridded, topless box (the walls were 4 ft high) by climbing onto a platform (8 in. square, 3 in. high) located near one corner of the box. After the rats learned to avoid, the entire apparatus was rotated 180° relative to the room and the rats placed in a corner midway between the old and new positions of the platform. Most (11 / 12) of the rats went to the *place* where the platform had been, rather than where it currently was.

All of these results attest to the importance of what is usually called place learning. This conclusion had already been reached by Woodworth (1938) in his review article, though an explanation for it was lacking. Tryon (1939) described the ability involved in terms strikingly similar to those employed in our discussion of navigation, suggesting it incorporated

> 'the *native* capacity of the animal to evolve directional abstractions regarding the plan of the maze. These abstractions are developed out of sensations *derived from stimuli received from the maze during learning* . . . when a rat has developed these directional sets, they guide his movements in the maze, even in the presence of radical stimulus changes—he becomes free of the specific stimulus features of the maze' (p. 414).

While most authors agreed with Tryon as to the nature of the behaviour shown, most disagreed as to the source of the information providing orientations. Hebb (1938a), for instance, felt that information from the distal environment, rather than the test apparatus itself, was crucially important in defining places and allowing for general orientation.*

This emphasis upon the information provided by the remote environment eventually led to a 'resolution' of the extensive battle between the proponents of cognitive map theory and S-R learning theory concerning the validity of place- and response-learning explanations of maze behaviour. This issue was fought out primarily on the miniature battlefield of the T-maze and involved dozens of studies in the late 1940s and early 1950s. Hull's supporters attempted to show that animals always make turns, while Tolman's supporters attempted to define conditions under which animals went to a place, regardless of the response involved.* Munn (1950) summarized much of this work by noting that

> 'experiments on place-*versus*-response learning have, in general confirmed Tolman's prediction that *in a heterogeneous environment* . . . rats will learn to run to one place from two different directions more readily than they will learn to make the same turn (right or left) to different places' (p. 413).

In such a situation the place-learning group reaches criterion faster than the response-learning group if a relatively constant heterogeneous environment is available. When a homogeneous environment is used (e.g. Blodgett and McCutchan 1947) this superiority disappears.

In his paper *A resolution of the place-vs-response question* Restle (1957) concluded that

> 'there is nothing in the nature of a rat which makes it a "place" learner or a "response" learner. A rat in a maze will use all relevant cues, and the importance of any class of cues depends on the amount of relevant stimulation provided as well as the sensory capacities of the animal. In place-response experiments, the importance of place cues depends on the amount of differential extra-maze stimulation' (p. 226).

Thus, according to Restle place learning was no different than response learning; it merely utilized a different class of cues. This, of course, left unresolved the issue of why place learning seemed to have different properties than response learning, e.g. flexibility.

It was still necessary to explain why, within this view, distal cues should be so effective in specifying places. Hebb (1949) suggested that it had something to do with the fact that distant objects remain more constant (in perception) as animals moved compared with nearby objects. Though Hebb mentioned the *order* between stimuli he did not stress this point, rather he stressed the single object. By so doing, he missed not only the theory of invariance later proposed by Gibson (1950) but also the central point of the role of distant objects. In our view distal cues are important in specifying *directions,* since they do not change relative positions as the organism moves in its local environment; on the other hand, this very property means that distal cues, by themselves, cannot distinguish amongst *places* in that environment. Places would seem to be defined by extra-maze cues which are close enough to the animal for its movement to change the angles between them but not so close that movement causes changes in their spatial ordering. There is another reason for assuming that the distal cue hypothesis of place learning cannot be correct. It is well known that when a cue is spatially distant from the site of the response to be made to it,

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* He concluded that 'lower mammals may have a type of visual organization . . . dominated by a perception of spatial relations between objects rather than of their intrinsic properties' (p. 350).

* Tulving and Madigan (1970) summarized this particularly lively period in rat psychology as follows: 'place-learning organisms, guided by cognitive maps in their head, successfully negotiated obstacle courses to food at Berkeley, while their response-learning counterparts, propelled by habits and drives, performed similar feats at Yale' (p. 440).

organisms have great difficulty in learning (cf. Cowey 1968). If place learning involved nothing more than the use of a distal cue it should fall prey to this difficulty; on the contrary, place learning often occurs much more rapidly than does cue learning, as the Menzel (1973) and other studies show.

In summary, Restle denied that there is anything distinctive about the way in which places are defined. By so doing, place learning could be incorporated within the broad S-R framework, and there was no need to talk about cognitive maps or qualitatively different forms of learning. In contrast, Tolman and his colleagues saw place learning as quite a different kind of learning. Within a cognitive map the *relations* between objects were crucial, not the relations between specific objects and the organism. The overall conclusion from the early work with animals, then, was that something like place learning existed, but that it was not anything unique. The concept of cognitive mapping as an explanation for place learning lost favour. We have just discussed several areas of research indicating that Tolman was probably on the right track. The continuation of the story about cognitive mapping requires that we turn to the burgeoning field of environmental perception, where geographers, architects, and psychologists join hands (reluctantly) to explore ways of conceptualizing spaces too vast to be experienced in a single take.

2.1.3. COGNITIVE MAPPING IN HUMANS

Environmental psychology, or behavioural geography, grew out of the need to explain man's perception of large-scale environments, those which extended beyond the range of immediate perception. In other words, it is directly concerned with what we have been calling unitary space. For most of the 20th century this problem was either ignored or treated as a mere extension of other perceptual problems. The failure of this approach to account for environment perception, which we have documented, has led to a radically different approach within which the notion of cognitive mapping is central. In a recent article Ittelson (1973) both summarizes the failure of the old methods and the reasons why environments must be considered in themselves:

> 'in the history of experimental psychology the overwhelming bulk of perception research has been carried out in the context of object perception, rather than environment perception, with the findings of the former providing the basis for understanding the latter. Virtually every major school of psychology in the past 100 years has investigated its perception problems in the context of object perception; has developed its theory of perception from the results of these studies; and has then transferred the explanatory system thus derived into the context of environmental perception. As a result, the investigation of perception has lost the essential esthetic unity without which any pursuit leads to chaos, rather than resolution' (p. 3).

and

> 'the distinction between object and environment is crucial. Objects require subjects—a truism whether one is concerned with the philosophical unity of the subject-object duo, or is thinking more naively of the object as a 'thing' which becomes a matter for psychological study only when observed by a subject. In contrast, one cannot be a subject of an environment, one can only be a participant. The very distinction between self and nonself breaks down: the environment surrounds, enfolds, engulfs, and no thing and no one can be isolated and identified as standing outside of, and apart from, it' (p. 12-13).

Ittelson goes on to describe those features of environments which demand that they be treated separately in theories of perception. The fact that they surround means that one cannot observe an environment; rather, the organism *explores* it. Ittelson states that 'the problem of exploratory behavior . . . becomes central to the study of environment perception' (p. 13). Further, he stresses the fact that environments are multi-modal and unitary. Added to these characteristics which separate environments from objects, and with which we would agree, Ittelson discusses features of environments which must be included in any assessment of perception. Thus, environments involve actions which are purposeful because they possess meaning and ambience. This emphasis upon action relative to a meaningful environment has been incorporated within most work in this area, and it has led to the assumption that cognitive maps must code both meaning and response. We shall argue, on the contrary, that such information, though certainly applied to environments as well as to objects, does not necessarily form part of what is encoded within cognitive maps.

2.1.3(a). Evidence for mapping. Three papers at the start of this century introduced the concept of 'mental maps' to the geographical and psychological literature. Gulliver's (1908) brief note mentioned such maps in the context of teaching children orientation. A much more interesting paper was published by Hutorowicz (1911), comprising an abridged translation of a monograph written by B. F. Adler on the *Maps of primitive peoples.* In this survey of the folklore of map making Adler collected maps and anecdotes concerned with the various forms of geographical representations used by different cultures. He commented on the island navigators and their use of navigation by the stars, and also noted that nomadic tribes in Siberia, as well as Eskimos, had devised a similar form of navigation using the North star. Adler quoted a number of interesting examples of the way in which so-called primitive people prefer to draw a map when asked for directions, and comments that

> 'they seem to think that this graphic delineation will be more helpful than mere verbal guidance' (p. 670).

This description of the widespread existence of maps was followed shortly after by Trowbridge's (1913) article on 'imaginary maps', which provided the first clues to the structure of cognitive maps. Trowbridge distinguished between two sorts of maps, varying in the focus of orientation: *egocentric maps* use the four points of the compass and are found in 'civilized' man; *domicentric maps* orient one according to some fixed reference point, usually the home, and are found in birds, beasts, young children, and 'primitive' man.

Trowbridge noted, but did not comment upon, the fact that the 'advanced' egocentric maps could not direct an organism accurately unless 'the path which he passed over is known' (p. 889), while the 'less-advanced' domicentric maps could provide accurate localization. The latter are more likely to keep one oriented, while the former can cause confusion and the loss of bearings. Much of this seminal article is concerned with the kinds of systematic errors introduced by the use of egocentric maps, including the way in which reliance upon compass bearings can induce massive errors in distance judgments. All of these distortions could be described as subjective rotations of egocentric space within the arbitrary reference framework of the compass. Such things as rivers and city-street orientations were often responsible for the distortions. At the same time, Trowbridge indicated that the people with these distorted imaginary maps *were* aware of proper directions, but that these were somehow overridden by the egocentric maps.

Given that the use of these imaginary maps often led to disorientation, it is surprising that Trowbridge did not comment further on the superiority of the domicentric maps. None the less, it seems clear from his report that an abstract orientation system, capable of introducing vast distortions in environment perception, is often superimposed upon a more primitive system capable of roughly veridical representation. These data suggest that the insistence upon subjective loadings of map information might refer primarily to imaginary maps, and not necessarily to the primitive maps that seem to provide an objective view of the environment and its dimensions.[*]

For some 40 years, excluding studies on the orientation of the blind,[**] the concept of mental mapping virtually disappeared from the literature,[***] to reappear only in the past decade. These recent studies (cf. Downs and Stea (1973) for a sample of recent work and references to most of what is available in this field) basically confirm the picture presented by Trowbridge, though few of them are directed towards an understanding of the primitive, but more accurate, maps. The reasons for this can be seen in the basic definitions given to mental maps by most authors, which follow Ittelson's ideas. Kaplan (1973) defined cognitive maps in a representative way:

> 'The cognitive map is a construct that has been proposed to explain how individuals know their environment. It assumes that people store information about their environment in a simplified form and in relation to other information they already have. It further assumes that this information is coded in a structure which people carry around in their heads, and that this structure corresponds, at least to a reasonable degree, to the environment it represents. It is as if an individual carried a map or model of the environment in his head' (pp. 275-6).

Kaplan assumed that there were four types of information that would *necessarily* be contained in a cognitive map, pertaining to four psychological processes: (1) recognition of location and objects; (2) prediction of what leads to what; (3) evaluation of what is good or bad; (4) action relative to the environment. This set of assumptions parallels those of Ittelson and has formed the basis for research in this area. It has proven much easier to study motivation and action than localization and prediction, for the former often distort the latter. Many studies have shown that the value one attaches to a place affects one's perceptions of its distance from other places (e.g. Briggs 1973).

There are various ways in which these data stressing the distortions and non-objectivity of cognitive maps can be interpreted. First, one could assume that cognitive maps must be value oriented. Lewin's field theory then becomes the progenitor of present-day research, and the notion of objective spatial mapping is discarded; this approach has dominated environmental psychology to date. On the other hand, one could assume that information pertaining to values is not part of the map itself, but rather is attached to an output from a veridical map. We feel this latter approach is the better one.

Trowbridge's data favour our view. The distortions seen in his egocentric imaginary maps were imposed by various environmental features and the cardinal system of orientation *upon a more primitive veridical map.* Assuming the subjectivity of the cognitive map raises logical difficulties, it is hard to imagine how the non-commutativity of distances (e.g. Lee 1970) could be encoded in the same structure which provides for the easy use of alternative paths to the same goal and for the rapid reversal of paths. Non-commutativity almost demands that the map represent paths in terms of the individual responses, or landmarks, involved in traversing them, and this might be why Kaplan and others insist that action patterns

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[*] In a recent book on *Mental maps,* Gould and White (1974) seem to have misunderstood the point of Trowbridge's work.

[**] A monograph on this topic was published by Worchel (1951). He described the use of a test of triangulation; subjects were walked two legs of a triangle and required to return to the start. Blind subjects were worse than sighted subjects, but their performance was reasonably good none the less. More important, age of blinding, as well as chronological age, did not correlate with performance. Considerable other work on spatial orientation in the blind has appeared (cf. Jones 1975 for a review). As this work is primarily concerned with the primacy of vision, or touch, in spatial knowledge, we shall not treat it here.

[***] A few papers did appear (e.g. Ryan and Ryan 1940, Lord 1941, Gregg 1939). Of course, we are omitting mention of Tolman and Lewin in this context, though they certainly carried forth the tradition of environmental perception during this time. One last pioneer in the wilderness worth noting was Bartlett (1932). His notions of mental schemas were close to the idea of mental mapping, though his interests were more related to the cognitive aspect of these representations than to any spatial component they might have. His work clearly prefigured the current popularity of research on imagery, about which we will have more to say later (pp. 389-91).

are also encoded in the map. We feel that this seriously overloads the system, as well as raising considerable difficulties in converting the concept to some neural reality. Some recent research supports our approach; Moore (1973) has shown that reversibility of spatial operations (going from A to B and B to A) correlates with the presence of map-like representations, particularly in basically unfamiliar areas. In the absence of such representations reversibility seems to involve the learning of two separate routes.* Similarly, the logic of how cognitive maps are built up through action, and the ability they impart to get from any place to any other place, argue against the assumption that actual behaviours are coded into the map. This would greatly limit the flexibility associated with behaviours based on cognitive maps.

Thus, there seems little doubt that map-like representations of the environment are constructed by humans, as well as by other species, but there remains some doubt as to the nature of the information included in these maps. It must be stressed that cognitive maps are *not* models, in the organism's head, of the environment. Kaplan (1973) seems to imply this, as the quote above indicates, while others argue against this view. Thus, Blaut, McCleary, and Blaut (1970) note that the cognitive structure 'has the functions of a map but not necessarily the properties of a pictorial mental image' (p. 337). This distinction is important, both in considering mental maps in particular and any form of abstract imagery in general, as we shall see later (pp. 389-91). 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 behaviour dependent upon place information can be generated. While we shall argue that this information base pertains solely to processes of recognition and prediction, in Kaplan's terms, we are aware that other forms of information might be included as well.

2.1.3(b). Ontogeny of cognitive mapping in humans. It is obviously of great interest to ask when these cognitive maps first appear in children. The study of the ontogeny of mapping, however, is hindered by the difficulty in getting relevant data from infants; it is virtually impossible to get an answer to the question of whether or not the mapping system is innate. However, it remains possible to assess Piaget's model in this light, for he postulates that the ability to generate representations of the environment involving unitary space becomes available only after extensive learning, certainly not before 10-12 years. Studies such as those by Olson and Baker (1969) and Shantz and Watson (1971) suggest that complex spatial abilities arise much sooner than Piaget would predict, but they do not directly test mapping.

More recently, Blaut and his co-workers (Blaut *et al.* 1970, Muir and Blaut 1969-70) have tested young children from various cultures on map reading. They found that this ability exists prior to training in children as young as four years old. They state that their evidence

> 'seems to require a developmental model of environmental behavior in which the early emergence of mapping behavior is explicitly predicted' (Blaut *et al.* 1970, p. 347).

Kosslyn, Pick, and Fariello (1974) have shown that while adults are better at estimating veridical distances, children (4-11 years) are none the less remarkably good at it. Further, the data provided by their subjects were scaled to determine whether they were best represented as using a Euclidean metric or a city-block reference frame. The data suggest that

> 'the Euclidean solutions more effectively capture the *Ss* spatial representations' (p. 713).

In fact, the city-block method provided a poor fit to the data.* This is reminiscent of Trowbridge's work. All these data, then, indicate that young children can use a mapping system and that it seems to work according to Euclidean principles. Taken in conjunction with the data reviewed earlier, it would seem as though Piaget's assumptions about the ages at which various concepts of space arise are incorrect. We cannot say, for humans, whether or not mapping is available at birth, though the perceptual capacities for it seem present shortly thereafter. As we have seen, the data from infra-humans point in the same direction.

2.1.3(c). Conclusions. The fact that most investigators have stressed the subjective aspect of mental maps means that the models derived from their studies are not really suitable for our purposes. These studies support the notion that such maps exist in adults and young children equally, but they do not tell us much about how these maps function. Before we can translate these ideas into a neural theory we must formalize them. The best way to do this is to go back to the beginning and look directly at the properties of maps and the major alternative means of representation, routes. This connection between mapping, the concern of the geographer, and cognition, the concern of the psychologist, has been central to the efforts of both environmental psychology and behavioural geography. The circle leading from Kant to cognitive maps comes full turn, as epitomized in a recent paper by Richards (1974), *Kant's geography and mental maps*:

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* Stea (1969) has provided some data on the relationship between familiarity, subjectivity, and non-commutativity, which suggest that 'the more familiar a subject is with a given trip, the more accurate is his estimate (of distance), but the more discrepant are his estimates in the two directions of travel' (p. 240). He assumes this discrepancy arises as a function of the diverging attractiveness of the two end points with increasing familiarity.

* The authors concluded that this 'mapping' did not include information about the movements involved in going from location to location.

> 'How then- if at all- do we manage to pass beyond the field of our immediate sensations and connect up the diverse experiences which we have of our environment? . . . The answer proposed by the contemporary behavioural geographer is that we impose a synthetic unity on the manifold of our spatial perceptions (to borrow Kantian terminology) through the construction of mental maps' (p. 10).

2.2. Maps and routes

One of the deficiencies in Tolman's notion of a cognitive map is that neither he nor any of his students tried to specify in detail the properties of maps and to contrast these with other potential means for getting around in the world. We hope to remedy this situation here by giving a detailed account of the properties of maps and routes. We hope the reader will bear with us here in what may appear at first glance an unnecessary digression into theoretical geography. We shall draw heavily on the properties of maps in subsequent chapters in our attempt to spell out the workings of a neural cognitive map.

2.2.1. TO GET SOMEWHERE

'How do I get to Upshire?' enquires the lost rambler.* Several different answers are possible. In the relatively rare case in which Upshire is visible from the start it can be pointed out, or its direction indicated. Usually this is not possible, and it is necessary either to provide the rambler with a more elaborate set of instructions as a *route* or to locate the rambler and destination on an Ordnance Survey map, leaving the choice of a path to the walker. Such routes and maps have quite a different form, though they can serve the same purpose at times. In the discussion which follows we shall set out the properties of routes and their components, *guides*, and *orientations*, on the one hand, and maps on the other. Then we shall compare and contrast them, pointing out the advantages and disadvantages of each. Some of our ideas on this subject owe their origin to the philosopher Toulmin (1953) and the geographer Board (1967). As illustrations we have chosen part of a route from a popular book on country walks in and around London (*Country Walks* 1971). A map of the area through which the route goes is shown as Fig. 2. The excerpt from the route, which starts at High Beach, is as follows:

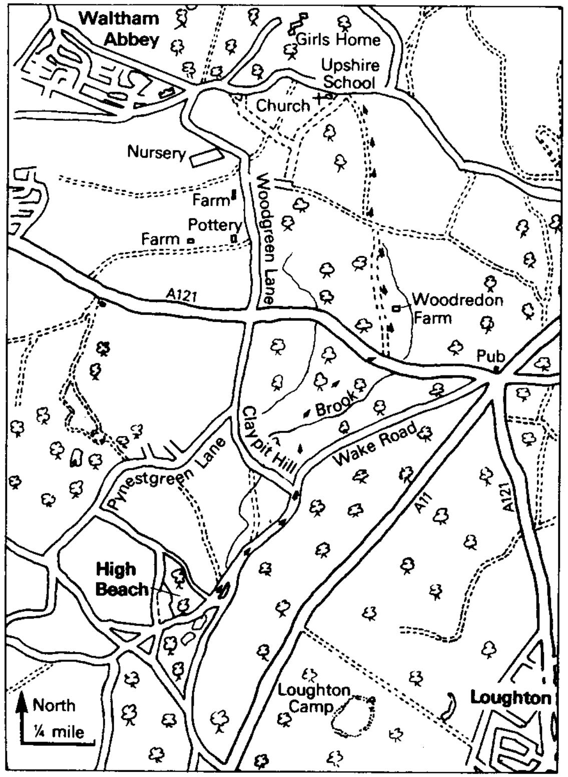

FIG. 2. A map of north-east London showing the area referred to in the *London Country Walks* (1971) route. (Based on the Ordnance Survey Map with the sanction of Her Majesty's Stationery Office).

> 'Keep forward for 500 yards beyond the little tea hut to a point where the road divides after bearing leftwards. Go along the right fork for 30 yards, then strike obliquely left into the forest, passing between two massive beeches. The old track, the Verderer's Ride, is now elusive, but there is no need to fear going too far astray. If you continue ahead, leaving the road gradually on the right, you soon find that the ground falls away sharply on the left. Bear slightly right, and pick your own way through the trees; all you have to remember is to keep near the upper slopes of the wood, keeping the lower ground on your left. Keep on for almost ½ mile, when you may hear the sound of main-road traffic ahead. After crossing one or two little forest brooks you will reach the road at the top of a hill, near a bus stop (if you strike the road lower down, simply turn right to the top of the hill). Almost opposite, at the crest of the hill, you will find a metalled drive that leads through the trees to Woodredon Farm. Do not miss the pretty Georgian house on the right as you pass the iron lodge gates (and the notice that keeps the next mile of your walk free from cars and bicycles). After passing the mansion on your left, the drive winds left-wards, then twists right again to emerge, after ½ mile, at Upshire.
>
> Upshire is a wayside settlement near the forest's edge. St. Thomas church was built in 1902 but looks much older. The spire is a useful landmark' (p. 37).

Route instructions, as this example shows, are a list of stimulus-response-stimulus (S-R-S) commands which lead the rambler from one sight to another, from the feel of walking on uneven ground to the sound of distant cars, from babbling brook to bus stop. To get from one landmark to the next he must strike obliquely left, bear slightly right, pass between

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* A rambler is a country walker.

beeches, keep forward, cross, continue, go. Careful examination shows that within each S-R-S instruction the emphasis can be on the stimulus or cue component, on the one hand, or on the response requirement on the other. We shall call the former type of instruction a *guidance* and the latter type an *orientation* or *direction.*[*]

A guidance directs attention to a particular landmark or object and requires that the rambler approach it or maintain a certain egocentric relationship to it, regardless of the behaviour involved. Guides are often localized and stationary. The most obvious guide is the destination itself, but this is often only perceptible just before it is reached. More often a guide is a prominent object or stimulus at the goal or before it: a tall building, mountains, the distant sound of traffic; 'the spire (of St. Thomas church) is a useful landmark'. Guides need not be localized or stationary; they can be extended (in *one* dimension) as are streams and trails, or they can be mobile. The best guide of all is a fellow rambler who knows the way to Upshire.

No specific motor behaviours are selected by guidance instructions. Rather, any behaviour can be used which, in the case of the fixed localized landmark, reduces the distance between it and the rambler or, in the case of mobile or extended guides, maintains their distance within certain narrow limits: thus, 'follow this trail regardless of how it twists and turns', 'keep near the upper slopes of the woods', not too close, not too far. To reach those massive beeches at the beginning of our journey might require either a very shallow left turn or a virtual about-face, depending on where one chose to 'strike . . . into the forest'.

The second type of route instruction, the orientation or direction, focuses on the response requirement to the neglect of the cue aspect. Orientations can be interpreted as sets of instructions for aligning the egocentric body axis relative to some other axis. In the simplest case this involves a rotation relative to the present orientation, i.e. a simple turn and continuation straight on. Our route to Upshire tells us to 'bear slightly right', 'strike obliquely left'. One of the problems with orientations is the difficulties organisms have in going straight solely on the basis of interoceptive and proprioceptive cues. Thus, we find that accompanying an orientation there is often a reference to a landmark and how to use it in order to 'keep straight'. The best such landmark is one which is distant and in a straight line with the starting point and the goal. By heading towards this landmark one can keep on this straight line and eventually intersect with the destination. This directional cue is similar in some respects to a guidance, but there are important differences which will become clear when we discuss the different problems each presents to the traveller who gets lost or is faced with an obstacle.

Usually one is not fortunate enough to find a directional cue directly behind the destination; instead, one must make do with one off to the side. This can be used in an approximate fashion: 'keeping the lower ground on your left', 'passing the mansion on your left'. A more sophisticated use of such a landmark to describe a straight line would involve calculations as to how fast the landmark ought to sweep across egocentric space for the speed at which the traveller is moving. Very distant stationary objects need only be kept in the same part of egocentric space. The use of the compass for direction finding is a good example of this strategy. The needle always points to an imaginary distant object which acts as a landmark for the North-South-East-West framework, and any given direction is measured as so many degrees rotation of the body axis relative to this distant framework. Finally, if the distant reference object is not stationary but moves in a predictable fashion, as does the sun for example, internal or external clocks can be used to calculate the relationship between the reference and the external axis. Homing pigeons and migratory birds clearly do this in reference to the sun, as we have seen.

2.2.2. ROUTES

Before turning our attention to maps we can outline some of the positive and negative features of route. One thing is clear: routes *imply goals which imply motivations*. Why do you want to go to Upshire? The second stimulus in each S-R-S link can be looked on as a subgoal in the overall goal directed chain.

Routes direct attention to particular objects or specify turns within egocentric space. They are inflexible, must be used in the correct sequence, and only rarely allow freedom of choice to the traveller. Three circumstances in which some choice exists come to mind: (1) the trivial one in which all possible routes converge on the goal (or subgoal) either because of their shape or the size of the goal; (2) the more common one exemplified in our passage where the goal is an extended guide crossing all possible routes, 'pick your own way through the trees . . . you will reach the road'; finally, (3) alternatives are sometimes provided in case one set of guide instructions proves inapplicable. As we shall see presently it is this general lack of flexibility in the face of an ephemeral environment which is one of the main drawbacks of routes.

The knowledge required to follow a route is usually quite simple. In the case of a guidance one must be able to identify it under its various aspects and tell how its distance is changing, usually by monitoring its size or intensity.[*] Orientations are equally simple, involving rotations within

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[*] The reader should be warned that we are giving these and similar geographic terms rather restricted definitions and they are not to be understood in the more general senses used in common parlance.

[*] This 'simple' knowledge, of course, is just that specified by Poincare and others as lying at the base of spatial representation. We have already argued that identifying objects requires, at least in developmental stages, a spatial framework similar to a map.

egocentric body space and maintaining stimuli in a particular part of that space. One result of this simplicity is speed. The actual pace of movement does not figure in any of these instructions and is limited only by other considerations such as the endurance of the traveller and the speed at which he can access and process information at the subgoals. During the remainder of the link the traveller is operating on information which is already available.

The major drawback of routes, their inflexibility, leads to another vulnerability. Consider the following ways in which one can get lost. The route instruction can be physically or mentally degraded (smudged word, forgotten orientation), landmarks or other guides may be destroyed or changed (snow, fallen trees), the translation of the instruction into behaviour may go awry (an elm is mistaken for a beech), a momentary lapse of attention leads to a subgoal being missed. It will be instructive to consider the strategies available when one loses the way or is forced to detour while following a route, because it is here that the differences between routes and maps are most clearly revealed. Getting lost is such a problem that routes often attempt to foresee possible lapses of attention or deviations and provide appropriate cautions, 'the old track ... is now elusive', or corrections, 'if you strike the lower road, simply turn right'.

Deviations from the intended route while following a guide can only be corrected by random wandering until the guide is located again. In the case of a prominent fixed guide this is often successful; extended guides such as trails or streams, on the other hand, will only be found if the appropriate direction of search is used or if a sophisticated strategy such as moving in ever-widening circles is adopted. Finally, it is almost impossible to find a lost moving guide without the most sophisticated communication strategies. Note that in all cases it is important to find not just any mountain or any stream, but to find the same mountain or stream. This brings up the problem of re-identification again, as discussed before. An additional problem with an extended guide is once having located it to follow it in the correct direction.

Orientations are even more difficult to re-establish once they have been lost. It is not enough merely to re-adopt the original orientation, since this was only appropriate along the original axis. Once off that axis an entirely new orientation is required. Thus, orientations are only useful when both the position of the traveller (the body axis) and some external axis are specified; they are purely egocentric tools. If one is forced off one's route by an obstacle, rather sophisticated geometric calculations would be required to regain the original orientation and axis. Landmarks are also useless since, again, they mark the orientation only in conjunction with the original axis. The farther away the landmark, the more the goal will be missed. One of the major problems with an orientation is simply knowing whether one is lost or not. Many readers will have had the experience of driving several miles along a road looking for a turn-off indicated by the route instruction with a mounting feeling of panic that it had been missed. Here, some indication of distance, no matter how crude, is useful.

Finally, there is the question of reversibility of operation. Given a map or route, one might ask if it can be used as easily in the reverse direction from goal to start. We have already seen the importance of this property of reversibility in discussing Poincare and Piaget, both of whom consider reversible displacement groups to lie at the root of spatial concepts. As we shall see, maps are unambiguously reversible, since they provide a means of getting from any place to any other place. For routes the answer is more complex and requires closer analysis. Let us start at the end of our route instruction, at Upshire, and see if we can transform it to take us towards High Beach. Such a reversed set of instructions might read as follows:

> 'Upshire is a wayside settlement near the forest's edge. St. Thomas church was built in 1902 but looks much older. The spire is a useful landmark. From Upshire, a metalled drive goes for 1/2 mile, first twisting left, then winding right-wards before passing a mansion on your right. Pass the iron lodge gates of Woodredon Farm (and the notice that has kept the last mile of your walk free from cars and bicycles); do not miss the pretty Georgian house on the left. The drive leads through the trees away from the Farm to the crest of a hill opposite a bus stop (on a road)'.

We can examine this reversed route and see how it was done and how useful it would be. It is immediately clear that extended guides such as the metalled drive remain useful, while distant landmarks such as the church spire are of little value. Walking away from such landmarks is hardly the reverse of walking towards them from a particular starting point.* Even with the extended guides there may be some trouble finding them. It is easy to find a large area such as Upshire when entering it from any one of many metalled drives; however, it would be quite difficult to find the correct metalled drive leading away from Upshire without additional information. In order to use orientations in the reverse direction it was necessary to make certain substitutions: left for right, retreat from for approach towards. In general these substitutions are successful and provide the primary basis for the reversibility of route statements. In our later discussion of language (cf. pp. 391-410) we shall compare this limited reversibility of routes with the syntactic transformations rules of Chomsky and others for transforming the surface structure of sentences (as in active to passive modes). Routes, then, are rapid and easy to use, but because of their inflexibility very vulnerable and easily rendered inoperative by changes in the route statement or the environment. Under certain

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* We have already seen, in our discussion of the South Sea island navigators, that two landmarks aligned in a certain way can give a direction which is equally useful both in approach and retreat.

circumstances transformation rules can be applied to them which allow them to be used in the opposite direction.

2.2.3. MAPS

In marked contrast to these properties of routes are those associated with maps. As with our discussion of routes it will be helpful to have in front of us a particular map to illustrate the discussion. We have chosen the map which corresponds to the previously used route instruction to facilitate comparison (see Fig. 2).

The simplest definition of a map is that it is the representation (usually two dimensional) of a part of space. The constituents of space are places, and thus an alternative definition of a map is the representation of a set of connected places which are systematically related to each other by a group of spatial transformation rules. The notions of place and space are logical and conceptual primitives which cannot be reduced to, or defined in terms of, other entities. Specifically, places and space are not, in our view, defined in terms of objects or the relations between objects. The absolute space defined by Kant exists in the absence of objects. It is sometimes convenient to locate a place by reference to the object occupying it, 'she was standing over there where the desk is now', but this is only a convenience. This freedom from reference to any specific object or set of objects is one of the most important properties of maps and is easily demonstrated by reference to our map. Consider two maps of the same terrain (and therefore containing the same places) which could be derived from this map. In the first, only man-made artifacts such as roads and houses would be represented; in the second, only natural objects such as trees, hills, and lakes would appear.

There are two broad classes of maps, *topographic* and *thematic*. Topographic maps are used for finding one's way around a part of the world and will be of primary interest to us here. In the topographic map the entities which are located in space are symbols for objects. The system for placing items on this type of map usually tries to preserve the Euclidean relationship between angles and dimensions, although there may be some compression in one dimension and small objects such as roads are often made disproportionately large so that they may be seen easily. The thematic map is a specialist map used to display or emphasize particular selected features or concepts such as the distribution of rainfall or the percentage crop yield over an area:

> 'Thematic maps represent not only facts, but ideas, hypotheses, and the results of analysis and synthesis' (Miller and Voskuil 1964, cited by Board 1967, p. 713).

Only rarely is the thematic map used to guide the traveller from place to place, and there can be a relaxation of the spatial transformation rules to suit the particular purpose of the cartographer. For example, some thematic maps represent the relative concentration of some feature by the area given to it, such that there is no relationship between space on the map and space in the real world.

Reading maps is a more complicated affair than following route instructions. Let us assume one is at High Beach and wants to get to Upshire. First, it is necessary to locate one's place and one's orientation on the map. This can be done by identifying several surrounding landmarks which can be seen (or heard) from one's position, and looking for these on the map (or vice versa). Two such objects and their spatial relationship will place the observer on the map. Here we have a complicated process in which the objects and their relationships in egocentric space are translated into three objects in unitary, or absolute, space. Alternatively, one need only identify one object together with a direction. Usually this latter can be obtained from a compass reading or from the location of a distant object such as the North star. In this process of locating a place on the map there is a considerable amount of surmise and conjecture. 'If that is Loughton in the distance, then there ought to be a church in this direction, but since there isn't then that can't be Loughton, but there is a church in this other direction, so that built-up area must be Waltham Abbey'. Additional predictions of objects to be seen from that position can be made, and the position further verified. When one's place on the map has been ascertained, along with the relative orientation of the map to the terrain, the next step involves locating one's destination on the map and calculating its direction from the starting point. Thus, Upshire lies almost directly to the north of High Beach. Now, one has the choice of many different routes which can be derived from the map, all of which converge on Upshire. Some are more direct than others, some go through wooded areas, others along country lanes, some are beautiful, others mundane, all end in Upshire.

The first striking feature of a map is its flexibility. Whereas a route specifies a starting point, a goal, and a particular direction of movement from the former to the latter, a map specifies none of these, either in its construction or its usage. It can be used with equal facility to get from any particular place to any other. Additional flexibility derives from the freedom from specific objects and behaviours. If one path is blocked another can be easily found and followed. If a storm destroys a prominent landmark, an alternative is readily available. As we have noted, unless these calamities are foreseen and alternatives specifically included in the route statement, they usually destroy the usefulness of the entire route.

This flexibility means that any given map can be used for a variety of goals and purposes. The map itself may have been designed with a purpose, as we have seen with thematic maps, but the heavy rainfall in London is no more the focus of a weather map than is the light rainfall in Birmingham. Furthermore, even if the map had been designed with a purpose in mind (the cartographer secretly held stocks in a London umbrella firm), it could be used for an entirely different purpose (the Royal Society of English Rainmakers cancels its annual convention in Birmingham). More often,

> 'the natural development of the map is the desire which necessity, or curiosity, imposes on mankind to explore the earth's surface, and to move from one part of that surface to another—working from the known to the unknown—on the path of experience and enquiry' (Fordham 1921, p. 1).

Another feature of maps is their high information content, which is equivalent to all of the possible routes between the places depicted on it. Consider, in addition, the way information is stored in a map. Each new item which is located on a map is automatically related to every other place and item already on the map. A change in a feature of the landscape occasions only a single alteration in a map, yet it changes every route statement in which that feature occurs. It has been estimated that the amount of information contained in a medium-sized map of the United States of America is 100-200 million bits (Roberts 1962).

A further, related, feature of maps is their high safety factor and their great resistance to degradation, both of which are achieved without any marked redundancy of information. The high information content of a map enables the traveller to constantly monitor his position by checking predictions from the map about the location of objects. Furthermore, a map can suffer a considerable loss of information, either through changes in objects in the environment or through deletion of information from the map, before it becomes useless. Thus, the problems of getting lost or being forced to take a detour, which presented such formidable challenges to the traveller relying on route instructions, are not significant for the map reader.

The disadvantages in using a map are associated with the necessity of using special codes for constructing them by placing items in specific locations, on the one hand, and reading routes off it on the other. The large number of routes from any place to another can also serve as a disadvantage. The decision as to which route to use, coupled with the time needed to employ the coding system, greatly increases the time required to read a map in comparison to that needed to follow a route. Yet another disadvantage of a map is that the information contained in it cannot be as exact as that contained in a route. As we have seen, there is inevitably some distortion in a map. Route statements, on the other hand, can be made as detailed as possible. If a flexible, yet detailed, system for finding places was desired, one could not do better than to use a map to decide which route to take to get there and then to have a series of route statements to give the final details required.

Before turning to an examination of how neural cognitive maps might work, it would be useful to summarize the differing properties of routes and maps. This summary is given as Table 1, and the information in this table will serve as the basis for translating the geographic concept of a map into a potentially useful psychological tool.

TABLE 1

Properties of routes and maps

	Route	Map
Motivation	The final stimulus is the goal; the route is built with this in mind	No object or place on the map is a goal; the map is usually built out of curiosity
Flexibility	Routes are rather rigid; they are rendered useless by any damage, or by the loss of a guidance, or direction	Maps are extremely flexible, and relatively invulnerable to noise and damage
Speed	Very fast	Relatively slow
Information content	Relatively little; each route contains only a small amount of data	Maps are one of the most efficient information storage devices known, with very large capacity
Access	No specialized knowledge is required for access; no coding strategies	Special knowledge of coding strategies required
Manipulation	None	Maps can be compared; places on maps can be compared

2.3. The psychological basis of cognitive maps

In the previous section we discussed two basically different ways of moving from one part of the environment to another involving the use of maps or routes. Maps were viewed as sets of connected places which provided the traveller with a large choice of possible paths between any two points in the environment. Because they do not rely on particular cues or behaviours maps were seen as flexible and relatively resistant to the effects of environmental change. In contrast, routes were best described as lists of guidances and orientations, the guidances acting as landmarks to be approached or followed by any available behaviour and the orientations specifying a particular movement to be made in the presence of a particular cue. Routes, unlike maps, were seen as inflexibly leading from one point to another and easily disrupted by alterations of relevant cues.

In this section we shall translate these theoretically derived notions into psychological mechanisms which could explain the types of spatial behaviour we described at the start of this chapter. The end point of the chapter will be the assertion that the hippocampus acts as a cognitive mapping system, which we shall call the *locale* system and which generates place hypotheses and exploration. Loss of this system forces an animal to rely on the remaining extra-hippocampal systems. In addition to discussing the properties of the mapping system, therefore, it will also be necessary to sketch in enough of the properties of the route, or *taxon,* systems to enable us to predict the behaviour of animals deprived of the hippocampus. The reader is warned, however, that our main emphasis is on the hippocampal locale system and that our treatment of the taxon systems may suffer from this perspective. For instance, we shall not attempt a detailed differentiation of guides and orientations, though it is likely that important differences exist between the two. Our main emphasis will be on the difference in the way that the locale system and the taxon systems store information. This difference, when combined with some simple assumptions about changes in synapses with use, will enable us to predict differences between the two systems which will be reflected in the place and route hypotheses generated by them and ultimately in the animal's behaviour based on these hypotheses.

Temporal changes after synaptic activation. We will assume that in both locale and taxon systems there are synapses which change with activation. When such a synapse is activated it undergoes both short-term and long-term changes in potency. These changes will differ in different synapses and will depend to some extent on the 'success' of the hypothesis based on the brain areas containing those synapses (see below). A hypothetical set of changes might be as follows: after activation of a synapse, the presynaptic element has a decreased efficacy for some time, perhaps due to transmitter depletion. With time, this decreased efficacy is reversed and the presynaptic element returns to normal potency or even slightly increased potency. In contrast the post-synaptic element is left more excitable after activation. This change also decays with time but may not reach the previous baseline or may even cross it to leave a long-term raised threshold for activation. Depending upon the relative time courses of these two changes, attempts to reactivate the synapse at different times subsequent to activation might find it hyper- or hypo-excitable. Furthermore, different afferents to the same postsynaptic cell might have different access to it. For example, shortly after activation by one pathway the cell might be poorly activated by another input on the same pathway but hyper-responsive to an input on a different pathway. We will discuss the evidence for these sorts of changes in greater detail when we discuss habitation in the taxon system (see pp. 244-7). Our main point here is that hypotheses will differ in the degree to which they show temporal changes in efficacy after activation in so far as they rely on the same synapses each time they are activated. As we shall see, this only applies to *taxon* hypotheses.

Reinforcement. Within our theory, reinforcement acts at two different levels. At the level at which different hypotheses compete for control of the output systems, reinforcement serves to maintain the ascendancy of the currently dominant hypothesis. Non-reinforcement by contrast, results in a tendency to switch away from the dominant hypothesis. At this level, then, reinforcement affects all hypotheses equally. Reinforcement also acts within each hypothesis system, and it is here that its action differs from system to system. Within the locale system, the role of reinforcement appears to be purely informational. Biological rewards are encoded just as any other stimuli would be; they are represented as occupying particular places in specific environments. This representation is encoded during exploration and is unaffected by the motivational state of the organism at that time (latent learning).* If the animal is subsequently motivated, this information can be read off the map and used in the form of a place hypothesis to obtain reward.

Within the taxon system, reinforcements might serve to strengthen the recently active synapses or to counteract the normal reduction in efficacy discussed above.

2.3.1. HYPOTHESIS BEHAVIOUR

In some learning situations the change in an animal's behaviour reflecting learning occurs slowly over trials, while in others there is an abrupt jump in the learning curve from near-chance performance to near-perfect performance. Typical of the former are such things as lever pressing or wheel turning, while the latter is seen in choice situations such as a simple T-maze or Y-maze (cf. Mackintosh 1974, pp. 147-50). Thus, the choice of the correct alley in a T-maze often goes from 50 to 100 per cent abruptly, while lever-pressing rates increase in a smooth, negatively accelerated fashion. It is reasonable to suppose that in the lever-pressing situation the neural changes underlying learning are incremental, while those in the maze are discrete and non-incremental. These discrete processes could involve the switch from one *hypothesis,* or strategy, of behaviour to another. 'Learning' occurs abruptly when the switch is from an incorrect to a correct hypothesis. The early work of Hamilton (1911) and Lashley (1929), and the subsequent, more elaborate, studies of Krechevsky (1932, 1933) clearly demonstrated such hypothesis behaviour. Krechevsky (1932) found, for example, that in a linear maze with four successive choice points a rat might choose to go through the left door at all four points on one trial, through the darkest doors on the next, then alternate right and left doors, and so on. Hypotheses can also be maintained across a number of trials. In a T-maze brightness discrimination most rats will choose either the left or right arm for a number of trials, and only then the darker or brighter

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* High levels of motivation will compete with, and curtail, exploration, leading to meagre maps, but will not affect the nature of the information stores in these maps.

arm. Careful testing in this situation shows that some of these 'position hypothesis' choices involve the use of places and others the use of body turns, or orientations. Thus, if one rotates the starting arm 180° one puts into opposition a place and an orientation hypothesis. If the animal returns to the same arm it is using a place hypothesis, because it had to make a different body turn to get there. In addition to simple orientation (body-turn), guidance, and place hypotheses, animals occasionally alternate left and right turns.

In the early, pre-solution stages of learning animals typically switch from one hypothesis to another with regard to its form; that is, they might switch from an orientation to a guidance, rather than to another orientation. Switching within modes, as stressed by Sutherland and Mackintosh (1971), occurs only after considerable learning, typically in reversal situations (see pp. 281-2) where one particular hypothesis has been consistently associated with reward. As we shall see later, this can provoke the so-called 'overlearning reversal effect' in which animals given added training show an increased ability to switch behaviours when the reward contingencies are changed; that is, when reward is switched from black to white in a two-choice brightness discrimination, for example. The fact that this effect is not usually seen in tasks requiring a position hypothesis (either place or orientation) is important, and will be discussed at length later (pp. 281-2).

As Restle (1957) has pointed out, the probability of a particular hypothesis being chosen depends on the lay-out of the experimental situation.* The availability of extra-maze cues, together with a large number of choices, favours the use of place hypotheses; a strong cue biases the animal towards guidance learning, while a paucity of cues coupled with a minimal choice of behaviours favours orientation hypotheses. In an environment which does not provide enough structure to activate strongly any hypothesis (e.g. uniform, closed, multi-choice mazes) all hypotheses will be weak and the animal might choose semi-randomly from trial to trial, approximating the type of behaviour described by Hull (1943). There are many factors which influence the selection of a hypothesis, such as rearing conditions, personality, previous experiences, inter-trial intervals, and so on. Some of these will be discussed at greater length when we describe the properties of the locale and taxon systems in detail below. In general, though, too little is known about the factors controlling selection. To use contemporary terminology, one would like to know the precise conditions under which one hypothesis *overshadows*, or dominates, others; this should prove a fruitful area for research. Though we cannot specify precisely the factors controlling hypothesis selection, there is a lot that can be said about the properties of the different hypotheses.

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* We do not accept Restle's suggestion that place learning is equivalent to distal cue learning (see pp. 73-4)

2.3.1(a). PLACE HYPOTHESES

The notion of place. Place hypotheses are based on information contained in the hippocampal cognitive-mapping system. In the next section we shall provide some speculations concerning the anatomical and physiological bases of this system. There, we shall define a place in an environment in terms of the activation of a specific array of hippocampal neurones. Here it will be more useful to speak of a *place representation;* this can be taken as a part of a cognitive map, while conversely a map can be viewed as a set of ordered, connected places. Such a place representation can be activated in either of two ways: (1) externally, by the simultaneous occurrence of two or more sensory inputs with the appropriate spatial co-ordinates in egocentric space; (2) internally, by an input from another place representation coupled with a signal from the motor system concerning the magnitude and orientation of a movement. Let us consider these in turn.

The first mode of activation involves a particular arrangement of external cues. When an animal is in a particular place the cues it perceives will have a unique spatial relationship with one another. This set has two important properties. First, no individual cue is *necessary* to the relationships among the rest of the set; places are *not* specified by complexes of cues in any simple sense. Any cue, or group of cues, can be removed from the total array without preventing the remainder (so long as at least two or three remain) from uniquely specifying, by their relations, a particular place. This property lies at the root of the difference between cue learning and place learning. Second, the spatial relationships among a set of cues remains constant when the animal stays in place but engages in rotational movements. Thus, when an animal sits in a place there is a unique spatial arrangement of the various stimuli available to it, and this arrangement is independent of the animal's specific orientation in that place. If the animal rotates its eyes or head the angular relations between these cues remains invariant. As Eriksson (1974) pointed out, sense data is sufficient to specify the relative spatial arrangements of things in the environment (see p. 48).*

When the animal moves, however, the problem of identifying a place becomes more complex. Here, not only is there subjective movement to be taken into account, but the sensed spatial angles between the objects in the environment change. As we have seen, the input from motor feedback processes enables the organism to map these objects accurately even though their angles are changing. Thus, some interaction between sensory data

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* Eriksson also pointed out that in order to obtain information about absolute distances some further information was needed. In the same way, though the relative arrangement of cues remains invariant through rotational movements, the position of the objects in question changes in terms of the egocentric space of the organism. Though the angles are the same, they are falling in a different part of the field. In order to reach out and grab one of these objects after a rotational movement the organism does require input from the systems which locate it in egocentric space.

and movement feedback is required to identify the procession of places as an animal moves through its environment. The vital importance of active exploration in the construction of maps, as we shall see shortly, rests on this observation.

The alternate mode of activating a place representation is an internal one. Here, inputs from other place representations are coupled with those from an internal 'dead-reckoning' system; this latter calculates the expected translation/rotation in space concomitant with a potential movement. During the building of a map motor feedback information is crucial in assigning representations of things to their appropriate 'place' in locale space. When an animal is in a familiar environment the activation of the system, without real movement, will enable the animal to predict what should appear in what place.

The cognitive-mapping system is assumed to contain a map for each environment the organism has experienced. These maps are built up in the following way: The animal brings to a new situation a *tabula rasa* of potential place representations. One of these is chosen to represent a specific location in that environment; this automatically determines the way in which the remainder of the locations in the environment will be represented. As the animal moves, the 'internal navigation' system will shift the focus of excitation within the map to other place representations appropriate to other locations. This is done, as we have suggested, on the basis of information about distances generated from both sensory and motor inputs. In order for the distances to be 'correct' in both the internal sense (that is, purely between the locations) and in the external sense (that is, between the framework of the observer and the locations) both these sources of input must contribute. There need be no representation within the map itself of the behaviours which moved the organism through the environment and which were instrumental in constructing the map.

According to the theory, exploration is behaviour designed initially to build and subsequently to update cognitive maps. When there is a mismatch between some sensory input to a place representation and the predicted sensory input, a set of *misplace* detectors is triggered, the output of which activates and directs the motor systems involved in exploration. This behaviour is directed towards the incongruence and new information can be incorporated into the map as a result of it. When the animal first enters a novel situation all the misplace detectors will be activated and exploration will continue until sufficient information is incorporated into the map of that environment. Thus, our theory accords curiosity the status of a major motivation, the driving of information incorporation into cognitive maps.

Properties of place learning. Given that an animal has built a map of an environment, it can subsequently use this information in its attempts to solve a wide variety of problems. Safety or danger can be attributed to environments, or places within an environment; approach or avoidance responses can be directed towards, or away from, places; places can be reached from any other place, by any available behaviours, even when landmarks are unavailable; new paths can be generated when old ones are closed, as in detour behaviour. Thus, the behaviour of Tolman's rats in his complex mazes is attributable to the workings of the cognitive map. The map is responsible for an animal's sensitivity to novelty in its environment, but at the same time it enables the animal to behave (in the molar sense of the term) in a consistent fashion in spite of occasional changes in the environment since the alteration of any particular feature of the environment does not destroy the usefulness of the map in identifying places.

On the other hand, the mapping system *is* sensitive to constant variability in the environment; such variability makes it difficult, if not impossible, to build a useful map. The novelty-seeking properties of the mapping system, so important to its function, have the disadvantage of pre-empting the output systems whenever novelty occurs.* Thus, in a continuously changing environment the mapping system becomes useless as a device for problem solving. In practice this means that such variability could block the locale system from directing behaviour and bias the organism towards other hypotheses. We shall suggest later that this effect of environmental variability is partly responsible for the marked persistence often seen in the behaviour of animals in such situations; the taxon hypotheses they are forced to use are inherently persistent, as we shall see shortly.

Unlike the extra-hippocampal systems the locale system is relatively free from the effects of time and repetition. Whenever an organism attends to an object it is encoded in the map. Subsequent attention to that object will have no further effect on the representation of that object in the map. In fact, by virtue of the workings of the misplace system the locale system will act in such a way as to direct the animal's attention away from objects whose presence it can predict towards those whose presence was unexpected. In this sense it is, as we noted, a novelty-seeking device. Incorporation of information about stimuli occurs in a non-incremental fashion. The map itself can become richer and more distinct (i.e., there is better and finer differentiation of places) but it is not altered in any fundamental sense with repeated exposures to the same environment. Since each representation of a stimulus is encoded in terms of its spatial relations to other stimuli, an identical stimulus occurring in different parts of the same environment, or in totally different environments, will have distinct, and differentiable, representations in each case. This, of course, is the way in which a spatio-temporal framework solves, by its very structure, the problem of re-identification so central to our philosophical discussion.

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* We shall suggest later that one of the possible advantages in the delayed, or postnatal, maturation of the hippocampus (pp. 112-16) lies in the fact that exploration could be quite maladaptive in infants.

This structure guarantees that there will be relatively little interference between the activations of traces representing the same stimulus in different contexts. On the other hand, some interference could arise within the mapping system between maps of an environment formed before and after some slight change. The up-dating of maps, consequent to such change, does not imply that the old map is literally erased. Some representation of every experienced state of the environment must be maintained, along with information as to which representation is current and which is no longer so.

In most situations place learning is unaffected by inter-trial intervals; this is not the case with route learning. In general, the insensitivity of the locale system to time factors can be attributed to the fact that a place representation can be activated by any of several *different* sensory inputs and that place hypotheses can be effected by any of several *different* motor programmes. *Thus neither the synapses on the input side nor on the output side need be re-used with successive activations of a place hypothesis.* Consequently the strength of a place hypothesis does not suffer from the temporal after-effects which are characteristic of taxon hypotheses. As we shall see shortly, guidance hypotheses involve the consistent reactivation of the input or sensory component while orientation hypotheses require the continual use of the same synapses in the output or motor side. Activation of a route then becomes dependent upon the momentary state of its various links and this state will depend on how recently it has been used. We shall discuss all this at greater length in the next section.

2.3.2. THE TAXON SYSTEMS

The taxon systems generate routes, which can be viewed as lists of guidance and orientation hypotheses.* Both of these rely on egocentric spatial systems. In a guidance hypothesis a positive or negative valence is attached to a specific cue, or item, and this can be approached or avoided by any available behaviour. In this mode the motor system acts as a goal-directed device, reducing the distance between the organism and the desired item, or, conversely, increasing the distance between the organism and its nemesis. In orientations the emphasis is on the specific form of behaviour which is to be executed in the presence of a cue. This type of hypothesis usually involves behaviours which include a change in orientation of the body axis relative to some aspect of the external world (e.g. a right turn at the corner).

In this context it is of no major importance whether the hypotheses within a particular route list are *unordered*, *ordered*, or *concatenated*. In an ordered list subsequent items can only be activated after the earlier ones. In a concatenated list there are actual links between the items such that the first facilitates the second, and so on. From our point of view the ordering of items in the chain may be due to factors such as the physical layout of the environment (e.g. the stimuli for response 2 are revealed by response 1) or the mapping system. In other words, many of the interpretations put on behaviours which seemed to involve interactions between the individual links in a chain (such as Hull's backwards chaining) can be seen as ways of avoiding the notion of a mapping system. Once the latter is accepted, these 'explanations' become both laborious and unnecessary.

2.3.2(a). Some possible principles underlying taxon hypotheses. In the absence of a body of pertinent data we must limit ourselves to a few tentative statements about the properties of guidances and orientations, without attempting to differentiate strongly between them. Our basic assumptions concerning the properties of taxon hypotheses rest upon the best guesses we can make concerning the physiological impact of activation within the taxon systems. It must be stressed that the 'taxon systems' embrace the vast majority of the central nervous system, including both sensory and motor systems, and the generalizations put forward here can scarcely be expected to apply at all levels of this set of systems. The force of this caution will be made clear in what follows.

The activation of a taxon hypothesis, or a representation within the taxon systems, can result in any of several independent physiological processes with different time courses, including potentiation and depression of both short-term and long-term significance. The complexity and multiplicity of these phenomena will be conveyed by a consideration of the following facts, all of which we would attribute to the action of the taxon systems:

(1) The activation of a representation in the taxon system elicits, immediately, a reduction in the threshold for re-activation of the same representation. For instance, with a brief inter-stimulus interval (ISI) the elicitation of a reflex can undergo sensitization rather than habituation (e.g. Pearson and Wenkstern 1971, Szabo and Kolta 1967); the ISI in these studies was 10 and 15s, respectively. Similarly, the perception of a word, or any item, in a tachistoscopic presentation temporarily lowers the threshold for perception of the item, the so-called *priming effect* (e.g. Neisser 1954). Finally, perception of one version of an ambiguous figure transiently biases the observer towards perceiving the same version again (Leeper 1935).

(2) The continued activation of a taxon representation, or several successive activations (with some ISI longer than 10-15 s) usually increases

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* We are using the terms guidance and orientation hypotheses to refer to what have traditionally been termed *cue* and *response* hypotheses. Since there are cues and responses involved in both forms of hypothesis, the traditional nomenclature is misleading.

the threshold for subsequent re-activation. For instance, constant perception of one version of the ambiguous figure, such as a Necker cube, leads to a spontaneous reversal of perception of the other version. Similarly, repetition of any action will lead to its eventual habituation. Thus, there are short-term effects resulting in either potentiation or depression, depending perhaps on the rate of activation.

(3) The accumulation of activations of any representation over a long period will lead to a more or less permanent reduction in the threshold for its activation. An example of this is seen in the easier recognition of familiar *versus* unfamiliar items in tachistoscopic tests; another example is seen in the ease with which well-learned habits are elicited.

(4) The repetition of any taxon habit *can* lead to the build-up of inhibition; this occurs readily when reinforcement is absent, but can be seen even when it is present (Kendrick 1958).

This perplexing picture arises, as we have noted, partly because we are considering within the same context the action of a variety of systems from the periphery to the depths of the central nervous system. These undoubtedly have potentiation and depression processes with quite different decay rates, such that the net outcome of the interaction of several of these systems could be just about anything, depending upon the consistency of input (cf. Kimble and Ray 1965) and the interval between activations. This latter point is worth stressing; in contrast to the locale system, the taxon systems are particularly sensitive to temporal factors. The temporal effects most prevalent in animals, and which we shall stress here, are those of short-term depression and long-term potentiation. Later, in discussing humans, we shall amplify our discussion of short-term potentiation effects.

2.3.2(b) Some properties of taxon hypotheses. The reader should bear in mind that one of the major premises of the present theory is that the behaviour of animals bereft of their hippocampus (and locale system) is largely determined by the properties of the remainder of the brain (and its taxon systems). Thus, the mode of action of the taxon systems is part of what one sees after hippocampal lesions and becomes important in any understanding of the effects of such lesions.

Because of the possibility of activation-produced depression in taxon hypotheses, they are more sensitive to the rate at which they are activated than are place hypotheses. At short intervals there will be a rapid buildup of depression, leading eventually to a blockage of the taxon hypothesis. Thus, as the inter-trial interval in a given situation shortens, there will be a bias towards the use of place hypotheses. Further, in the absence of other mechanisms inhibiting behaviour only the rapid repetition of the same taxon hypothesis will enable the animal to cease using an inappropriate hypothesis when conditions have suddenly changed, as in reversal and extinction (see pp. 281-4 and pp. 337-48). This principle is central to an understanding of the abnormal persistence often seen in animals totally dependent upon taxon hypotheses and the concomitant mechanisms by which they can be inhibited when necessary. While similar principles of potentiation and inhibition might be working in the locale system's short-term reaction to activation, they would not lead to persistence in the same fashion as seen in the taxon systems, owing to the multiple channels available for activation of any representation. Thus, the persistence associated with taxon hypotheses is correlated with their inflexibility.

As we noted, representations in the taxon systems should, in the long term, be strengthened with repeated activation; such an effect does not occur within the locale system. Thus, all things being equal, repeated exposure to a situation in which both place and taxon hypotheses are appropriate will see a shift in favour of the taxon hypothesis with continued training (assuming the inter-trial interval is not too brief). This is one way of conceptualizing the autonomy, and stereotypy, of habits after many repetitions. Here, it becomes important to note a difference between guidance and orientation hypotheses. The former involve *any* response directed at a particular cue, and thus need not involve the repetitive activation of the same output channel. Different movements might be involved on repeated approaches to, or avoidances of, a given cue. On the other hand, orientation hypotheses, by definition, involve similar or identical responses, even if these are defined at the micro-level. Because of this such hypotheses could be particularly prone to long-term potentiation effects, leading to marked stereotypy in their execution. Some such process is probably involved in *autonomous habits,* which are remarkably persistent and progressively less dependent upon any particular eliciting cue. Thus, there are differences in the long-term effects of repetition on these two types of taxon hypotheses. With a guidance hypothesis a variety of responses can be used to act persistently relative to a given cue; with orientation hypotheses stereotyped responses are emitted with little regard to the cues offered by the environment.

The mechanisms preventing serious interference effects in the place system are lacking in the taxon systems. Representations in the latter do not incorporate the (spatial) context which could differentiate one presentation of a guide from another. Consequently, there can be considerable confusion, expressed as interference between behaviours appropriate in different contexts. The best way of avoiding this kind of confusion is through the provision of cues which specify the appropriate taxon hypothesis, thus acting as substitutes for the context normally provided by the locale system. This interference effect, based on the lack of information defining the appropriate interpretation of a guide, might be less prevalent with orientations.

The general absence of context information characterizes the memory storage properties of the taxon systems. Concepts and categories, the look, the feel and the sound of things, the goodness or badness of objects: all these are represented within the taxon systems. What is missing is the spatio-temporal context within which this knowledge was acquired; this is provided by the locale system, where representations from the taxon systems are located within a structure providing such context. Behaviour which can proceed without contextual information, and there is much that belongs to this class, will not require more than intact taxon systems. However, such behaviour will be subject to the principles of those systems; the meaning of this will be made clear when we analyse the effects of hippocampal lesions upon behaviour.

2.3.3. CONCLUSIONS

In this chapter we have discussed the evidence for cognitive-mapping behaviour and its translation into place-learning mechanisms. Cognitive maps were contrasted with routes, and the types of behavioural strategies dependent upon the two were derived from the properties of maps and routes, as defined by geography on the one hand and physiology on the other.

Our thesis is that place hypotheses are dependent upon the hippocampal cognitive-mapping system, and that guidance and orientation hypotheses do not require the hippocampus. The primary effect of dysfunction in the locale system should be the loss of the two functions dependent upon cognitive maps: exploration and place learning. However, the radically different properties of the locale and taxon systems confer quite different characteristics on behaviour based on these. Place hypotheses allow for flexibility, rapid change, and the retrieval of context-specific information; taxon hypotheses lack all of these properties. Some of the differences between the properties of the two systems are summarized in Table 2. Before moving to an analysis of the effects of hippocampal lesions, where these principles are brought out most clearly, we shall discuss the way in which the anatomical structure and physiological functioning of the hippocampus can be understood in terms of the cognitive-mapping system described above.

TABLE 2

Properties of the taxon and locale systems

	Taxon	Locale
Motivation for learning	Biological need: to obtain reward or avoid punishment	Cognitive curiosity: to construct and update a map of the enviroment
Learning change	Incremental or decremental	All-or-none
Persistence	High esp. orientation hypotheses	Low
Temporal changes after activation	Marked changes in threshold and strength as a function of time after activation: sensitive to intertrial interval	Minimal changes with time after activation; insensitive to intertrial interval
Interference between similar items	High	Low

3

Anatomy

3.1. Introduction

IN the following two chapters we shall review recent anatomical and physiological studies of the hippocampus and show that structurally and functionally it is well suited to act as a cognitive map. As we described earlier, the map system is assumed to have two basic components: a mapping space and a locative mechanism for building and changing maps. The mapping space consists of a set of interconnected places, each place being receptive to a large number of potential input items. The locative system selects the appropriate place for any particular input and effects changes in the representation of items in response to environmental change. In keeping with the arguments of the previous section, this locative system derives its instructions, at least in part, from the movement-programming system.

Anatomically, the mapping space could be provided by a large matrix of identical neurones structured in such a way that each neurone (or group of neurones) would represent a place in a given environment. At least two inputs to this matrix would be required: (1) an input providing sensory information about the environment; (2) an input providing information that the animal is changing its position in space or changing its receptor surfaces so as to sample inputs from a different part of space. In this chapter we shall discuss the nature of hippocampal anatomy in light of these requirements and shall try to identify (1) the mapping space with the matrix of pyramidal cells in the hippocampus, (2) the input about environmental items with the various pathways from the cortical taxon stores via the entorhinal area, and (3) the locative input with the brain stem-septal-hippocampal pathway.†

The mapping system will need several outputs. Since it subserves place hypotheses which can guide an animal's behaviour, it should project to areas capable of driving behaviour. Similarly, the mismatch system, which halts ongoing behaviours and drives exploration, will have connections (1) to the motor areas generating this pattern and (2) to that part of the hypothalamus driving pituitary output in response to environmental uncertainty.

3.2 The internal structure of the hippocampus

Removal of the posterior and temporal neocortex of an animal such as the rat reveals the large sausage-shaped hippocampus underneath (see Fig. 3).

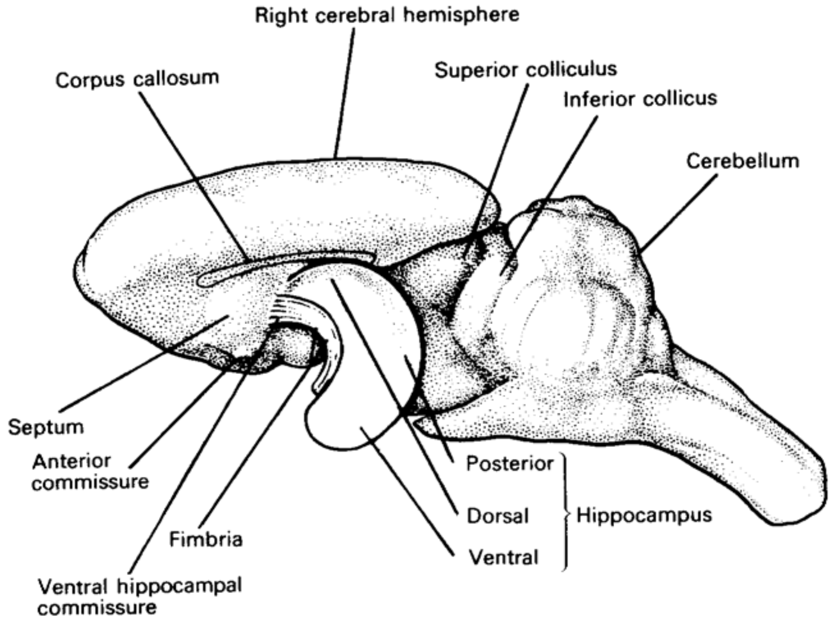

FIG 3. Drawing of the left rat hippocampus. All other forebrain structures except those at the mid-line have been removed.

In this animal the hippocampus occupies a large portion of the forebrain. For descriptive purposes it can be divided into a dorsal portion lying just behind the septum, a posterior portion where it begins to bend ventrally and laterally, and a ventral portion lying in the temporal part of the brain. The part of the hippocampus visible on its dorsal aspect is the hippocampus proper, while the fascia dentata is buried inside and on the bottom surface of the sausage. The fimbria is a large fibre tract which is visible on the lateral edge of the exposed hippocampus. The dorsal fornix which runs close to the mid-line beneath the corpus callosum is not shown in Fig. 3. Before we discuss these various features of hippocampal anatomy in greater detail, it will be useful to look briefly at the phylogenetic differences in hippocampal structure.

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†We shall concentrate our discussion on the hippocampus as the core of the cognitive map. It is clear, however, that this core is supported by several ancillary structures whose functions can be characterized as organizing and channelling the inputs and outputs of the hippocampus. These structures, comprising the medial and lateral septum rostral to the hippocampus and the subicular area caudal to it, have important roles in the functioning of the cognitive map. Though considerable knowledge exists concerning the septal region, detailed investigation of the subiculum has only recently begun. Where pertinent to the discussion we shall fill in the details of these ancillary structures.

3.2.1. COMPARATIVE HIPPOCAMPAL ANATOMY

Recent developments in comparative anatomy suggest that it is no longer reasonable to expect strict homologies between brain structures in animals occupying different branches of the phylogenetic tree. There is considerable evolution within each phylum, so that one can find sophisticated neural organizations as well as primitive ones in that phylum. For example, in addition to the well-described sharks with simple brains one can find sharks with well-developed and highly differentiated forebrains. Similarly, it is now clear that the comparative anatomy of particular brain areas such as the hippocampus is more complicated than the classical linear evolutionary view suggested. While comparative anatomists have traditionally identified a homologue of the mammalian hippocampus in all vertebrates, including the most primitive, the cyclostome (e.g. hagfish and lampreys: Ariens Kappers, Huber, and Crosby 1936, pp. 1248-1255, Heier 1948, Crosby, Dejong, and Schneider 1966), there are several points which must be kept in mind:

(1) Homologies can be sought in terms of either structural or functional similarity, and these might not necessarily be the same.

(2) The same neural elements could be used differently to produce a module with an entirely different function. Thus, pyramidal cells used in one way might serve to construct a cognitive map in one species, while used in a slightly different way could serve a different function in another species. The grid-like structure of the hippocampus could be used as a spatial map in an animal such as the rat, or as an olfactory correlation device in the reptile, or as a semantic map in the human.

(3) The same function could be performed in different species using quite different structural modules.

These questions about homologies can only be adequately tested through the use of behavioural experiments where the same form of behaviour is tested in several species, in ways appropriate to each species, and through single-cell neurophysiological experiments in freely moving animals; neither of these has been provided in sufficient detail as yet.

We think it is reasonable to assume that behaviours such as homing, migration, and territoriality are evidence of cognitive mapping and suggest as a working hypothesis that species demonstrating these behaviours have a homologue to the mammalian hippocampus, usually situated in the medial wall of the pallium (see, e.g., Fig. 4(a)). In the reptile a strip of densely staining pyramidal-shaped cells lines the medial and dorso-medial surface of the hemisphere (the so-called archipallium or mediodorsal cortex, see Fig. 4(b)). Between the archipallium on the medial surface and the paleopallium, or pyriform area, on the far lateral surface lies the neopallium or dorsal cortex, the presumed homologue of the neocortex of higher animals. The massive expansion of this neocortex in mammals is associated with dramatic shifts in the location of the hippocampus and pyriform cortex.

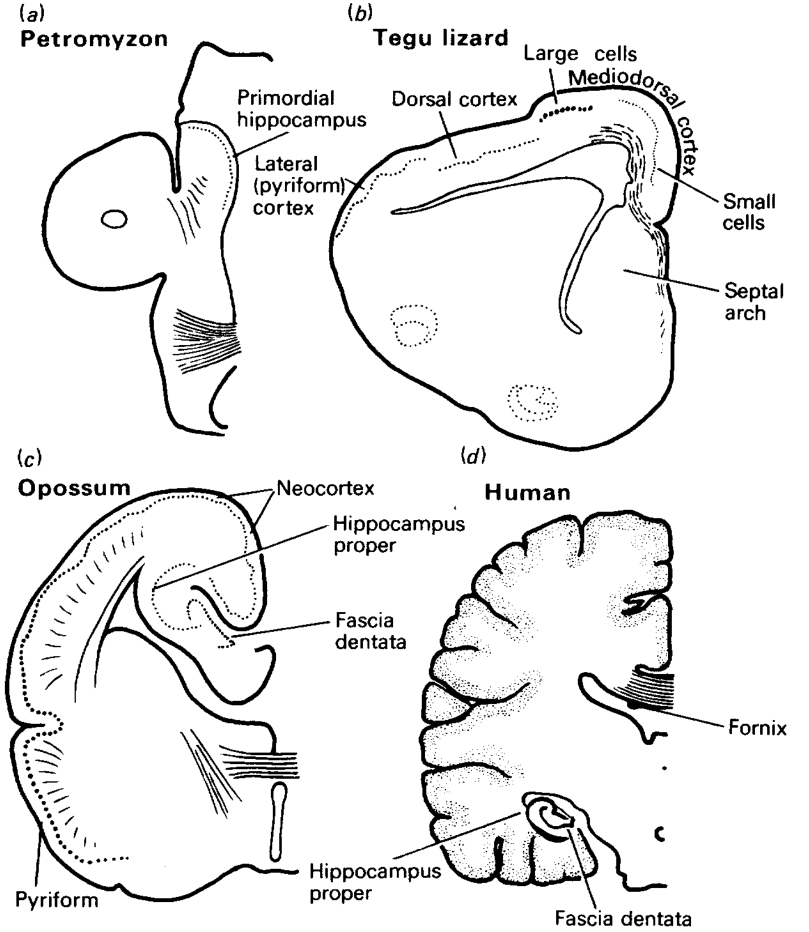

FIG. 4. Cross-sections through one hemisphere of the forebrains of different animals and man to show the size and location of the hippocampus (or hippocampal homologues) in relation to other neural areas: (a) Petromyzon (after Heier 1948, p. 32), (b) Tegu lizard after Lohman and Mentink 1972, p. 327), (c) opossum (after Loo 1931, p. 49), and (d) human. The cross-sections have been drawn roughly equal in size and are, therefore, not in scale relative to each other.

The reptilian hippocampus can be divided into a small-celled sector on the dorso-medial surface and a large-celled sector more dorsally located. While these have traditionally been considered to be the homologues of the fascia dentata and the hippocampus proper, respectively, this conclusion must be treated with caution in view of recent work on the connections of these two areas with each other and with the rest of the brain. For example, the small-celled sector, unlike the mammalian fascia dentata, projects not only to the large-celled sector but also directly to the septum and hypothalamus (Lohman and Mentink 1972, Lohman and Van Woerden-Verkley 1976). We shall discuss mammalian septo-hippocampal connections in greater detail later in this chapter. It is worth noting here that the sizes of the hippocampus and the septum are highly correlated among the insectivores and primates (Andy and Stephan 1966).

In acallosal mammals such as marsupials the hippocampus is folded over upon itself and protrudes into the lateral ventricles (Fig. 4(c)). The two cell layers of the hippocampus proper and the fascia dentata are bent into two intersecting U's. This infolded cross-section is characteristic of all higher animals. In the opossum the posterior tail of the hippocampus, along with the expanding neocortex, moves into a temporal position. With further development of the neocortex the rostral portion of the hippocampus is invaded by fibres of the corpus callosum and progressively decreases in size until, in humans, only a rudiment remains (Fig. 4(d)).

The absolute size of the hippocampus increases steadily with phylogenetic development. Stephan (1966) has shown that the hippocampus in man is twice as large as that seen in monkeys, where it, in turn, is somewhat more than twice as large as that seen in basal insectivores (e.g. shrews and hedgehogs). Similar conclusions can be drawn from a study of fibre numbers in the ventral hippocampal commissure. Andersen (1960c) reported a steady increase in the number of fibres as one goes from the rat (365,000), through the rabbit (625,700), to the cat (1,191,000). As we have seen, the sizes of hippocampus and septum are correlated, and a study of fibre numbers in the post-commissural fornix, an efferent path of the subiculum, indicates a similar increase in this ancillary system (Powell, Guillery, and Cowan 1957). There are five times as many fibres in this tract in man as there are in monkey, cat, and rabbit; these animals, in turn, have five times as many fibres as does the rat.

This vast increase in the size of the hippocampus is not uniform through its dorsal-ventral extent, nor with respect to its internal layering. As we have noted, that portion of the hippocampus situated most dorsally is pushed aside by the developing neocortex and corpus callosum, leaving only the hippocampal rudiment located dorsally. This sequence *does not* mean that the human hippocampus is analogous primarily to the ventral portions of, for instance, the rat hippocampus. In fact, the opposite is more likely the case. The dorsal hippocampus contains a greater proportion of the hippocampal subfield known as *regio superior* than of the subfield known as *regio inferior,* while the opposite is the case in the more ventral aspects of the hippocampus. Stephan (1975, p. 511) has noted that, compared with basal insectivores, there is a six-fold increase in regio superior with only a two-and-a-half-fold increase in regio inferior in man. We shall comment on the possible functional significance of these shifts later.

While increasing in absolute size, the hippocampus decreases in relative size with phylogeny. This is entirely due to the expansion of the neocortex, which is more than 200 times larger in humans than it is in the basal insectivores. Thus, the neocortex represents 80 per cent of the forebrain in humans, the hippocampus less than 1 per cent; in the basal insectivores the figures are 13 and 8.5 per cent, respectively (Stephan 1966).

3.2.2. INTERNAL STRUCTURE OF THE MAMMALIAN HIPPOCAMPUS

The hippocampus in mammals is the paradigm of simple cortex, consisting primarily of one basic cell type and its associated interneurones. These basic neurones are packed together in one layer of a three-layered structure, in contrast to the six layers of neocortex. The transition from the complex structure of entorhinal neocortex to hippocampal archicortex can be seen in a horizontal section through the posterior arch of the hippocampus (Fig. 5; facing p. 108). On the basis of cytoarchitectonics, Lorente de No (1934) divided the interposed *subicular area* into *parasubiculum*, *presubiculum*, *subiculum*, and *prosubiculum* (see Fig. 5). Little is yet known of the function of these four or five layered cortical areas, partly because of the numerous fibres of passage which are invariably interrupted by lesions, vitiating the use of traditional anatomical and behavioural studies. Fortunately, recent anatomical advances involving the use of techniques such as autoradiography and enzyme transport combined with histofluorescence have begun to reveal the connections of these, and other, areas in previously undreamed of detail. Further information about the architectonics of these areas can be obtained from Lorente de No (1934) and Blackstad (1956).

The hippocampus itself is divided into two major U-shaped interlocking sectors, the fascia dentata (*area dentata, dentate gyrus*) and the hippocampus proper (*cornu ammonis*). The fascia dentata has an internal or buried blade and an external or exposed blade (Fig. 6).† The hippocampus proper can be further divided. On the basis of differences in cell morphology and fibre projections Cajal (1911), and more recently Blackstad (1956), identify a *regio superior* and a *regio inferior*. The regio superior abuts on the prosubiculum and contains a double row of medium-sized

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† The nomenclature used to refer to the two blades of the fascia dentata is extremely confusing, differing from one author to the next. Relative spatial terms such as inner/outer, internal/external, and endal/ectal are potentially ambiguous since they identify different blades depending on whether the whole brain or the hippocampus is taken as the reference framework. On the other hand, terms such as medial/lateral and dorsal/ventral are only appropriate over limited parts of the structure. We have adopted a suggestion of P. Andersen (private conversation) and called the blade internal to the hippocampus the *buried blade* and the blade on the external surface of the hippocampus the *exposed blade* (see Fig. 6).

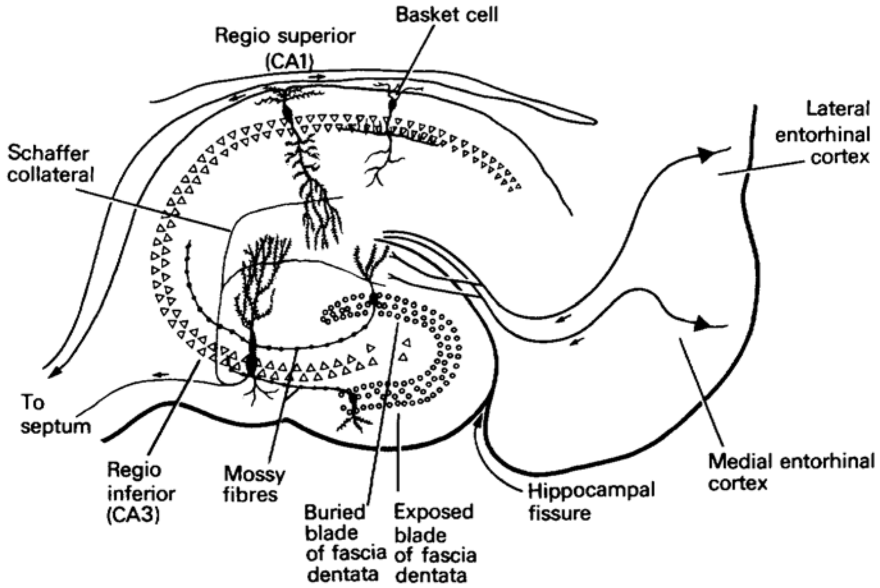

FIG. 6. Schematic diagram of the intra-hippocampal connections. Horizontal section through the right hippocampus as in Fig. 5 except that caudal is to the right.

pyramidal cells whose main apical dendrite gives off only small side branches and does not divide for several hundred microns (Fig. 6). The regio inferior is the semi-circle close to the fascia dentata containing the giant pyramidal cells whose apical dendrites bifurcate shortly after leaving the soma (Fig. 6). As can be seen in the figure the part of the dendrite close to the cell body has thorn-like spines, and these, as we shall see, receive contacts from the mossy fibres of the dentate granule cells. Lorente de No (1934) divided the hippocampus proper into four fields, CA1-4, CA standing for cornu ammonis. He called the regio superior CA1 and subdivided the regio inferior into CA2 and CA3;[†] CA4 designated the scattered cells inside the hilus of the fascia dentata. Although these cells are not lined up like the pyramidal cells of CA3, they were included in the hippocampus proper because they have pyramid-like characteristics, receive the mossy fibres,

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[†] CA1 is sometimes called Sommers sector in the pathological literature; it appears to be particularly susceptible to anoxia and/or vascular disturbance and is thus often selectively destroyed in diseases such as idiopathic epilepsy and various types of poisoning (Greenfield *et al.* 1961, Meldrum and Brierley 1972).

TABLE 3

Areas of the hippocampal formation

Doinikow (1908)	Brodmann (1909)	Rose (1926)	Lorente de No (1934)	Blackstad (I956)
Dentate fascia		Dentate fascia	Dentate fascia	Dentate fascia
End blade		h3, h4, h5	CA3c, CA4	Hilus of the dentate fascia
Transition blade		h2	CA2, CA3a, CA3b	Regio inferior (probably including CA1c of Lorente de No)
Lower blade		h1	CA1 Prosubiculum a,b,c, subiculum b	Regio superior Subiculum
	Area 27		Subiculum a and presubiculum	Presubiculum
	Area 29e			Area retrosplenialis e
	Area 49		Parasubiculum (including transition zone)	Parasubiculum
	Area 28a		Entorhinal fields B, C	Medial entorhinal (including transition zone)
	Area 28b		Entorhinal field A (including Lorente de No's area 35)	Lateral entorhinal
	Area 35		Unlabelled	Perirhinal area

From Chronister and White 1975, p. 21

and send their axons into the fimbria. However, unlike the CA3 pyramids, they do not receive an input from the *basket cells,* a group of interneurones we discuss shortly.

The CA2 field, according to Lorente de No, consisted of CA3-type pyramids which did not receive the mossy fibres of the dentate granule cells. The existence of a separate area, defined as such, has been challenged by Blackstad and his colleagues (e.g. Blackstad 1956, 1963), who can find no difference between CA2 and CA3 cells in terms of their connections or histo-chemical staining properties. Table 2 reproduces Chronister and White's (1975) comparison between Lorente de No's nomenclature and that of Rose and the now seldom used one of Doinikow. They have also added Blackstad and Brodmann to the table. Since both the nomenclature

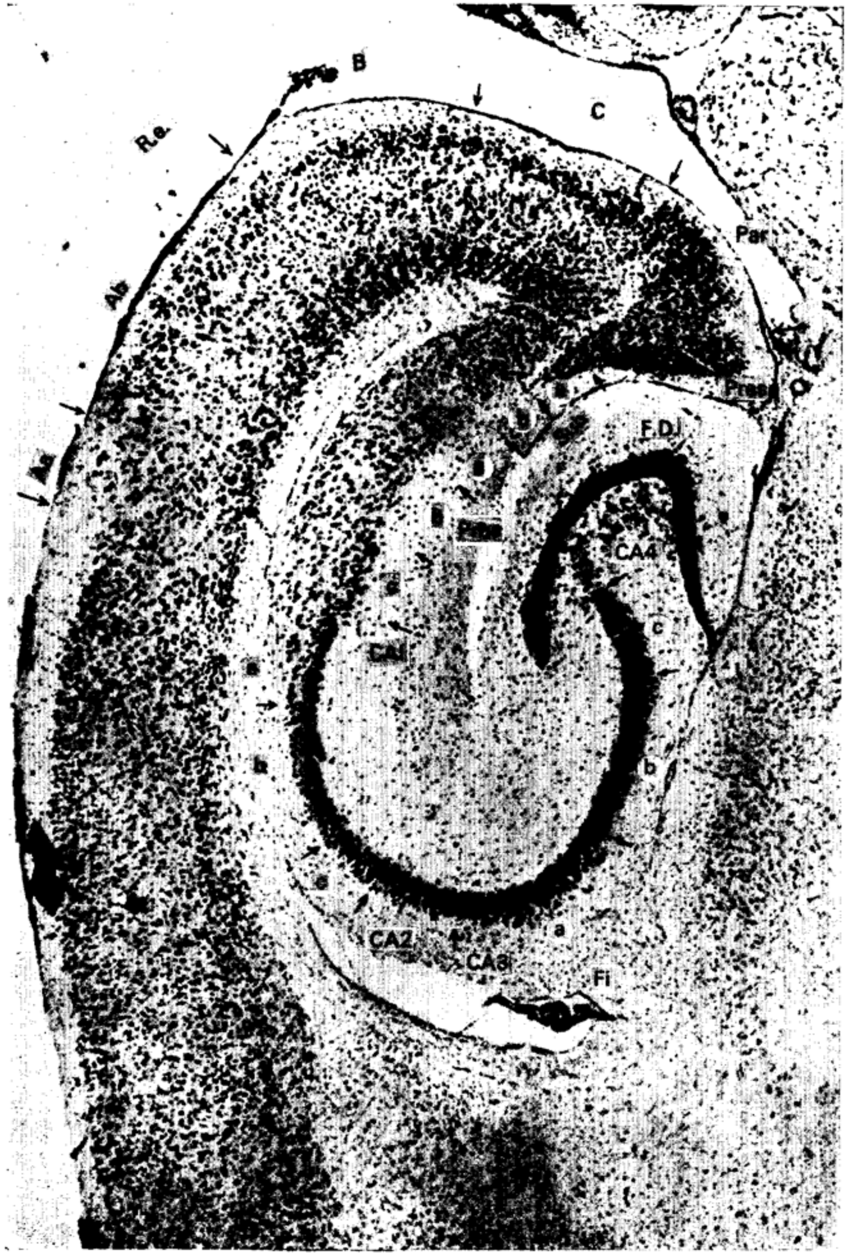

FIG. 5. Photograph of a horizontal section of the right side of the adult mouse brain as seen from above. The section passes through the posterior arch of the hippocampus and entorhinal cortex (R.e.). Caudal is up. Note the gradual transition from the six-layered entorhinal cortex through the parasubiculum (Par), presubiculum (Pres), subiculum (Sub). prosubiculum, (Pros.) to the three-layered cortex of the hippocampus proper (CA1-CA4) and the fascia dentata (F.D.). Lorente de No further subdivided some of these areas (a, b, c). Fi is the fimbria. Nissl stain. (Modified from Lorente de No 1934.)

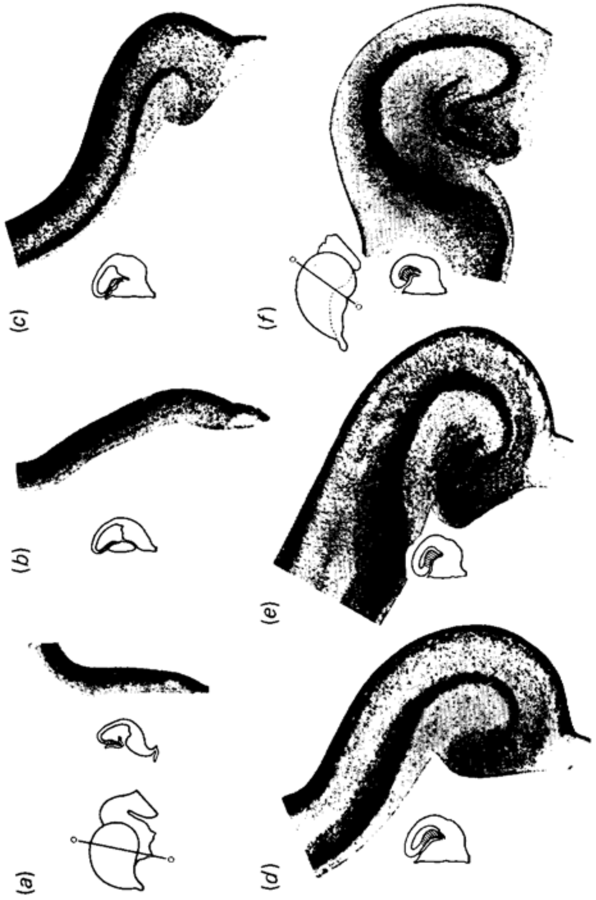

FIG. 8. Prenatal development of the rabbit hippocampus. Cross-sections through the hippocampus at embryonic stages (a) 15 mm, (b) 20 mm, (c) 29 mm, (d) 41 mm, (e) 60 mm, and (f) 90 mm. The insets show the outline of one hemisphere, and the shaded portion the position of the embryonic hippocampus. (Reconstructed from Stensaas 1967*a, b, c, d, e,* 1968.)

of Lorente de No and that of Cajal/Blackstad are used widely at present we shall refer to both, with a slight preference for Lorente de No.

With the exception of CA4, the basic pattern in all sectors of the hippocampus is the same: an ordered sheet of large neurones whose cell bodies are all packed together in one layer and whose dendrites all run off in the same direction, extending for many hundreds of microns. Many of the inputs to each sector traverse the dendrites at roughly right angles, making synapses *en passage* within a restricted region of the dendritic fields of many neurones in turn. The large neurones are the giant pyramids of CA3, the smaller pyramids of CA1, and the granule cells of the fascia dentata. Examples of these are seen in Fig. 7. As shown in this figure the pyramidal cells have both *apical* and *basal dendrites*, while the granule cells have only apical dendrites.

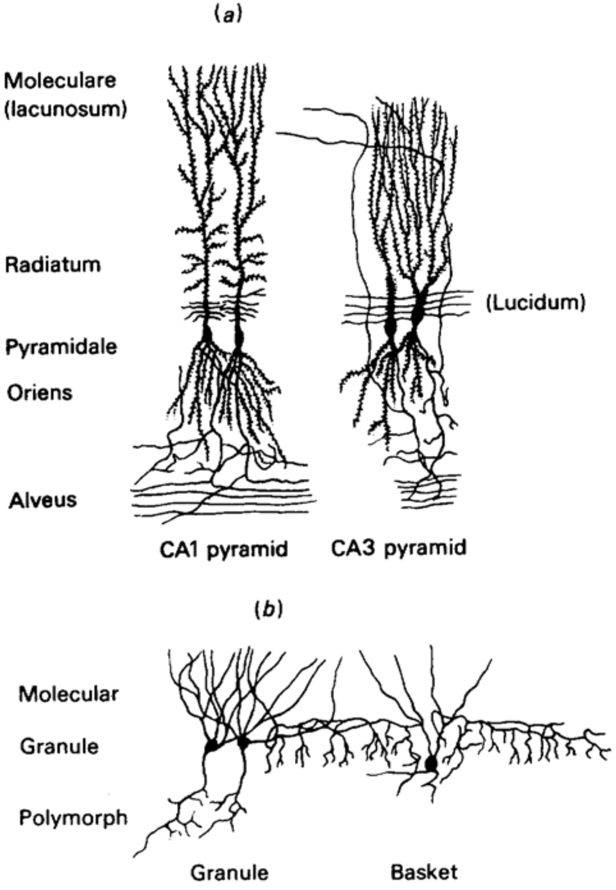

FIG.7(a). Examples of CA1 and CA3 pyramidal cells. (After Cajal 1911, Fig. 475).(b) Examples of dentate granule cells and a basket cell of Cajal. (After Lorente de No 1934, Fig. 10.)

An example of the most important type of interneurone in the hippocampus, the basket cell of Cajal, is also shown in Figs. 6 and 7. Its cell body is situated either in, or slightly below, the principal cell layer and its axon ascends through the cell-body layer, travelling a considerable distance. The axon moves orthogonal to the dendrites of the pyramidal or granule cells, giving off numerous descending collaterals which end in basket-like plexuses around the cell bodies. The basket cells differ from the pyramidal and granule cells in a number of ways, and this is seen most clearly in CA3. In contrast to the pyramids, the basket cells do not have the characteristic thorns on their apical dendrites and thus are not in contact with the mossy fibre input. Further, the basket cells do not send their axons out of this area, while the pyramidal cells do. Other interneurones (e.g. the stellate and fusiform cells of Cajal) send their axons into the apical dendritic layer to make contact with the distal dendrites of the pyramids and granules. As we shall see in a subsequent section, there is physiological evidence that at least some of the interneurones exert a widespread inhibitory control over the pyramidal and granule cells.

3.2.3. INTERNAL LAYERING

3.2.3(a). Fascia dentata. The fascia dentata has three layers (see Fig. 7): (1) the *granule* layer containing the densely packed cell bodies of the granule cells; (2) the *molecular* layer formed by the intertwining apical dendrites of the granule cells and their afferents; (3) the *polymorph* layer in the hilus of the fascia dentata which merges with the CA4 field and contains the initial segments of the granule-cell axons as they gather together to form the mossy fibre bundle. It also contains a few types of non-granule cells, the most important of which is the basket cell discussed above.

3.2.3(b). Hippocampus proper. Although it is basically a three-layered structure like the fascia dentata, the hippocampus proper has been divided into as many as seven layers, each defined by a particular feature of the large pyramidal cells or their afferents (see Fig. 7). Starting from the ventricular surface these layers are as follows: (1) The *alveus*, containing the axons of the pyramidal cells, directed towards the fimbria or the subiculum (some afferents to the hippocampus also travel in the alveus); (2) the *stratum oriens*, a layer between the alveus and the pyramidal cell bodies which contains the basal dendrites of the pyramidal cells and some of the basket cells, as well as afferents from the septum; (3) the *stratum pyramidale*, or pyramidal layer, which is dominated by the cell bodies of the pyramids; (4) the *stratum radiatum* and (5) the *stratum lacunosum/moleculare* which are, respectively, the proximal and distal segments of the apical dendritic tree (the stratum radiatum accounts for 70 per cent of the tree in the rabbit and 42 per cent in the guinea pig, according to Hjorth-Simonsen (1973)). There are some scattered cells throughout these layers, making contact with various parts of the pyramidal cell dendrite. The dominant feature conveying lamination to these layers, however, comes from other afferents to the dendrites which typically stream past at right angles, making numerous synapses *en passage* as described above. These inputs will be discussed in greater detail later. In the CA3 field an additional layer is recognized: the *stratum lucidum,* interposed between the pyramidal cell bodies and the stratum radiatum, receiving the mossy-fibre input from the dentate granule cells.†

3.3. Ontogenetic Development of the Hippocampus

3.3.1. GROSS MORPHOLOGY

A brief look at the ontogenetic development of the mammalian hippocampus will show how it comes to assume its striking, highly laminated, double-horseshoe shape. Fig.8 (facing p.109) illustrates six stages in the development of the rabbit hippocampus, compiled from the papers of Stensaas (1967 *a,b,c,d,e,* 1968). The details of hippocampal structure are all drawn to the same scale. Beginning its life as a narrow strip of cortical tissue trapped along the medial wall of the hemisphere, the hippocampus finds nowhere to go as it expands except into the ventricle. It first pushes laterally and then doubles back underneath itself. It has been suggested that the U-shape of the adult fascia dentata is due to the pressure of the hippocampus proper invaginating into the granule-cell layer during its development (Bayer and Altman 1974).

In the earliest stages (Fig. 8 (*a*)) the cell bodies of the precursors of both neurones (*neuroblasts*) and astrocytes (*spongioblasts*) are concentrated in a layer adjacent to the ventricle called the *matrix lamina.* Here, new neuroblasts are continually formed by cell division. After their final division the immature neurones migrate towards the pial surface where they congregate in the cortical plate. As in all cortical tissue, with the exception of the fascia dentata, the formation of the cortical plate takes place in an orderly inside/out fashion; newly formed neurones of the hippocampus proper push up through the older neurones of the cortical plate and come to rest at the edge facing the pial surface. Stensaas (1967*c*) has suggested that the migrating neuroblasts encounter a condition at this level which discourages movement and promotes the growth of dendrites.

As development proceeds, the number of neurones in the cortical plate rapidly increases and the hippocampus begins to take on its characteristic enfolded shape (Fig. 8*d*, *e*). During this period the future fascia dentata is represented by a pool of germinal cells gathered above the end of the cortical plate towards the fimbria. The development of the fascia dentata lags well behind that of the rest of the hippocampus, and indeed behind the rest of the brain in general, continuing postnatally in some species.

3.3.2. POSTNATAL DEVELOPMENT OF FASCIA DENTATA GRANULE CELLS

The postnatal development of the rabbit brain has not been studied anatomically,† but in other altricial‡ animals (e.g. rat and mouse) the fascia dentata does not reach its final adult form until some time after birth. Both the differentiation and migration of cells have been studied in great detail in the rat using the techniques of autoradiographic labelling and low-level X-irradiation. The former technique is based upon the fact that tritiated thymidine will only be incorporated into the DNA of cells which are formed shortly after injection (i.e. during mitosis). Thus, cells in the final mitotic stage at the time of injection will be most heavily labelled, while those dividing again subsequently will contain less labelled DNA. Low-level X-irradiation selectively kills cells which are dividing but has no effect on mature cells. The number and distribution of labelled or pyknotic (killed) cells at different times during development can be used to construct a picture of the generation and migration of neurones. Using these techniques Angevine (1965) and Altman and his colleagues (Altman and Das 1965, Altman 1966, Bayer and Altman 1975, Hine and Das 1974) have shown that the vast majority of the pyramids of the hippocampus proper are formed prenatally in the mouse and rat. On the other hand approximately 85 per cent of the dentate granules originate after birth, as do a fair proportion of the interneurones in all parts of the hippocampus. Of the 85 per cent of granule cells formed postnatally 25 per cent originate during the first four days after birth and 20

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† In addition to histological evidence for lamination, there is also evidence from histochemical studies of putative transmitter systems (e.g. Storm-Mathisen and Blackstad 1964, Storm-Mathisen 1976) and oxidative enzyme systems (Mellgren 1973, Mellgren and Blackstad 1967). For instance, Storm-Mathisen (1972) has shown that gamma-aminobutyric acid (GABA), as indicated by the presence of glutamic acid decarboxylase (GAD), is primarily distributed in two layers of the hippocampus, the stratum pyramidale and the stratum lacunosum/moleculare. This putative inhibitory neurotransmitter is thus found in the two layers thought to contain inhibitory interneurones, the basket cells in the former layer and the stellate and fusiform cells in the latter (cf. Cajal 1911 Figs 473, 474) Other work indicates that inputs to the hippocampus involving both nor-adrenergic fibres (Swanson and Hartman 1975) and serotonergic fibres (Moore and Halaris 1975) also terminate in these layers and that they have inhibitory effects as well. We shall discuss this at length later.

†A recent electrophysiological study has investigated the postnatal development of the commissural connections in the rabbit (Thomson 1975).

‡Altricial animals are confined to a nest and nursed for some time postnatally.

per cent more in the next four days (Bayer and Altman 1975). The formation of synapses between the perforant path axons from the entorhinal cortex and the dendrites of the granule cells takes place in step with cell formation; the biggest surge in synaptic formation occurs in the period between postnatal days 4 and 11 when the number of synapses in the exposed blade doubles every day and the synaptic density increases 20 times. The total number of synapses in the exposed blade increases 1000-2000 times from the fourth postnatal day to adulthood, while the buried blade, which develops earlier, increases 300 times (Crain *et al.* 1973). Electron microscopic studies of these synapses in the adult rat show that they have very complex geometrical configurations. This adult level of complexity is not attained until day 25 after birth (Cotman, Taylor, and Lynch 1973); whether this has any functional significance is not clear.

The development of the fascia dentata is organized according to several spatial gradients. As we have already noted, the buried blade develops before the exposed blade. In addition, the ventral fascia dentata develops before the dorsal part. Finally, the accretion of new cells to the cell-body layer follows an outside/in gradient. Newly formed neuroblasts migrate to the cell layer and nestle in underneath the existing granules (Angevine 1965, Altman and Das 1965, Schlessinger, Cowan, and Gottlieb 1975), in contrast to other cortical areas where the new immigrants push up through the older residents and settle at the top of the cortical plate. It was originally thought that the germinal pool from which the granule cells were formed was located in the juxtaventricular matrix lamina near the fimbrial end of the hippocampus (Altman and Das 1965). Neuroblasts formed here would then have to migrate through the alveus and pyramidal layer of CA3 to reach the fascia dentata. More recent studies (Schlessinger *et al.* 1975, Bayer and Altman 1975) indicate that a germinal pool of precursor cells is actually set up in the anlage of the fascia dentata itself.

3.3.3. PRINCIPLES OF AXO-DENDRITIC CONNECTION

It has been suggested that these gradients of development might explain the laminated structure of the adult hippocampus (Gottlieb and Cowan 1972). The site of contact between a developing axon and a dendrite would be determined by the sequence in which the afferents present themselves and the availability of appropriate receptor sites on the dendrites. This would explain, for example, the fact that the commissural afferents from the opposite hippocampus occupy the portion of the dendrite closest to the soma on the basis that they arrive before the entorhinal afferents. These latter latecomers would be obliged to synapse on the outer segments of the dendrite.

There are two pieces of evidence which might be taken to argue against a first-come, first-served principle as the only one in operation. The first

is that a lesion of the commissural system in neonatal rats does not result in perforant path fibres occupying sites closer to the granule cell body (Hjorth-Simonsen and Jeune 1972, Lynch, Deadwyler, and Cotman 1973); they remain restricted to the outer portions of the dendrite. Interestingly enough, however, a neonatal lesion of the perforant path does result in an encroachment of the commissural fibres into the upper zone normally reserved for the perforant path fibres (Lynch, Stanfield, and Cotman 1973, Zimmer 1973). The second series of studies which casts doubt on the Gottlieb-Cowan principle is that done on the synaptic connections within the hippocampus of the mutant 'reefer' mouse (Bliss, Chung, and Stirling 1974, Bliss and Chung 1974). Because of a defective gene for the control of migrating neuroblasts, these animals end up with a considerably disorganized hippocampus.† The granule-cell bodies do not line up in an orderly fashion but instead are scattered haphazardly throughout the whole region of the area dentate. Contact between the incoming perforant path axons and the granule-cell dendrites is achieved by the axons maintaining their normal positions relative to the pial surface and the aberrant granule cells extending their dendrites up to meet them. The synaptic contact, then, is a result of the dendrite seeking the axon. An entirely different principle is revealed by the synaptic contacts formed between the Schaffer collaterals (from CA3 pyramids) and the CA1 dendrites. Here, it is the axon which alters its course to meet preferred parts of the dendrite; the two layers of cell bodies (more distinct than in the normal mouse) are mirrored by two layers of Schaffer collateral synapses. Electrophysiological experiments by Bliss and his colleagues show that in both systems functional synaptic contacts are established.

However, Zimmer (1974) has strengthened the case for the 'spatial-proximity' notion by demonstrating that the reason for the failure of the perforant path fibres to encroach upon the termination sites of commissural fibres after decommissuration resides in the presence of ipsilateral association fibres which occupy the newly available sites. In agreement with this, Hjorth-Simonsen and Zimmer (1975) and Zimmer and Hjoith-Simonsen (1975) have demonstrated the importance of spatial proximity in the termination of afferents from the entorhinal area to the fascia dentata both in rabbit and rat. These more recent studies give new currency to the Gottlieb-Cowan principle.

3.3.4. DEVELOPMENT OF THETA

Another approach to the development of the hippocampus is to study the times of onset of the various patterns of the adult EEG. Vanderwolf *et al.* (1975) have studied the onset of theta activity in the rabbit, rat, and guinea

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† Other structures such as the cerebellum and neocortex are equally disorganized, but these do not concern us here.

pig.[†] At birth the EEG's of altricial animals such as the rat and rabbit do not show any theta. A theta pattern (related to movements) develops in both species around 12-14 days postnatally. A second type of theta (not associated with movements) develops around 22-24 days postnatally in the rabbit. In the guinea pig, a species which is behaviourally mature at birth, both types of theta can be recorded at birth.

3.3.5. FUNCTIONAL IMPLICATIONS OF DELAYED MATURATION OF HIPPOCAMPUS

Before we leave the subject of the ontogeny of the hippocampus, we should enquire into the possible functional significance of the postnatal development of various features of the adult structure. There are at least two possible results of this delayed development. Either (1) the hippocampus is functioning before the conclusion of postnatal neurogenesis, but in a manner different from that in the adult, or (2) the hippocampus does not function at all until it has completely developed.[‡] Aside from the postnatal development of fascia dentata cells there is also postnatal development of the cholinergic input systems to the hippocampus, as shown by Mellgren (1973). This may account for the fact that the two separate theta patterns noted above develop at different times. As we shall suggest later, the CA1 region seems responsible for the triggering of exploration, and the time of onset of theta activity from CA1 agrees nicely with the onset of exploration in neonates (Douglas, Peterson, and Douglas 1973). This suggests a possible function of postnatal development; the delayed maturation of the hippocampus could have the negative effect of delaying the onset of exploration. During the period when the animal is still dependent upon the mother it would be dysfunctional for it to be continually leaving the nest to explore its environment. Spontaneous alternation, which is a reliable measure of exploration, develops around day 28 in rats (Douglas *et al.* 1973), but is already present in the guinea pig during the first week of life.

The possibility that the hippocampus functions in a different way in neonates can only be tested adequately by studying the properties of single neurones, and this has not yet been done.

3.4. Afferents to the hippocampus

The cells of the hippocampus receive afferents from several sources: (a) intrinsic afferents from cells of the same sector; (b) intrinsic afferents from other sectors; (c) commissural afferents from the opposite hippocampus; (d) extrinsic afferents from outside the hippocampus. We shall discuss each of these in turn.

3.4.1. INTRINSIC AFFERENTS FROM THE SAME SECTOR

There are two types of interactions between pyramidal cells within the same CA field: a direct excitatory one and an indirect inhibitory one. It is likely that the indirect inhibitory effect is mediated by an interneurone, probably the basket cells described above. As we have seen, the axons of the basket cells make contact with numerous adjacent pyramidal cells; the basket cells in turn get part of their input from these same pyramidal cells. This feedback loop could form the basis for a Renshaw-type inhibitory circuit. The evidence that the basket cells play this role comes from several physiological studies.

In the first of these Kandel and Spencer and their colleagues (e.g. Kandel, Spencer, and Brinley 1961, Spencer and Kandel 1961c) showed that stimulation of the fornix was almost invariably followed by a prolonged inhibition of the CA3 pyramidal cells which was associated with an intracellular hyperpolarization. This potential could be reversed by artificially hyperpolarizing the cell with current injected through the recording electrode; this indicates that the potential was a true inhibitory post-synaptic potential (IPSP). In some cases the IPSP was preceded by an excitatory post-synaptic potential (EPSP) or an antidromic spike. By using a preparation in which the fornix had been sectioned several weeks prior to the acute experiment, and thus contained no afferent fibres to the hippocampus, they showed that the inhibitory effects could be generated by antidromically activating the efferent fibres from the hippocampus. They also concluded that the excitatory effects were due to afferents to the hippocampus, a conclusion which has been modified by later work from the same laboratory, as we note shortly. On the basis of latency measurements they decided that the IPSP was mediated by a direct axon collateral of the output fibres or by, at most, one interposed interneurone.

Andersen, Eccles, and Løyning (1964a,b) confirmed these findings and argued that the inhibition was mediated by a Renshaw-type interneurone which they identified as the basket cell of Cajal. This was supported by three lines of evidence: (1) the finding of a rapid oscillation on the ascending limb of the IPSP which they attributed to the action of the interneurone; (2) the fact that the maximum for the positive inhibitory potential evoked by weak stimulation of various input paths was centred around the stratum pyramidale; (3) their ability to record from cells with high-frequency bursts to the stimulus and no IPSP, which they felt were the interneurones in question. This last group of cells was located in the stratum oriens as well as in the stratum pyramidale. The authors concluded that the widespread IPSP following antidromic stimulation is mediated by an axon collateral from the pyramidal cell onto the basket cell, which in

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[†] Theta (θ) activity is a characteristic rhythmic electrical activity, involving frequencies in the range of 4-12 Hz in most species. We will discuss the functional significance of this activity later (see pp. 160-90).

[‡] This position has been espoused by Altman, Brunner, and Bayer (1973) who pointed out the behavioural parallels between infant rats and adult rats with hippocampal damage. A critical review of their presentation of the literature on the effects of hippocampal lesions in adults is found in Nadel, O'Keefe, and Black (1975).

turn has a widespread inhibitory action on many pyramidal cells. Subsequent studies (Andersen *et al.* 1969) indicate that the inhibitory effect of one interneurone may extend over 1 mm. or more. Although there is no direct evidence for the fascia dentata it is likely that a similar mechanism operates there.

In addition to this indirect inhibitory interconnection between cells in the same sector there is evidence for a direct monosynaptic excitatory connection between pyramidal cells of the CA3 field. Lebovitz, Dichter, and Spencer (1971) used the de-afferented fornix preparation described above to demonstrate this. In addition to the expected antidromic activation of many pyramidal cells and the subsequent widespread inhibition found by Kandel and Spencer, they also recorded some cells which were orthodromically activated with short latencies. Since a small percentage of these cells (12.5 per cent) could also be antidromically activated by stimulation of the fornix, and therefore must have been pyramidal cells, the authors concluded that some CA3 pyramidal cells must make monosynaptic contact with each other. A possible anatomical substrate for this contact is the longitudinal association pathway described by Lorente de No (1934). According to Lorente de No, this pathway arises in the CA2 and CA3 fields, either as a collateral of the pathway known as the *Schaffer collateral* system or as a collateral of the pathway destined for the fimbria, and connects cells within the same sector. Hjorth-Simonsen (1973) has confirmed the existence of this pathway using the Fink-Heimer method, but believes that its origin is restricted to the same fields as the Schaffer collaterals.

3.4.2. INTRINSIC AFFERENTS FROM OTHER SECTORS

The major interconnections between the three sectors, demonstrated anatomically (Lorente de No 1934, Raisman, Cowan and Powell 1965, Hjorth-Simonsen 1973) and physiologically (Andersen, Blackstad, and Lømo 1966, Fujita and Sakata 1962, Gloor, Vera, and Sperti 1963), are primarily unidirectional, starting from the fascia dentata, coursing through the CA3 field, and ending in CA1 (see Fig. 6).

The thin (rat, 0.2-0.5 µm (Laatsch and Cowan 1966), 0.1-0.2 µm (Blackstad 1963); rabbit, 0.3µm (Hamlyn 1962)) slowly conducting (cat, 0.75-1.0 m s^{-1} (Gloor, Vera and Sperti 1963)), unmyelinated axons of the dentate cells gather together in the polymorph layer in the hilus of the fascia dentata and course out of the hilus as the *mossy fibres* of Cajal. They run in two separate bundles, one above the pyramids of CA3 in the stratum lucidum (Fig. 6) and one below the pyramids in the stratum oriens (Fig. 6).[†] The lower bundle stops abruptly within the CA3 field (between sub-

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[†] The exact course of these bundles may differ from species to species, and within different strains of the same species. Barber *et al.* (1974) have shown such differences in different strains of mice, including the existence of an intrapyramidal bundle running within the pyramidal layer in one strain.

areas termed CA3b and CA3c), but the upper one continues to the border of CA1.[†] All along their course the mossy fibres make numerous synapses *en passage* with the dendrites they pass, first in the CA4 field and then in the CA3 field. They make contact both with large spines or excrescences, which are a prominent feature of the CA4 and CA3 pyramidal cell dendrites (Hamlyn 1961, 1962, Blackstad and Kjærheim 1961, Laatsch and Cowan 1966), and with the dendrites themselves. A combined electrophysiological–electron-microscopical study has shown these synapses to be excitatory Gray's type I (Andersen *et al.* 1966). These synapses are particularly interesting in that they contain a large amount of zinc. Thus, zinc levels in the hippocampus increase postnatally in conjunction with the development of the mossy-fibre system (Crawford and Connor 1972), while lesions of the mossy-fibre bundle cause a rapid loss of zinc from the synapse (Haug *et al.* 1971). The hippocampus overall has a concentration of zinc which is three times higher than the rest of the brain (Fjerdingstad, Danscher, and Fjerdingstad 1974a,b).[‡] It was originally reported that the temporary depletion of zinc through the use of H_2S rendered the mossy-fibre synapses inoperable (von Euler 1962, Segal 1972). However, subsequent experiments which have used non-toxic, specific chelating agents such as diphenylthiocarbazone (Crawford, Doller, and Connor 1973) or DEDTC (antabuse, Danscher, Shipley, and Andersen 1975) have failed to replicate this effect and have concluded that it might be due to the nonspecific toxic effects of the gas. The substance of which zinc is a component has not yet been identified (Blackstad *et al.* 1970), nor has its role in synaptic transmission, if any, been discovered.[***] Edström and Mattsson (1975) have shown that zinc stimulates the rapid transport of material in axons by its effect on microtubules, and this might form the basis for its action in the mossy-fibre system.

The axons of both the giant CA3 pyramids (Fig. 6) and the modified pyramids of CA4 divide, with one branch entering the fimbria and going to the septum, while the other branch, or branches, remain within the hippocampus. Three projections have been described for these collaterals:

(1) The *Schaffer collateral* system arises from the CA4, CA3c, and perhaps some of the CA3b pyramids and runs in the stratum radiatum of CA1, making powerful excitatory synapses *en passage*

[†] Lorente de No believed that the mossy fibres only extended to his CA2 field, but see p. 109 for a discussion of Blackstad's criticisms of this view.

[‡] They also showed that the hippocampus has a 10 times higher concentration of lead that the rest of the brain, but without localizing it within the hippocampus.

[***] Zinc is a component in a number of enzyme systems, including alcohol dehydrogenase. The possibility that Korsakoff's psychosis, which often develops after chronic alcohol over-consumption, is in some way related to the zinc in the mossy fibres, or elsewhere, has never been directly tested. Zinc is also a component in glutamic acid dehydrogenase as well as being a strong inhibitor of glutamic acid decarboxylase, enzymes important in the metabolism of both glutamic acid and GABA (cf. Storm-Mathisen (1976) for discussion and references). Thus, zinc might play some role in regulating the activity of these putative neurotransmitters.

(Fig. 6) (Lorente de No 1934, Hjorth-Simonsen 1973, Andersen, Blackstad and Lømo 1966); these fibres are also of rather small diameter (0.1-0.2 μm in the rat (Blackstad 1963)).[†]

(2) Collaterals of CA4 and CA3c (perhaps from the same cells which give off the Schaffer collaterals) bend around the buried blade of the fascia dentata and run parallel to the long axis of the hippocampus, making contact with the proximal dendrites of the dentate granule cells (Fig. 6) (Zimmer 1971).

(3) The longitudinal association pathway of Lorente de No, which has already been described (p. 118).

The CA1 pyramidal cells send their axons out of the hippocampus via the alveus (Fig. 6). This projection will be discussed later as part of the hippocampal efferent system. It is generally agreed that there is no projection from CA1 to CA3 or CA4 (Raisman, Cowan, and Powell 1966, Hjorth-Simonsen 1973). The possibility of a projection from CA1 to the fascia dentata is somewhat more controversial. Raisman *et al.* (1966) found degeneration in the dentate area following posterior CA1 lesions, but Hjorth-Simonsen (1973) has argued that their lesions encroached upon the perforant path; lesions which do not do so yield no degeneration in the fascia dentata.

The question arises as to the topography of the projections within this circuit: do the granule cells in a small area send their axons to an equally restricted area of CA3, or is the projection more diffuse? Blackstad *et al.* (1970) and Andersen, Bliss, and Skrede (1971) agree that the projections are extremely precise and restricted, each small group of granule cells sending an excitatory (Andersen, Holmqvist, and Voorhoeve 1966a,b) projection to a small group of cells in the next link of the circuit.[‡] Further, the entorhinal input to each of these cell groups is similarly restricted (Andersen *et al.* 1971, Lømo 1971a, Hjorth-Simonsen 1972), as we shall see shortly. On the basis of these and other anatomical considerations Andersen *et al.* (1971) proposed that the principal organization of the hippocampus is a sandwich of many of these relatively independent lamellae, which are roughly perpendicular to the long axis of the hippocampus (see Fig. 9). These lamellae, of course, are not completely independent; as we have noted there are fibre bundles connecting cell groups in the longitudinal direction as well.

Within this restricted projection from one field to the next there is an interesting shift in the pattern of termination as one moves from one end of the lamella to the other (Zimmer 1971, Hjorth-Simonsen 1973). For

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[†] These collaterals may account for the curious finding (Daitz and Powell 1954) that, after a lesion in the fornix-fimbria system, afferents to the hippocampus degenerate normally while the efferents on the hippocampal side of the lesion survive intact. No chromatolytic changes are seen in the hippocampal cells giving rise to both the efferent output and the Schaffer collaterals.

[‡] Lynch *et al.* (1973) supported this story with a horseradish peroxidase transport study of the mossy-fibre system; with 0.1 μl injections terminals were restricted to within 250-350 μm.

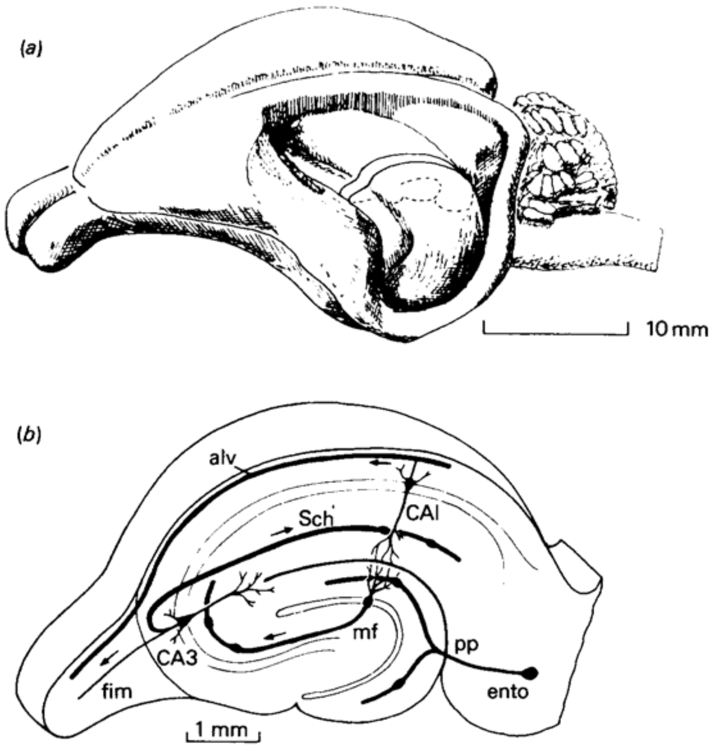

FIG. 9. Lamellar organization of the hippocampus: (a) Lateral view of the rabbit brain with the parietal and temporal neocortex removed to expose the hippocampal formation. The lamellar slice indicated has been presented separately in (b) to show the proposed circuitry. alv, alveus; ento, entorhinal cortex; fim, fimbria; pp, perforant path; Sch, Schaffer collateral. (From Andersen *et al.* 1971).

example, Zimmer, in his study of the projection from the CA3-CA4 field to the dentate granule cells, found that degeneration in the middle of the lamella filled the whole of the projection area on the proximal one-third of the dendrites but only part of this projection area at the extremes of the lamella. As one moves from the rostral end of the hippocampus in a temporal direction there is a shift in the localization of fibre terminations; rostrally they terminate proximal to the cell body while temporally they terminate distal to the cell body. There is a similar medio-lateral movement such that the most rostral projections are confined to the exposed blade, while those at the temporal end go to the buried blade. A similar imbricated pattern of termination has been reported by Hjorth-Simonsen (1973) for the longitudinal association pathway and, to a lesser extent, for the Schaffer collateral pathway.

This pattern strongly suggests that each cell, or group of cells, in CA3-CA4 projects to different points on the dendritic trees of several granule cells, either through multiple collaterals or through a single axon making multiple synapses *en passage*. We shall suggest later (pp. 225-7) that a mechanism exists for blocking the receptivity of most of the dendritic tree of these cells at any given time. The combined operation of these two mechanisms could act as a sorting device such that a given input would only activate a small subset of its target neurones.

3.4.3. COMMISSURAL AFFERENTS

Extensive connections exist between the two hippocampi crossing the midline in the ventral and dorsal hippocampal commissures or *psalteria*. The *ventral hippocampal commissure* consists of fibres running in the fimbria which turn back at the rostral end of the hippocampus to run in the fimbria of the opposite side (Fig. 3). These fibres are relatively small (0.6, 0.9, and 1.2 μm for cat, rabbit, and rat, respectively) and slowly conducting (3.3, 5.6, and 7.5 m s^{-1}) according to Andersen (1960c). The *dorsal hippocampal commissure* forms a thin layer of fibres which run beneath the splenium of the corpus callosum at the posterior arch of the hippocampus where it begins to turn downward.

Following large lesions of the contralateral hippocampus, or section of the ventral hippocampal commissure, degeneration is observed in the stratum oriens of all fields, in the stratum radiatum of CA1 and, to a lesser degree, CA3, and in the proximal (juxtagranular) one-third of the stratum moleculare of the fascia dentata (Blackstad 1956, Raisman *et al.* 1965, Laatsch and Cowan 1967). A small amount of degeneration in the stratum lacunosum/moleculare of CA1 (and stratum moleculare of subiculum) follows section of the dorsal hippocampal commissure. This general picture has been confirmed in an electrophysiological study by Thomson (1975).

The site of origin of these commissural projections has been studied in a variety of ways. In early work Andersen (1959, 1960a, b, c) and Raisman *et al.* (1965) used electrophysiological and classical degeneration techniques and observed a similar pattern; there was a symmetrical connection between homotopic points on the two sides. That is, CA3 projected to CA3; while CA1 (posterior) projected to CA1 (posterior); CA1 (anterior) did not seem to send commissural afferents, though it did receive efferents. Finally, there was some debate concerning fascia dentata commissural inputs; Raisman *et al.* suggested a diffuse origin while Andersen, Bruland, and Kaada (1961a) presented electrophysiological evidence for a non-homotopic commissural input to fascia dentata from CA3.

More recent studies, using either autoradiography (Gottlieb and Cowan 1972) or the retrograde transport of horseradish peroxidase (Segal and Landis 1974), have clarified the picture. There are clear-cut homotopic projections from CA3 to CA3, but non-homotopic inputs to both CA1 and fascia dentata. These two areas both seem to receive their commissural inputs from CA3 as well. Gottlieb and Cowan (1973) have pointed out that this pattern of commissural innervation indicates that

> 'each field of the hippocampus which contributes to the commissural projection also gives rise to an ipsilateral association pathway which follows the same intrahippocampal course, and terminates within the same region' (p. 420).

In other words, CA1 and fascia dentata receive inputs from CA3 on both sides. The significance of this restricted, highly specific, pattern is not clear at present.

3.4.4. EXTRINSIC AFFERENTS TO THE HIPPOCAMPUS

The hippocampus receives extrinsic afferents from a variety of other structures, including primarily the entorhinal cortex, the medial septal area, and several brain-stem sites. Difficulties in interpreting the exact input patterns arise because some brain-stem afferents course through the septum and the retrosplenial cortex on their way to the hippocampus. Thus, traditional anatomical techniques can lead to misinterpretations based on lesions in fibres of passage. The same, of course, applies to the study of hippocampal efferents, as we shall see. More recently enzyme histochemical and retrograde transport methods have facilitated the analysis of distinct input (and output) pathways. None the less, some controversy persists, particularly regarding hippocampal efferents, as we note later. Here, we shall discuss afferents under four headings: *(a)* entorhinal efferents; (*b*) brain-stem afferents; (*c*) septal afferents; *(d)* other afferents. The evidence concerning the mediation of hippocampal theta activity by various of these afferents will be discussed in a subsequent chapter.

3.4.4(a). Entorhinal afferents. Cajal (1911) described three pathways to the hippocampal region from the entorhinal area: (1) the *perforant* path; (2) the *alvear* path; (3) the *crossed temporo-ammonic* tract. The crossed temporo-ammonic tract distributes to the presubiculum; the alvear path (if it exists*) remains in the deep layers of the subiculum and contributes only a small number of fibres to the stratum oriens of CA1. Major interest, therefore, has centred on the other pathway, the perforant path, since it appears to be the major avenue of entorhinal afferents to the hippocampus.

According to Lorente de No (1934), Blackstad (1958), and Raisman *et al.* (1965) the perforant path originates in the lateral entorhinal cortex (see

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* Nafstad (1967), in a combined degeneration-electron-microscope study, found fibres of passage in CA1 but terminal degeneration in less than 1 per cent of the synaptic terminals. Similarly, Hjorth-Simonsen (1972) could find no terminal degeneration in CA1 following lateral or medial entorhinal lesions. Segal and Landis (1974) confirmed these findings in a horseradish peroxidase transport study; there was no label transported to the entorhinal cortex if the peroxidase was injected into CA1 alone. Finally, Andersen, Homqvist, and Voorhoeve (1966b), on the basis of an electrophysiological study, concluded that the activation of CA1 pyramidal cells from the entorhinal area was not direct but by way of dentate and CA3 sectors.

Figs. 5 and 6), crosses the subiculum to reach its upper layers, and then either courses within these upper layers to enter the hippocampus proper (Fig. 6) or perforates through the obliterated hippocampal fissure to the fascia dentata (Fig. 6). More recent work (Nafstad 1967, Hjorth-Simonsen and Jeune 1972, Van Hoesen, Pandya, and Butters 1972, Hjorth-Simonsen 1973, Van Hoesen and Pandya 1975b) confirms this pathway but indicates that the fibres originate from the medial entorhinal cortex as well (Figs. 5 and 6). While the lateral pathway ends on the distal one third of the granule cell dendrites the medial pathway occupies the medial one-third (Hjorth-Simonsen 1972, Van Hoesen, Pandya, and Butters 1972, Fifková 1975). Although most of the projection is ipsilateral, there is evidence for a small contralateral projection (Goldowitz *et al.* 1975). There is some indication from one of Hjorth-Simonsen's experiments and from Van Hoesen and Pandya (1975b) that a lesion on the border of the lateral and medial entorhinal cortices yields degeneration intermediate to that described above; thus it is possible that one is dealing with a continuum rather than with discrete projections here. Given this, a dentate granule cell could tell where its input comes from by how high up on its dendrites the input synapses. On the other hand, this may be another example of the imbricated pattern described above (see pp.120-2) for various intrahippocampal projections. As can be seen from Fig. 6 there is also an input to the stratum lacunosum/moleculare of CA3, the lateral path terminating on the most distal portion of the dendrites, the medial path on the more proximal portion. While Van Hoesen, Pandya, and Butters (1972) failed to find a projection to the hippocampus proper from the entorhinal cortex in the monkey, Van Hoesen and Pandya (1975b) reported a projection limited to the uncal extremity of CA3 and the area between CA1 and CA3 in the rest of the hippocampus.[*]

The Hjorth-Simonsen and Jeune (1972) study provides anatomical evidence for a degree of topographical specificity of the entorhinal input to the dentate granule cells. Lesions of the dorsal entorhinal cortex caused degeneration restricted to the dorsal hippocampus; progressively deeper lesions yielded degeneration more posteriorly and then more temporally within the hippocampus. A similar precision in this projection has been demonstrated in the horseradish peroxidase study of Segal and Landis (1974) and in the careful electrophysiological studies of Lømo (1971a). It thus appears that the lamellar organization proposed for the hippocampal internal circuitry (see pp. 120-2) also extends to the entorhinal inputs (Andersen *et al.* 1971) (see Fig. 9).

The perforant path projection to the dentate granule cells has been shown to be powerfully excitatory (Andersen, Holmqvist, and Voorhoeve 1966a, Lømo 1971a), as has the projection to the CA3 pyramidal cells (Gloor, Vera, and Sperti 1963). The perforant path/dentate synapses are subject to long-term increases in excitability following a brief tetanus of electric shocks in the anaesthetized rabbit (Andersen, Holmqvist, and Voorhoeve 1966a, Lømo 1966, 1971b, Bliss and Lømo 1970, 1973), unanaesthetized rabbit (Bliss and Gardner-Medwin 1971, 1973), unanaesthetized rat (Douglas and Goddard 1975), and in *in vitro* slices of rat hippocampus (Deadwyler, Dudek, Cotman, and Lynch 1975).[*]

Inputs to the entorhinal area. Until recently information about inputs to the entorhinal cortex and other parahippocampal areas has been unsystematic and scattered. What was available suggested weak or uncertain inputs from the following cortical areas: (1) prefrontal and cingulate cortices via the cingulum bundle (Adey 1951, Adey and Meyer 1952, White 1959, Cragg 1965, Raisman *et al.* 1965, McLardy 1971, Leichnetz and Astruc 1975)[**] although Domesick (1969, 1970) has presented strong evidence that, in the rat at least, most of the fibres in this bundle are thalamo-cortical and end in cingulate cortex and presubiculum; Shipley (1974, 1975) has demonstrated a projection from the presubiculum to the dorsal and medial entorhinal cortex;[***] (2) temporal cortex (Cragg 1965); (3) parietal areas, either directly (Pandya and Kuypers 1969, Pandya and Vignolo 1969, Petras, 1971) or indirectly via the cingulate cortex (Cragg 1965) and the prepyriform cortex (Cragg 1961); (4) pyriform cortex (Powell, Cowan, and Raisman 1965). Niemer, Goodfellow and Speaker (1963) and McKenzie and Smith (1970) have obtained electrophysiological evidence of projections from large areas of the neocortex to hippocampus, perhaps via entorhinal areas, but the precise pathways were not studied.

In addition, there is evidence suggesting that both olfactory (Cragg 1960, 1961, Heimer 1968, White 1965, Price and Powell 1971, Kerr and Dennis 1972) and visual (Casey, Cuenod, and MacLean 1965, Cuenod, Casey and MacLean 1965) sensory systems gain access to the entorhinal area. It has been suggested that the visual input may not be mediated by the neocortex but may be direct from optic fibres bypassing the lateral geniculate nucleus (MacLean and Cresswell 1970), or via the newly discovered optic-tract input to the anterodorsal nucleus of the thalamus (Conrad and Stumpf 1975). Consistent with this notion is the report by Sager, Nestianu, and Florea-Ciocoiu (1967) of a long-latency (40 ms) visual evoked potential in the hippocampus which survived destruction of the geniculo-striate system. Cragg (1960) recorded the evoked potentials

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[*] A projection from the prorhinal area, adjacent to the entorhinal cortex, to the junction of stratum moleculare/stratum radiatum in field CA1*a* was also seen in this study.

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[*] Schwartzkroin and Wester (1975) have recently shown that the Schaffer collateral pathway from CA3 to CA1 also possesses this property, while the CA3-septum pathway does not (Andersen, personal communication). Thus, only the synapses within the hippocampus have this marked plasticity.

[**] See p, 131 for a description of a direct prefrontal-hippocampus projection reported in this study.

[***] This input is a highly organized one, indicating that the topographical specificity already noted for the hippocampus and the perforant path input extends back to the presubiculum, and possibly beyond that to the anterior thalamus (Shipley and Sorenson 1975).

generated in the hippocampus by olfactory-bulb stimulation in several species and found that this input decreased in importance with phylogenetic development. In rat, responses were easily recorded throughout the extent of the hippocampus, while in monkey only a small part of the anteroventral hippocampus responded. Lastly, the amygdala projects to the entorhinal cortex and to the subiculum (Krettek and Price 1974). This pathway could be responsible for the (multi-synaptic) connections from the amygdala to the hippocampus which have been demonstrated electrophysiologically (Gloor 1960, Gloor, Vera, and Sperti 1963).*

Van Hoesen, Pandya, and Butters (1972, 1975) and Van Hoesen and Pandya (1975a) have provided a thorough analysis of cortical afferents to the entorhinal area in the monkey. They found three main projection areas: (1) the adjacent prepyriform cortex, parasubiculum, and presubiculum (Brodmann's areas 51, 49, and 27) as well as the perirhinal and prorhinal cortex; (2) the adjacent area TF-TH of Bonin and Bailey, temporal cortex caudal to the entorhinal area; (3) the caudal portion of the orbito-frontal cortex, Bonin and Bailey's FF (Walker's areas 12 and 13). With the exception of the input from the perirhinal area, which projects to contiguous portions of the entire entorhinal cortex, all of these sources of input are highly specific in terms of laminar and sub-areal termination within the entorhinal region. Thus, the prepyriform cortex projects primarily to the lateral entorhinal area, as does the orbito-frontal cortex, while areas 49, 27, TF, and TH project primarily to the medial entorhinal area. The extent of interconnections in these areas is truly bewildering, and is best described in Fig. 10, taken from Van Hoesen, Pandya, and Butters (1972). This figure makes it clear that there is a cascading of inputs from a number of cortical areas through all adjacent regions leading ultimately to the entorhinal cortex. This pattern of inputs to the entorhinal area strongly suggests that the hippocampus is concerned not with information about any particular modality, but rather with highly analysed, abstracted information from all modalities.

3.4.4(b) Brain-stem afferents. Afferents to the hippocampus arise in several brain-stem areas. The median raphe nucleus, and particularly the nucleus centralis superior, projects to the hippocampus, accounting for most of the serotonin (5-hydroxytryptamine, 5-HT) found in this structure (Ungerstedt 1971, Storm-Mathisen and Fonnum 1972, Kuhar, Aghajanian, and Roth 1972, Storm-Mathisen and Guldberg 1974, Lorens and Guldberg 1974, Segal 1975, Moore and Halaris 1975). This projection courses primarily through the fimbria, fornix, and cingulum, terminating mostly in stratum lacunosum/moleculare (Fuxe and Jonsson 1974, Moore and Halaris 1975) of CA1 and CA3, and in a restricted part of the hilar zone beneath the granule cells in fascia dentata. Some inputs are seen to the stratum radiatum and the stratum oriens, more densely in CA3 than in CA1. There appears to be a higher 5-HT activity in more posterior portions of the hippocampus (Storm-Mathisen and Guldberg 1974). Iontophoretic application of 5-HT inhibits hippocampal (pyramidal) cells (e.g. Biscoe and Straughan 1966) as does stimulation in the raphe nucleus itself (Segal 1975). Thus, it appears as though a 5-HT inhibitory pathway from the median raphe nucleus to the hippocampus is well established.* De France, McCrea, and Yoshihara (1975) have shown that serotonin can facilitate units in CA1 and suggested that these effects are mediated via the Schaffer collateral system. Similar effects of serotonin were seen on cells in the lateral septum receiving inputs from the fimbria and CA3.

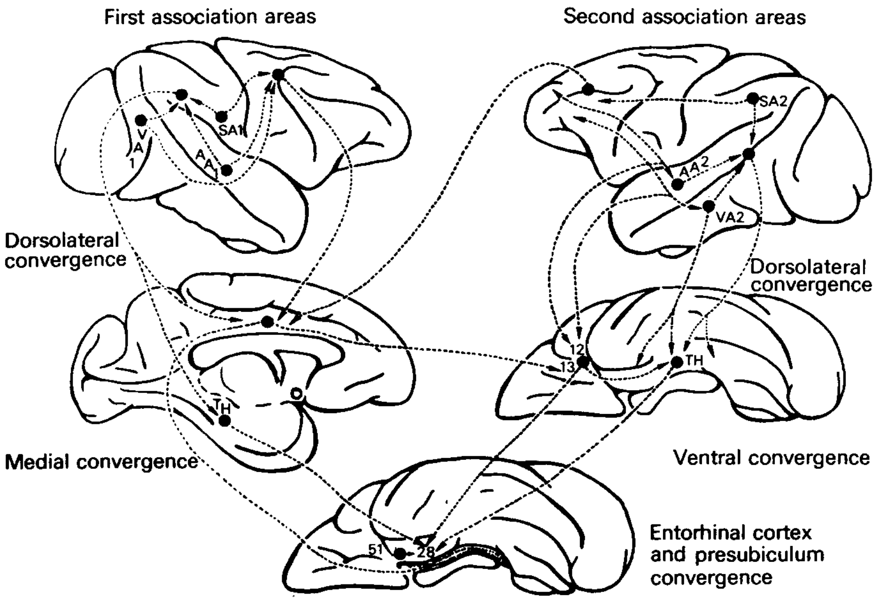

FIG. 10. Convergence of afferent information onto the entorhinal cortex from primary (SA1, AA1, VA1) and secondary (SA2, AA2, VA2) association areas of the neocortex in the monkey. (From Van Hoesen, Pandya, and Butters 1972; copyright 1972 by the American Association for the Advancement of Science.)

The locus coeruleus also projects to the hippocampus, accounting for much of the noradrenaline (NA) in this structure (Blackstad, Fuxe and Hökfeldt 1967, Ungerstedt 1971, Lindvall and Bjorklund 1974, Ross and Reis 1974, Fuxe, Hamberger, and Hökfeldt 1968). A recent immuno-

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* Recent work in the dog suggests that monosynaptic connections exist between amygdala and ventral hippocampus (Kosmal, personal communication); the functional significance of these is unclear.

* Wimer, Norman, and Eleftheriou (1973) have reported large differences in the amount of hippocampal 5-HT between two strains of mice and changes in amount of 5-HT with active avoidance training or etherization.

fluorescence study using dopamine-β-hydroxylase (DBH) as a marker has greatly clarified the projection pathway and termination sites of this system (Swanson and Hartman 1975). Fibres course through the medial forebrain bundle and septum, entering the hippocampus via retrosplenial cortex. There does not appear to be a projection via the fimbria or fornix. These NA fibres terminate rather specifically in the stratum lacunosum/moleculare of CA1 and CA3, the hilus of the fascia dentata, and the stratum lucidum of CA3. Here, they are in proximity to the mossy-fibre terminals. Mostly inhibitory effects have been reported after iontophoretic application of NA (e.g. Biscoe and Straughan 1966), and this is also seen after stimulation in the locus coeruleus (Segal and Bloom 1974*a*,*b*).

It is worth noting that these two systems, both inhibitory, take primarily different paths to the hippocampus, NA entering retrosplenially and 5-HT entering rostrally via the fimbria. Stimulation studies (see pp. 154-5) indicate that the lateral raphe pathway is influential in the elicitation of a particular pattern in the hippocampal EEG (termed SIA) which is different from that elicited via the more medial, locus coeruleus, pathway. Stimulation here elicits the characteristic hippocampal theta activity. A full discussion of these effects, which are partly mediated via the septum, is given in the next chapter (pp. 154-60). Gray *et al.* (1975) have provided pharmacological evidence implicating some noradrenaline system in theta activity (but see Robinson, Pappas, and Vanderwolf (1975) for a contradictory report).

In several physiological experiments (Grantyn 1970, Grantyn and Grantyn 1970, 1971, 1973*a,b*) the existence of an input to the hippocampus from the mesencephalic reticular formation has been demonstrated. Some hippocampal neurones respond to reticular stimulation with a hyperpolarization, some with a depolarization, and others with an oscillation between the two. This input survives a lesion of the septum and is probably mediated by the neocortex (Grantyn, Grantyn, and Hang 1971).

3.4.4(c). Septal afferents. A major input to the hippocampus arises in the septal area. There is some disagreement as to the exact origin of these fibres, their course, and the extent and locus of their termination within the hippocampus. Behind these strictly anatomical questions lies an important functional one: which part of this input is involved in the generation of hippocampal theta activity (see pp. 154-60)? The relevant studies are listed in Appendix Table A1

Most of these studies localize the origins of the septo-hippocampal projection to the medial septum and the dorsal (vertical) limb of the *diagonal band of Broca* (DBB); and deny any projection from the lateral septum and ventral (horizontal) limb of the DBB. Of the four studies reporting discrepant results, only three used classical anatomical techniques. All purported to show that medial septum-dorsal DBB projected to dorsal hippocampus while lateral septum-ventral DBB projected to

ventral hippocampus. There are two possible reasons for this discrepancy: (1) lesions might have involved fibres of passage, such as those coursing through the septum from the brain stem (see above); (2) there might be a topographic projection from the medial septum to the hippocampus such that the dorsal medial area projects to the dorsal hippocampus, and then as one moves ventrally and laterally within the medial septum the target in the hippocampus moves first posteriorly and then ventrally. Evidence for this exists in several studies, and most convincingly in a report by Segal and Landis (1974). They injected horseradish peroxidase into the CA3/CA4 field of either the dorsal or ventral hippocampus and examined the septum for retrograde transport of this enzyme. Only cells within the medial septum were labelled. Further, dorsal hippocampal injections yielded label in cells in the medial part of the medial septum, while ventral hippocampal injections yielded label in cells of the more lateral parts of the medial septum. Mellgren and Srebro (1973), in a combined acetylcholine esterase and Fink-Heimer degeneration study, reported results consistent with this notion.[*] Finally, studies of the depletion of choline acetylase (ChAc) in hippocampus after various lesions also indicate that the projection involving acetylcholine is restricted solely to the medial septal region (Srebro *et al.* 1973, Oderfeld-Nowak 1974, Storm-Mathisen 1972, Storm-Mathisen and Guldberg 1974).

The path taken by the septo-hippocampal fibres is also in dispute. Some authors (e.g. Andersen, Bruland, and Kaada 1961*a,b*, Powell 1963) trace the fibres in the dorsal fornix and thence across the top of the hippocampus in the alveus, others (e.g. Raisman *et al.* 1965, McLennan and Miller 1974a) trace them in the fimbria, and still others (e.g. Lewis and Shute 1967) trace them in both. Finally, Bland, Andersen, and Ganes (1975) found that a knife cut through the apical dendritic region of CA3 eliminated theta activity in their physiological experiment, suggesting this as the pathway for the septo-hippocampal fibres. Similarly, in some experiments (Raisman *et al.* 1965, Raisman 1966, Genton 1969) degeneration after septal lesions is restricted to hippocampal fields CA3 and CA4 and the fascia dentata, while others trace degeneration to all hippocampal fields in addition to the fascia dentata (Siegel and Tassoni 1971*b*; Ibata, Desiraju, and Pappas 1971, Hjorth-Simonsen 1973, Mellgren and Srebro 1973, Mosko, Lynch, and Cotman 1973). In some of these latter studies the CA1 projection was not as dense as the CA3/fascia dentata projection. Studies employing cholinergic stains (Lewis and Shute 1967, Lewis, Shute, and Silver 1967, Storm-Mathisen 1972, Storm-Mathisen and Fonnum 1972, Mellgren and Srebro 1973, Mosko *et al.* 1973) support the idea of a widespread cholinergic input to all fields in both dorsal and ventral hippocampus. In addition to the

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[*] De France, Kitai, and Shimono (1973*a,b*) reported antidromic activation of cells in the ventral lateral septum with fimbrial stimulation, but this could not be replicated by McLennan and Miller (1974*a*).

long-term loss of acetylcholine esterase (AchE) and choline acetylase (ChAc) staining after fimbrial section or septal lesions, it has been reported that immediately after fimbrial section there is a 60 per cent increase in AchE and ChAc in the hippocampus (Sethy *et al.* 1973). Further, stimulation of the surface of the septum (Smith 1972) or the medial septum itself (Dudar 1975) increases the release of AchE from the surface of the hippocampus.

There is somewhat more agreement on the question of the final termination of the septal afferents to the hippocampus; most authors find a major projection to the stratum oriens and stratum radiatum of the hippocampus and to the polymorph zone of the fascia dentata. In addition, some find a small projection to the molecular layer of CA4 and fascia dentata. No degeneration is found in the cell-body layers. Although it is assumed by most authors that the septal projection is to the pyramidal cells and granule cells, the heavy concentration of terminals in the stratum oriens and in the zone just above the cell bodies has suggested to Mosko *et al.* (1973) that the target cells might be interneurones and, more specifically, the basket cells of Cajal.

The septo-hippocampal projection is one of the most controversial in the brain, perhaps because it has been widely studied and used as a model system by so many investigators. Yet the exact origin and terminal distribution of this path has not been determined. No single methodological difference, such as species or stain used, seems sufficient to account for the reported discrepancies.* We have noted several possible explanations. First, the use of lesions involves the possibility of destroying fibres of passage. Second, and consistent with much of the evidence, the septum might project to the hippocampus in an orderly topographic manner. In addition to the medio-lateral septal difference noted above, evidence has been provided for a dorsal-to-dorsal and ventral-to-ventral projection (Mellgren and Srebro 1973) as well as an anterior-to-anterior and posterior-to-posterior projection in the monkey (De Vito and White 1966). Consistent differences in the placement of septal lesions would then account for the discrepant reports.

In summary, there is almost definitely a group of cholinergic fibres centred in the medial septum projecting sparsely to all ipsilateral fields of the hippocampus and to the fascia dentata. These fibres travel in the fimbria to CA3-CA4 and perhaps inside the CA3 hilus to the fascia dentata. Another projection may travel in the dorsal fornix and alveus to CA1 and also to the fascia dentata. The major termination zones are found just above and below the main cell bodies, and this could indicate terminations on the basket cells as well as on pyramidal cells. Evidence from iontophoretic

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* It should be noted that Ibata *et al.* (1971) mention that they could not find terminals in the hilus of the fascia using a Fink-Heimer method, although they were apparent with a Nauta-Gygax method.

studies (e.g. Bland, Kostopoulos, and Phillis 1974) indicates that these inputs are excitatory, more so in the fascia dentata than in the hippocampus proper.*

3.4.4(d). Other afferents. Aside from these major afferent systems to the hippocampus two other sources of input have recently been uncovered. Heath and Harper (1974) have demonstrated an input from the fastigial nucleus of the cerebellum to CA2, CA3, and CA4 and to the fascia dentata. The terminal sites include the stratum oriens, stratum radiatum and stratum lacunosum in the hippocampus proper, and the polymorph layer in the fascia dentata. Finally, Leichnetz and Astruc (1975) have demonstrated a direct projection in monkey from the medial prefrontal cortex to CA1 and CA3 localized within stratum oriens, stratum pyramidale, and stratum lacunosum/moleculare. This pathway courses through cingulate and uncinate approaches to the hippocampus, entering either through the alvear or perforant paths. Some degeneration was seen in the commissure and the alvear path of the contralateral hippocampus, indicating a bilateral projection. There was a suggestion of some specificity in the two modes of projection, the alvear input terminating on the basal dendrites and somata of pyramidal cells (and perhaps basket cells), the perforant input terminating on apical dendrites.

3.4.4(e). Evidence of hippocampal afferents from single unit studies. On the basis of the anatomical studies reviewed above, one might expect to find three different types of unit responses in the hippocampus: (a) specific responses to complex sensory stimuli mediated by the entorhinal afferents; (b) diffuse arousal responses to a wide range of sensory stimuli mediated by the brain-stem afferents arriving via the septal-fornix input and, to a lesser extent, via the entorhinal area; (c) motor information conveyed by the brain-stem afferents from posterior hypothalamus and reticular formation. It should be noted, however, that the third type of input can only be demonstrated adequately in animals which are free to move. Motor-related inputs are either movement-generated proprioceptive or vestibular feedbacks on the one hand, or collaterals from the movement-generating circuits themselves on the other. While the existence of the former type of input could be demonstrated in acute, paralysed animals by the passive manipulation of individual limbs or the whole animal, the collateral motor input would be more difficult to detect and might

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* There was an important difference between CA1 and fascia dentata cells, however: the excitation elicited by acetylcholine was blocked by atropine in CA1 but not in fascia dentata. Thus, the CA1 receptors were muscarinic while the dentate ones may be nicotinic. This difference may be of some significance to an understanding of the two different types of theta activity, as we note later (see p. 166).

easily be misinterpreted as a long-latency sensory response.*

Studies on the activity of single hippocampal neurones in response to sensory stimulation or during motor behaviour have been published from nine laboratories (Brown and Buchwald 1973, Feder and Ranck 1973, Green and Machne 1955, Lidsky, Levine, and MacGregor 1974*a,b*, Molnar and Arutyunov 1969, O'Keefe 1976, O'Keefe and Dostrovsky 1971, Ranck 1973, Segal 1974, Vinogradova 1970, Vinogradova, Semyonova, and Konovalov 1970, Yokota, Reeves, and MacLean 1970).** Most of these studies report that sensory stimuli have a non-specific arousing effect on hippocampal neurones, usually of rather long latency (> 100 ms.) Evidence for more specific sensory responses has been reported in four studies (Brown and Buchwald 1973, Green and Machne 1955, Molnar and Arutyunov 1969, Segal 1974). In the first of these, Green and Machne (1955) found that while many hippocampal units in the paralysed, unanaesthetized rabbit were multi-modal, activated by all the sensory stimuli that they tried, others could only be excited by one stimulus such as a touch on a particular part of the body and not by visual or auditory stimuli. There was no obvious tendency for units responsive to the same stimuli to cluster into anatomically discrete areas. In lightly anaesthetized cats Molnar and Arutyunov (1969) asked whether hippocampal neurones responded to one or more sensory inputs. When they used single light flashes, clicks or shocks to the skin, the neurones appeared to be unimodal but if repetitive stimuli were delivered virtually all neurones responded to all modalities. In the freely moving rat Segal (1974) also reported finding some units which responded to light flashes but not to tones. Brown and Buchwald (1973) concentrated on auditory stimuli and reported two types of specific response in the paralysed, unanaesthetized cat. Some units responded differentially to tones of different frequencies; some units were excited by white noise presented to the ipsilateral ear, but were inhibited by the same noise in the contralateral ear. Finally, Yokota *et al.* (1970) recorded short-latency EPSP's but no action potentials in hippocampal pyramidal cells in chronic monkeys following shocks to the olfactory bulb. The short latency might indicate a specific response, but since no other modalities were tested one cannot be sure. In other studies (O'Keefe 1976, O'Keefe and Dostrovsky 1971, Ranck 1973, Vinogradova 1970, Vinogradova, Semyonova, and Konovalov 1970) only non-specific responses to all sensory stimuli tested were found.

Finally, in the studies from our own and from Ranck's laboratory, in which the animal was free to move and its movements carefully monitored, units specifically related to movement were reported. In addition, unit activity which might in part reflect responsiveness to complex sensory inputs was also described. A fuller description of these unit types and possible reasons for the apparent discrepancies amongst the different studies is given in the section on single-unit activity in the freely moving animal (pp. 190-217).

3.5. Efferents from the hippocampal region

Appendix Table A2 lists those studies which have traced the degeneration resulting from damage to the hippocampus, the dorsal fornix, the fimbria, or the columns of the fornix. Until recently, there was considerable agreement that the hippocampus projects to (1) the lateral preoptic and lateral hypothalamic areas, (2) the septal region, (3) the thalamus, (4) the mammillary bodies, (5) the rostral mid-brain, and (6) caudally to the subiculum and the entorhinal cortex. Somewhat more controversial was the origin of a projection to the medial hypothalamus. Within these accepted projection areas there was some disagreement as to the exact sites of termination, and there appeared to be clear species differences as well. Almost all of the anatomical studies listed used techniques (e.g. Nauta or Fink-Heimer methods) which stained degenerating fibres after lesions. These, as we have noted, cannot distinguish between fibres originating within the area and those originating elsewhere but passing through the lesioned area. In the first autoradiographic study of the hippocampus Swanson and Cowan (1975, personal communication) have found that in the rat the hippocampus projects rostrally only to the septum. The source of the fibres to the thalamus, hypothalamus, and mammillary bodies is the more caudally placed subicular region. It had already been established that a major outflow of the CA1 field in the rat (Hjorth-Simonsen 1973) and the rabbit (Andersen, Bland, and Dudar 1973) goes to the subiculum. What was formerly considered to be a direct connection from hippocampus to thalamus and hypothalamus must now be considered indirect. One must keep in mind the possibility that there are minor species differences in the hippocampal efferents, but a radical difference between the rat and other species is unlikely. Swanson and Cowan (1975a) found that the efferent fibres of the subiculum and hippocampus clustered into different fibre tracts, the former in the post-commissural fornix, the latter in the pre-commissural fornix. Chronister, Sikes, and White (1975) have also reported that the post-commissural fornix receives fibres from periallocortex adjacent to the subiculum, notably presubiculum and parasubiculum, and that these two components of the post-commissural fornix have different target sites; we shall consider these in the context of a description of both fornix tracts, and

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* Black and his colleagues (see pp. 174-5) have shown that theta activity in the hippocampal EEG can be conditioned to a sensory stimulus in the paralysed, unanaesthetized dog, but that this theta activity is probably a concomitant of motor outflow signals and not related to sensory processing of the signal since the dogs invariably moved in response to the sensory stimulus when subsequently tested in the unparalysed state.

** Studies of the changes in hippocampal unit activity during sleep and wakefulness have not been included. These are discussed in the section on single unit activity in the freely moving animal (pp. 212-13).

their sites of termination. Subsequently, we shall discuss the caudally directed efferents and, finally, the topography of the efferent projections.

3.5.1. THE FORNIX-FIMBRIA SYSTEM

The rostrally directed output fibres of the hippocampus, subiculum, and adjacent areas gather together in the fimbria and perhaps the dorsal fornix; these join at the anterior part of the hippocampus to become the columns of the fornix. At this level many of the fibres cross the mid-line in the ventral hippocampal commissure and run caudally in the contralateral fimbria to reach the other hippocampus; these connections have been discussed already (p. 122). The bulk of the fibres in the fornix continue rostrally to penetrate the septo-fimbrial nucleus at the rear of the septal area. Here, the fibres split into the two components noted above, the postcommissural and pre-commissural fornices. As we just noted, the latter originates primarily in hippocampus, the former primarily in adjacent allocortical areas.

3.5.1(a). Pre-commissural fornix. The pre-commissural fornix distributes to most of the nuclei of the septal area, including the lateral septum, the diagonal band of Broca, and the bed nucleus of the anterior commissure; fibres also terminate in the nucleus accumbens septi, now thought to relate more to ventral caudate than to septum (cf. Heimer and Wilson 1975). Fibres traverse the septal region and continue into the preoptic area as the fascicles of Zuckerkandl, some terminating in the lateral pre-optic region while others turn caudally into the medial forebrain bundle to distribute to the lateral hypothalamus as far caudal as the optic chiasm. The existence of these lateral pre-optic and hypothalamic connections could not be established in the rabbit (Sprague and Meyer 1950, Cragg and Hamlyn 1960), and the pathway here might be an indirect one. Access to the more caudal lateral hypothalamic areas is gained indirectly from the large contribution to the medial forebrain bundle by septal and pre-optic areas.

3.5.1(b) Post-commissural fornix. The post-commissural fornix divides into two approximately equal components (A and B of Guillery 1956): one is destined for the thalamus, the other for the mammillary bodies and rostral brain stem. Chronister et al. (1975) have shown that the former, thalamic, projection derives from the pre- and parasubiculum, while the latter, mammillary, projection derives from the subiculum. This latter projection has been confirmed in a horseradish peroxidase study by Meibach and Siegel (1975).

The number of fibres in the post-commissural fornix before and after the thalamic component has split off has been assessed in several species by Powell *et al.* (1957). The numbers for rabbit, cat, and monkey are surprisingly similar (*c.*200,000 before and 100,000 after) and are about four to five times that seen in the rat. A huge increase is seen in humans, where there are about five times as many fibres as seen in monkeys. Comparable figures were reported by Simpson (1952) and Daitz (1953). The various investigators note that the pre-commissural components of the fornix, which could not be quantified, were at least as extensive as the postcommissural ones.

The thalamic destination of the post-commissural fornix fibres appears to vary from species to species. Valenstein and Nauta (1959), in a comparative study, found that the fibres distributed primarily to the antero-ventral and antero-medial thalamus and only sparsely to the intra-laminar thalamus in the rat and monkey, but that this relationship is reversed in the cat and guinea pig, with the dominant projection going to the intra-laminar group. Although they had no pure fornix lesions in the monkey studies they did find a projection to lateralis dorsalis and medialis dorsalis; this prompted them to speculate that the fornix input becomes more involved with the parietal and frontal lobes in higher species. Since the projection of the anterior thalamic group is to lateralis dorsalis and medialis dorsalis as well as to cingulate cortex, this change in termination sites with phylogeny amounts to the substitution of a direct for an indirect path. However, these terminations in the monkey must remain uncertain in the absence of pure fornix lesions, especially since there is evidence that the septum has a large input to medialis dorsalis in the cat (Guillery 1959); however, Raisman (1966) could not demonstrate this connection in the rat. The other major input to the anterior nuclei of the thalamus is from the mammillary bodies via the mammillo-thalamic tract (the tract of Vicq d'Azyr), which indicates amongst other things that the anterior thalamic nucleus compares the outflow from the subiculum, pre-, and parasubiculum, but only after a transformation of the subicular output in the mammillary bodies. Gergen (1967) reported that in many areas of the thalamus of the monkey, and in particular anterior nuclei and paracentralis, more than half of the cells are influenced by hippocampal stimulation, while Parmeggiani (1967) reported responses to low-frequency stimulation of the hippocampus in the following thalamic nuclei: AV, AM, VA, MD, CL, LD, paracentralis, and habenularis. Mok and Mogenson (1974) have confirmed this last projection.

The hypothalamic component of the post-commissural fornix distributes its fibres almost entirely to the mammillary bodies, and primarily to the lateral and posterior parts of the medial mammillary bodies. As we just noted, these nuclei in turn project to areas AV and AM in the thalamus via the mammillo-thalamic tract. The other projection of this area is to the mid-brain via the mammillo-tegmental tract. In addition to this indirect connection with the motor areas of the mid-brain, there are direct connections via fornix fibres which bypass the mammillary bodies to project directly to the mid-brain. The magnitude and site of termination of this direct projection varies with the species studied (Valenstein and Nauta 1959). In all species there is a termination in the rostral part of the mid-brain central grey.

In addition, in the guinea pig the fibres distribute to the subthalamic region and the central and caudal mesencephalic tegmentum, in particular the nucleus centralis tegmenti superior of Bechterev. The direct subthalamic projection also appears in the monkey, but it is indirect in the cat via a relay in the mammillary bodies. There is evidence from single-neurone recording studies for predominantly, but not exclusively, excitatory inputs from the hippocampus to the mammillary bodies (Poletti, Kinnard, and MacLean 1970), the lateral septum, preoptic region and various hypothalamic nuclei (Poletti, Kinnard and MacLean 1973), the thalamus (Yokota and MacLean 1968), and to the reticular formation (Grantyn *et al.* 1972).

Even before the work of Swanson and Cowan (1975a) there was some question as to whether the medial hypothalamic pathway to periventricular areas originated in the hippocampus or in the subiculum. Its origin in the hippocampus had been affirmed in the rat (Guillery 1956, Nauta 1956, Valenstein and Nauta 1959, but see Raisman *et al.* 1966, Chronister *et al.* 1975, for denials), in the guinea pig (Johnson 1959, Valenstein and Nauta 1959), in the rabbit (Cragg and Hamlyn 1960), in the cat (Siegel and Tassoni 1971a, but see Valenstein and Nauta 1959 for a denial), but *not* in the monkey (Valenstein and Nauta 1959). Raisman *et al.* (1966) had put forth a strong case that the pathway actually originated in the subiculum and ran in the fimbria. Lesions of the hippocampus or fimbria almost invariably destroy these fibres, but with lesions which spared both (Nauta 1956, Raisman *et al.* 1966) there was no degeneration in the medial cortico-hypothalamic tract. This tract is the most likely candidate for the hippocampal influence over the pituitary-adrenocortical system (see pp. 357-62).

3.5.1(c). Topography of the fornix-fimbria system. The topography of the rostrally directed efferents is somewhat controversial. There are two basic disagreements: (1) are there any rostrally directed efferents from CA1 (2) does the hippocampus project to the medial septum at all? Hjorth-Simonsen (1973) showed, in a degeneration study, that CA1 projected in a topographically organized way to the subiculum. This organization conformed to the lamellar pattern described earlier for the afferent systems. Andersen *et al.* (1973) extended this notion in an electrophysiological study in the rabbit. While they could antidromically activate CA3 pyramids from the fimbria, CA1 pyramids could only be antidromically activated from the caudal alveus and subiculum. This suggested that CA1 projected *only* in a caudal direction, a view opposed to the traditional story which suggested that CA1 efferents projected via the dorsal fornix and dorsal fimbria to the septofimbrial nucleus, the diagonal band of Broca, part of the ventrolateral septum, and the nucleus accumbens septi (Raisman *et al.* 1966). CA3 and CA4 were thought to project via the fimbria to the dorsolateral septum, the diagonal band, and the nucleus accumbens.

Siegel and his colleagues (Edinger, Siegel, and Trocano 1973, Siegel *et al.* 1974, Siegel and Tassoni 1971*a*, Meibach and Siegel 1975) suggest instead that the split in projections lies between the dorsal and ventral hippocampus, rather than the CA fields. They posit that the dorsal hippocampus projects to the medial septum via the dorsal fornix, while the ventral hippocampus projects via the fimbria to the lateral septum.* The autoradiographic study of Swanson and Cowan (1975a, Swanson, personal communication) favours Raisman's hypothesis. CA1 was found to project rostrally only to the ipsilateral lateral septum. CA3 projected solely to the lateral septum as well, but bilaterally and in a highly organized fashion, such that the more lateral one went in the hippocampus, the more lateral *within the lateral septum* the termination appeared. Similarly, the more ventral one went in the hippocampus, the more ventral within the lateral septum was the termination. The confusion rests, to some extent, upon the definition of what constitutes the medial septum.** Additionally, lesions have usually encroached upon fibres of passage, making clear-cut results unlikely.

Thus, according to Swanson and Cowan, the hippocampus projects solely to the lateral septum in a fashion which maintains the precise spatial ordering present in the afferent and intrinsic connections. Though the autoradiographic technique is not perfect, it does provide a cleaner analysis than most degeneration methods, and this work indicates that Siegel's data are probably contaminated by lesions beyond the confines of what was intended. If anything, diffusion of autoradiographic label, which constitutes the major methodological problem with this technique, would tend to expand the sites thought to receive hippocampal efferents. Thus, we can conclude that there are direct efferent connections between the hippocampus and the lateral, but not medial, septum.

3.5.2. CAUDALLY DIRECTED EFFERENTS

A direct projection to the entorhinal cortex has been convincingly demonstrated by Hjorth-Simonsen (1971). Earlier studies (Adey, Sunderland, and Dunlop 1957, Votaw 1960*a*) had hinted at this pathway; according to Hjorth-Simonsen it originates in the CA3 field of primarily ventral hippocampus and terminates in layer 4 of the entorhinal area. Deadwyler, West, Cotman, and Lynch (1975) have confirmed the basic features of this pathway in an electrophysiological study. Long-latency potentials were evoked in all areas of dorsal entorhinal cortex following dorsal hippocampal CA3 stimulation. The authors suggest that this effect

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* Some support for this notion comes from the observation by McLennan and Miller (1974a) that two distinct potentials can be evoked by fimbrial stimulation: one in the dorsolateral septum, the other more ventral. The latter potential had a long latency and required more posterior fimbrial stimulation, suggesting that it arose in ventral hippocampus or subiculum.

** Chronister and White (1975) noted that, in Golgi preparations, the medial and lateral septum appear quite different in terms of dendritic arborization patterns.

was mediated by the longitudinal association bundle, accounting for the effect of dorsal stimulation upon an output presumably generated in the ventral hippocampus. A projection from CA1 to the subiculum has been reported (Andersen *et al.* 1973, Hjorth-Simonsen 1973). The most likely pattern of outputs from the hippocampus is schematized in Fig. 11. Note that we accept the Swanson and Cowan results in preference to those of Siegel.

3.5.3. PHYSIOLOGICAL ACTION OF HIPPOCAMPAL EFFERENTS

Physiological studies of the hippocampal efferents have been largely concerned with the role of the hippocampus in the control of sensory inputs. These include studies on the effects of hippocampal stimulation on sensory-evoked potentials, as well as studies correlating changes in hippocampal activity with changes in evoked potentials. Appendix Table A3 lists these studies. There is good, though not total, agreement that hippocampal stimulation enhances one or more components of the evoked potential in many areas. Effects of hippocampal stimulation have been found in the olfactory bulb, the primary and secondary visual and auditory cortex, several specific sensory thalamic nuclei, mid-line thalamic nuclei, the hypothalamus, and the cerebellum. Several studies have investigated the mechanisms involved in these effects, and the consensus is that one pathway involves the projection through the fornix to anterior and mid-line thalamus and to reticular formation. The enhancement of the late components of the visual evoked potential appears similar to that produced by mid-brain reticular stimulation (Sierra and Fuster 1968). Stimulation of the hippocampus in cerveau isolé cat (in which the influence of structures caudal to the mesencephalon is eliminated) enhanced the visual cortical evoked potential, but similar stimulation in an encéphale isolé preparation (with rostral-caudal connections intact) resulted in a decreased evoked potential (Redding 1967). In this study reticular stimulation enhanced evoked potentials in both preparations.

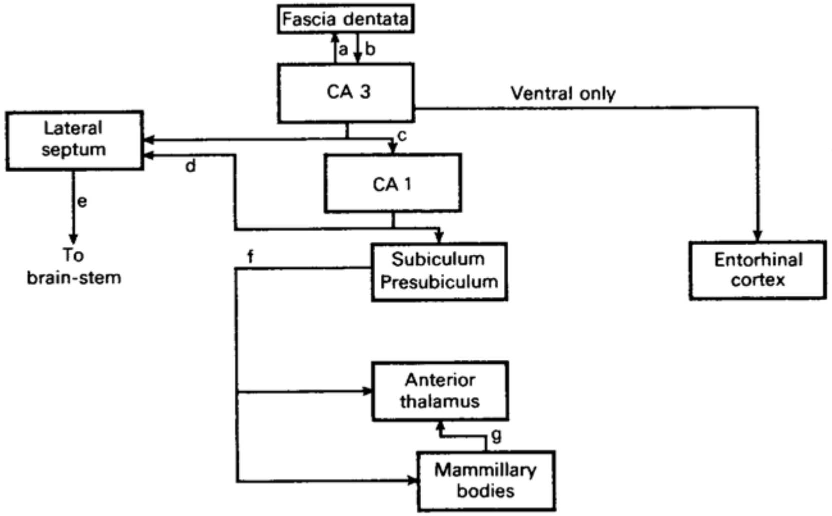

FIG. 11. Schematic representation of the efferent connections of the hippocampus: a, Zimmer path; b, mossy fibres; c, Schaffer collaterals; d, pre-commissural fornix; e, medial forebrain bundle; f, post-commissural fornix; g, mammillo-thalamic tract.

These findings indicate that the hippocampal effect is a mixed one, being predominantly excitatory via rostral thalamic structures and predominantly inhibitory via more caudal reticular structures. A subsequent study demonstrated that these effects were mediated by the fornix (Redding 1969). Lesions of the mid-line thalamic nuclei block the enhancement, while the locus of the effect appears to reside in the upper layers of the cortex (Parmeggiani and Rapisarda 1969), again suggesting an indirect effect through the reticular system. One study looking at the long-term effects of fornix lesions on the auditory evoked potential reported an enhancement (Ungher, Rogozea, and Sirian 1971). In a related type of study Pond and Schwartzbaum (1972), Schwartzbaum and Kreinick (1973), and Schwartzbaum (1975) found that the late components of the visual evoked potential were decreased during lever-pressing associated with hippocampal theta activity, as compared with non-theta related behaviours. We shall discuss the relationship of hippocampal slow waves to behaviour shortly.* Finally, one other study of the effects of hippocampal stimulation deserves mention, that of Cazard and Buser (1958, 1963). They reported an effect of extreme importance: long-term changes in visual and auditory evoked

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* A phenomenon which might be related to these controls over sensory input is the invasive hippocampal rhythm (IHR); this is a theta rhythm recorded from the cortex during periods of large amplitude, well-synchronized hippocampal theta. It has been reported to occur, for example, during the conditioned stimulus prior to the onset of an avoidance response (Pickenhain and Klingberg 1967, Fig. 5) or prior to a lever press (Yoshii, Miyamoto, and Shimokochi 1965, cited in Yamaguchi *et al.* 1967). Yamaguchi *et al.* have studied this phenomenon in curarized cats. It occurs primarily in occipital and temporal cortex (i.e. above the hippocampus) during high-amplitude, regular theta and is associated with a regularization and slight increase of the evoked potential. The authors maintain that the IHR is not due to volume conduction from the underlying hippocampus. They note that (1) the important feature for the appearance of cortical IHR is the regularity, not the amplitude, of the hippocampal theta, (2) the cortical distribution is limited by bipolar recording, and (3) there is sometimes a difference in frequency between the cortical and hippocampal rhythms. These reasons do not seem particularly compelling; to rule out volume conduction one must either have maps of the potential showing that it changes in a way consistent with a cortical, and not a hippocampal, generator and/or single neurone activity lawfully related to the potential. It is quite common for a penetrating electrode to record a cortical theta rhythm which is totally attributable to a generator in the hippocampus. Cortical units that fire in synchrony with the cortical theta rhythm have never been observed, although such 'theta' units are easily detected in the hippocampus (O'Keefe 1976, and unpublished observations). Thus, while not denying the validity of changes in cortical activity during hippocampal theta, it seems unlikely that a cortically generated theta rhythm has been demonstrated. These criticisms apply equally to those studies which have demonstrated theta rhythm in brain-stem studies without mapping the depth profile or recording single units (e.g. Le Moal and Cardo 1975). Winson (1974) has shown that theta recorded in the brain-stem is attributable to a generator in the hippocampus.

potentials following multiple pairings of visual and auditory inputs preceded by hippocampal stimulation. A tetanus to the hippocampus was usually followed after 45-60s by an increase in cortical evoked potentials, particularly in motor cortex; this increased to a maximum after several minutes. The phenomenon did not always occur, and in any particular acute rabbit could only be elicited two or three times. In chronic animals repeated elicitation of the effect over a period of several days resulted in an increased evoked potential to the sensory stimulus alone for several weeks. The investigators controlled for most of the obvious possible sources of artefact, including hippocampal seizures. It would be interesting to see if the pairing was essential, or if the phenomenon, like the facilitation seen in perforant path and Schaffer collateral synapses (see p. 125), is the result of repeated stimulation alone.

A few studies have looked at the effects of hippocampal stimulation upon motor activity, and these and other effects resulting from hippocampal stimulation, will be discussed at greater length in Chapter 12.

In summary, the hippocampus has extensive connections, either directly or indirectly through its connection to the subiculum, with other limbic nuclei and to sensory and motor areas in the brain-stem, thalamus, and entorhinal cortex. It can modify sensory inputs and effect motor outputs. In addition, it can probably mobilize the pituitary-adrenocortical steroid system under conditions specified later (see pp. 357-62).*

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* Perhaps the most widely known hypothesis linking the hippocampus with other brain areas, particularly limbic areas, is the Papez circuit (Papez 1937). Papez was impressed by the fact that emotions continued after the eliciting factor was removed and opined that this might depend on neural activity circulating within a limbic loop. Since he believed that the cingulate cortex was the cortical receptive area for the emotions, the circuit he proposed was as follows: cingulate cortex-cingulum-hippocampus-fornix-mammillary bodies-mammillothalamic tract-anterior thalamus-cingulate cortex. Parmeggiani, Azzaroni, and Lenzi (1971) have recently studied the transformation of repetitive activity through various parts of this circuit and suggested that it does, in fact, serve as a feedback circuit for the hippocampus. Some of the recent anatomical work cited in this chapter casts doubt on the existence of the circuit, at least in the form envisaged by Papez. The cingulate cortex does not project to the hippocampus, but the anterior thalamus does project to the presubiculum. The hippocampus does not project to the mammillary bodies directly, but does so by way of the subiculum. One new version of this circuit might be: anterior thalamus-presubiculum-mammillary bodies-anterior thalamus. Of course, there are countless circuits which one could imagine through these structures given the freedom of four or five synapses. At this stage of our understanding of the neural structures involved it may not be a very profitable exercise.

4

Physiology

THE present chapter will be concerned with the physiological functioning of the hippocampus and how this function can be understood in terms of the cognitive mapping theory. In the first section we shall consider the hippocampal electroencephalogram (EEG): its form, its underlying mechanisms, and its relation to the behaviour of the animal. In particular, we shall be concerned with *theta* activity, that rhythmic pattern in the hippocampal EEG which has captured the attention of many investigators. The various suggestions concerning its relation to behaviour will be discussed, and we shall propose a function for theta within the mapping system. Following this we shall consider in some detail the properties of single neurones in the hippocampus, primarily as investigated in experiments with freely moving animals. Our own work in this area will form the bulk of this discussion, which will attempt to define the elements in the hippocampus which comprise the mapping system. Finally, we shall propose a physiological model for the functioning of the cognitive map. Our intention here is not to provide a finely detailed, finished model of the mapping mechanism; we do not have sufficient information at present to draw a ‘wiring diagram’. However, it will be possible, on the basis of available information, to outline a model which would fulfill the functions of the cognitive map as we have described them. It remains for future research to fill in the details.

4.1. Origins of the hippocampal EEG

4.1.2. INTRODUCTION

The hippocampal EEG recorded from lower mammals such as the rat, rabbit, or cat can be classified into three distinct patterns* (see Fig. 12):

(1) A slow sinusoidal rhythm which normally ranges from 3 to 7 Hz in rabbit, dog, and cat, and 6 to 10 Hz in the rat and gerbil. This rhythm has been called *theta,* or rhythmical slow activity (RSA, Vanderwolf 1969).

(2) Large-amplitude slow waves in which the dominant frequency is slower than in theta and which, with some electrode placements,

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* We shall return to the problem of classification of the EEG patterns (pp. 162-3), where we shall discuss some of the pitfalls involved.

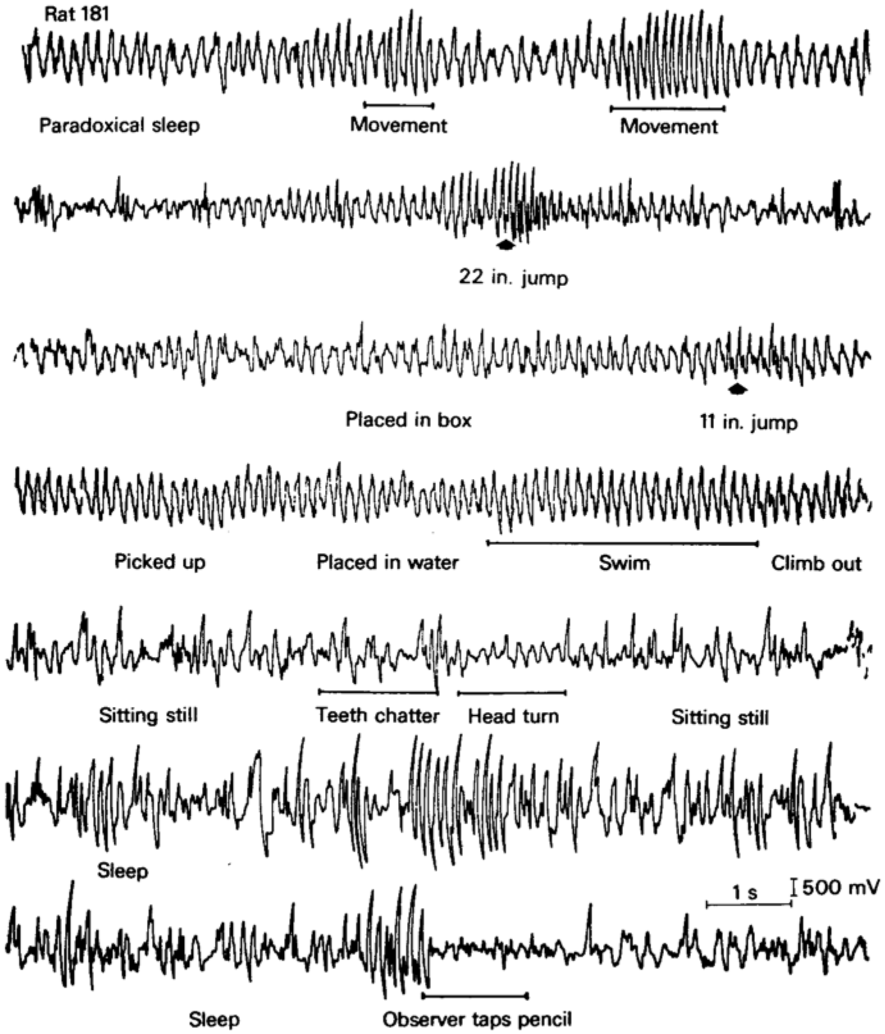

FIG. 12. Electrical activity at a single hippocampal site during sleep and various behaviours in the rat. Note the following: RSA during paradoxical sleep, struggling when held in the hand, swimming and head movement; large-amplitude irregular activity during sitting still while alert and while chattering the teeth; irregular slow activity and 'spindling' during slow-wave sleep and small-amplitude irregular activity when the rat was awakened but did not move about. Note also the following: increased RSA frequency and amplitude associated with twitching during paradoxical sleep and with jumping in avoidance tasks; different frequencies and amplitudes of RSA associated with head movements, swimming, jumping 11 in, and jumping 22 in. Calibration: 1 s, 500 µV; half-amplitude filters, 0.3 and 75 Hz. Electrode placement: CA1, hippocampus major. (From Whishaw and Vanderwolf 1973.)

contain sharp spikes of 50-100 ms duration. Vanderwolf (1969) has termed this state large irregular activity (LIA).

(3) A desynchronized, high-frequency, low-amplitude pattern which rarely lasts for more than a second or two in the rat, but can be more sustained in rabbit and cat. We shall follow Vanderwolf (1969) in calling this small irregular activity (SIA).

Single-unit activity recorded in both acute (e.g. Green and Arduini 1954) and chronic (O'Keefe and Dostrovsky 1971, O'Keefe 1976, Feder and Ranck 1973, Ranck 1973) animals shows a close relation to both theta and LIA (see below, pp. 196-201), and thus there is a prima facie reason to believe that these slow waves are providing information about underlying neural events. Whether they are actually part of the causal sequence of events, as suggested by some authors, is not clear. It seems unlikely that large extracellular voltage changes (up to 5 mV) would have no influence on neighbouring cells.

The hippocampal EEG can be related not only to neural mechanisms but also to the behaviour of the animal; thus, it serves as a useful bridge between the two. A considerable number of experiments has been carried out with the aim of identifying behavioural and/or psychological correlates of these EEG states, in particular theta activity, and these will be discussed in the next section. In this section we shall review what is known about the anatomical and physiological bases of the hippocampal EEG, starting with intra-hippocampal mechanisms and then going on to a discussion of the contribution of other brain areas. We shall conclude that the circuitry of the hippocampus is tuned to oscillate at theta frequencies and that these oscillations are normally driven by pulsed inputs from the medial septum and diagonal band of Broca. These latter areas, in turn, receive inputs from a wide variety of motor and other regions of the brain-stem. In addition, it is possible that the input from the hippocampus to the septum is itself important in triggering hippocampal theta activity.

4.2. Theta mechanisms within the hippocampus

The hippocampus contains neural circuitry which, when activated, tends to oscillate at the frequencies of hippocampal theta. Four lines of research provide information about this circuitry: (1) mapping studies of the distribution of theta within and across the hippocampus; (2) studies on the location and operation of inhibitory synapses within the hippocampus; (3) extracellular and (4) intracellular studies of hippocampal cells during theta.

4.2.1. THETA MAPPING STUDIES

The distribution of theta amplitude and phase has been measured by moving a recording electrode from the surface to the depth of the hippocampus, or by moving it from one part of the hippocampus to another. The first depth-mapping studies were done by Green and his colleagues in curarized rabbits (Green, Maxwell, and Petsche 1961, Green *et al.* 1960, Green and Petsche 1961). They compared potentials recorded by a microelectrode penetrating the CA1 field of one hippocampus with those recorded in the apical dendrites on the other side. The theta recorded from the microelectrode showed two maxima, one in phase with the control theta and located in the stratum oriens/pyramidale, and the other 180° out of phase and located deeper in the stratum lacunosum/moleculare of CA1. Reference to Fig. 13 will make clear what is meant by phase shifting in this context. The amplitude of the lower peak was approximately twice as large as that of the upper one in the basal dendrites.

The main findings of the Green *et al.* studies have been confirmed by Artemenko (1972) and Bland *et al*. (1975) in the curarized rabbit, and by Winson (1976*a,b*) in the freely moving rabbit. In Winson's study the profiles recorded during arousal-related theta and during movement-related theta were the same (see p. 166 for a discussion of the two types of theta). The only discrepancy in comparing these reports is that the later studies located the deeper peak in the buried blade of the fascia dentata and not in CA1.

Winson has also studied the depth profile in both curarized and freely moving rats (Winson 1974, 1976*a,b*). While the curarized rat is very similar to the rabbit, the freely moving rat shows an interesting difference. As in the other preparations there is a 180° phase difference between the theta recorded in the CA1 field and in the fascia dentata, but the transition between the two is different. Instead of the abrupt reversal in the stratum radiatum, there is a gradual phase shift occurring over a depth of 175-350 µm. (see Fig. 13). This figure also summarizes the amplitude data from several papers. It is clear that the maximum amplitude occurs in the fascia dentata. This finding, obtained with a microelectrode, conflicts with the many reports that very poor theta is recorded with a bipolar macroelectrode in the fascia dentata of the freely moving rat. Whishaw and Vanderwolf (1973) have mapped theta in the urethane-anaesthetized rat and found the maximum amplitude in the CA1 apical dendrites, with a much smaller amplitude in the fascia dentata. This finding could be explained as follows. When one tip of the bipolar electrode is at the hippocampal fissure and the other near the soma of the CA1 pyramids, the theta recorded from one is 180° out of phase with that recorded from the other and thus the signal will be doubled. On the other hand, when both tips are below the hippocampal fissure, the theta signals are in phase and thus subtracted, yielding a much smaller resultant. When either electrode is in the apical dendrites of CA1, where the signal is phase shifted an intermediate amount, the theta signals will interfere and primarily higher frequencies (and poor theta) will be the resultant.*

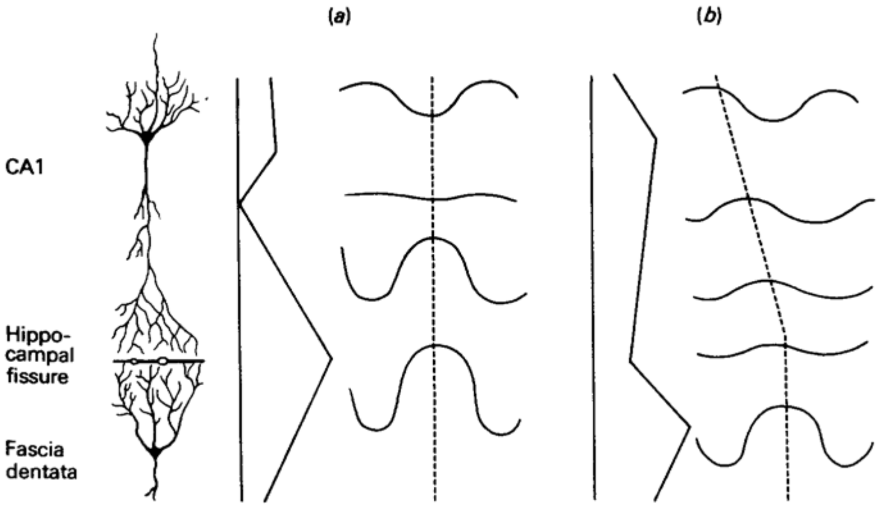

FIG. 13. Changes of theta amplitude and phase at different depths in the hippocampus of different preparations: (a) rabbit and curarized rat; (b) the freely moving rat. In each panel the left graph shows relative amplitude on the abscissa, and the right shows a single theta wave at each level and the phase relations amongst them. (After Winson 1976*c*, Fig. 1.)

We can conclude from the above studies (in particular Winson's) that there is a clear species difference in the distribution of theta. While curare has no effects on the rabbit profile, it markedly changes that of the rat. Winson has suggested that differences might exist in the septal cholinergic afferents to the hippocampus in the two species. Both muscarinic and nicotinic synapses have been identified on pyramidal cells (Bland et al. 1974, Bird and Aghajanian 1975), and there is good evidence that at least some of the fibres coming from the septum are muscarinic (Yamamura and Snyder 1974). Whether the nicotinic fibres are also of septal origin is not yet known.

The relationship between theta amplitude and phase at different points across the surface of the hippocampus has also been mapped. In early studies on curarized rabbits Petsche and Stumpf (1960, 1962) simultaneously recorded the EEG from many points on the hippocampal surface and reported a systematic phase shift as one went from rostral to caudal, or from medial to lateral, hippocampus. The theta waves acted as though they originated in the septum and travelled caudally and laterally at an average speed of 30-40 cm s^{-1}. Thus, points 8 mm apart would be phase shifted 20-25 ms, or 36°-40° for a 5 Hz theta frequency. Higher-frequency

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* An additional factor which must be kept in mind is that urethane abolishes the movement-related theta; Whishaw and Vanderwolf are thus mapping only the arousal-related theta. While there is no difference in the amplitude plot of these two types of theta in the rabbit (Winson 1976*a,b*), there might be in the rat.

waves travelled faster. That is, the phase relations between different electrodes were held constant in spite of frequency changes. Adey, Dunlop, and Hendrix (1960) reported that early in a learning task theta from the dorsal hippocampal electrode led that from the entorhinal cortex by 20-35 ms, but lagged by up to 65 ms later in training (see below for a fuller discussion of the relationship of theta to learning, pp. 186-9).

A recent experiment on the urethane-anaesthetized rabbit (Bland *et* al. 1975) appears to contradict these results. They could find no phase shift at all between widely spread points on the hippocampal surface. In their experiment large areas of the hippocampus were oscillating in synchrony. Another major discrepancy between the Petsche and Stumpf and the Bland *et* al. studies concerned theta in the CA3 field. Whereas in the former study this had been the locus of the highest-amplitude theta, Bland *et* al. could find no theta here at all. Again, we find major discrepancies between studies, and although the Bland *et* al. study is the more convincing of the two, the Petsche and Stumpf study may not be inaccurate; a reason for the discrepancy might be the existence of two or more theta systems which are differentially sensitive to anaesthetics and curarizing agents.

4.2.2. INHIBITORY CIRCUITS IN THE HIPPOCAMPUS

Part of our understanding of the intra-hippocampal mechanisms involved in theta comes from electrophysiological studies of inhibitory circuits within the hippocampus; these have been summarized already (see pp. 117-118). Recapitulating briefly, a series of studies by Kandel and Spencer (Kandel and Spencer 1961a,b, Spencer and Kandel 1961a,b,c, Kandel *et al.* 1961) showed that inhibitory potentials in pyramidal cells were mediated by a direct axon collateral of these cells and perhaps one interposed interneurone. This conclusion was confirmed and extended in studies (Andersen and Eccles 1962, Andersen *et al.* 1964 a,b) indicating that a widespread inhibition is mediated via an axon collateral onto a basket cell, which projected in turn onto many pyramidal cells. A similar conclusion has been reached by Horowitz and his colleagues on the basis of potential maps recorded from the hippocampus following single shocks to the fornix (Horowitz 1972, Horowitz, Freeman, and Stoll 1973). The inhibitory transmitter is probably GABA (Stefanis 1964, Curtis, Felix, and McLennan 1971).

Evidence for an inhibitory process in the apical dendrites as well as in the cell-body layer comes from the work of Fujita and Nakamura (1961). Following stimulation at the level of stratum oriens and pyramidale of CA1, they recorded a large positive wave in the apical dendrites. Since this wave inhibited the dendritic spike produced by stimulation of the Schaffer collaterals of the CA3 pyramids, they considered it to be an active IPSP in the dendrites. As they point out, because of the laminated pattern of the afferents, a selective inhibition of the dendrites would block some of those afferents while leaving the cell accessible to others closer to the soma. Alternatively, the inhibition might act on some dendrites but not on others. There is evidence, then, for inhibitory processes originating from the stratum oriens and stratum pyramidale, and acting on both cell bodies and dendrites of the pyramidal cells.

4.2.3. EXTRACELLULAR RECORDINGS FROM HIPPOCAMPAL NEURONES DURING THETA

In studies of both paralysed (Green *et al.* 1960, von Euler and Green 1960a,b, Macadar *et al.* 1970) and freely moving (O'Keefe and Dostrovsky 1971, Ranck 1973, Feder and Ranck 1973, O'Keefe 1976) animals, hippocampal units have been found which fire in synchrony with the hippocampal theta waves. The chronic studies report two classes of unit, both related to theta but in a different way. The first type of unit bears such a close relation to theta in the EEG that Ranck has called it a 'theta' unit. These cells increase their firing rate whenever a theta rhythm appears and usually fire in bursts, with each burst locked to a particular phase of the theta cycle. Figs. 20 and 21 show examples of two of these units, one with a good phase relation to the theta waves and the other with a poor one. Fox and Ranck (1975) have mapped the distribution of these theta units in the freely moving rat and find that it is very different from the distribution of the second class of units, the 'complex spike' units. These latter are almost always found in the layer containing the projection cell somas, the stratum pyramidale and the dentate granule layer. While a small number of theta units are also found in these strata, the majority are found outside them: in CA1, in the stratum oriens; in CA3, in the stratum lucidum/radiatum and moleculare; in fascia dentata, within the hilus. This distribution matches very closely the histological distribution of interneurones; this has led Fox and Ranck to suggest that the theta units might be the basket cells of Cajal (see pp. 108-11). There is, as yet, no direct evidence for this identification.*

An alternative possibility which must be kept in mind is that these theta units represent the activity of specialized terminals of the septal afferents (Frederickson, personal communication). In our own studies (O'Keefe 1976) we have found that some of these units fire on the positive-negative phase of the theta wave while others fire on the negative-positive phase. It is unlikely that both types are inhibitory interneurones acting on the pyramidal cell soma. Possibly one type is excitatory or, if inhibitory, is acting on the apical dendrites and not the soma. Since only type-II flattened vesicle synapses have been found on the soma (Andersen *et al.* 1964b), the latter possibility seems more probable.

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* Antidromic activation of complex spike units, but not theta units, from stimulation of hippocampal efferents would constitute direct evidence, as would intracellular dye injections into the two types of unit. A fuller description of the two different types of unit recorded from the hippocampus of the freely-moving rat can be found on pp. 196-211.

The second type of hippocampal unit, the complex spike unit, only fires in phase with theta under certain circumstances. The phasing of these units to theta appears to be gated by some extrinsic mechanism, while the first type of unit is more directly related to theta itself. These units are located in the stratum pyramidale (Fox and Ranck 1975) and are probably pyramidal cells.

4.2.4. INTRACELLULAR RECORDINGS FROM PYRAMIDAL CELLS DURING THETA

A more direct investigation of the physiological basis of the theta rhythm was undertaken by Fujita and Sato (1964). They recorded intracellularly from rabbit CAI cells during theta and found, in 61/71 cells, an intracellular theta rhythm which was in phase with the gross extracellular theta. Many of the cells fired during the depolarizing phase of the intracellular theta. Evidence from experiments in which ions were injected or allowed to diffuse from the recording micropipette indicated that at least part of the mechanism producing the intracellular theta was an EPSP drive. The authors conclude that both EPSP's and IPSP's are probably involved.

A similar conclusion was reached by Artemenko (1972) on the basis of the intracellular study of two different types of cell: *A* cells were inhibited by sciatic or contralateral hippocampal stimulation, were depolarized and fired during the positive phase of extracellular theta, and were hyperpolarized during extracellular theta negativity; *B* (or basket) cells were far fewer in number than were the *A* cells, and were often activated by external stimulation and fired during the extracellular theta negativity. According to Artemenko, extracellular theta recorded from the pyramidal cell area would have two components, the negative phase consisting primarily of EPSP's of the basal and proximal apical dendrites of the basket cells and the positive phase due to the subsequent hyperpolarization of the pyramidal cell soma and dendrites.

4.2.5. CONCLUSION

It appears that one of the functions of hippocampal theta is to maintain large areas of the hippocampus (and perhaps other parts of the brain as well) in the same, or related, phases of excitability. There are two plausible mechanisms which might be responsible for this high degree of phasing: intra-hippocampal coupling amongst the pyramidal cells and/or an extra hippocampal generator wired to drive the different parts of the hippocampus with the appropriate time relations.

Intra-hippocampal coupling could exist either through direct electrical coupling of the dendrites of contiguous cells, or less directly through axon collaterals from one pyramid to another or through an interposed interneurone. There is no evidence for electrical coupling of dendrites during normal hippocampal function, although Purpura *et al.* (1966) have suggested that such coupling might occur during seizures. There is no evidence as yet of the gap junctions between hippocampal dendrites which would be required for this coupling. Excitatory collaterals between neighbouring pyramids probably exist in small numbers and are usually masked in physiological studies by the overwhelming inhibition which follows an afferent volley. It is this inhibition which has been the focus for speculation on the physiological basis of the coupling between hippocampal pyramids (pp. 146-7).

There is general agreement that synchronous activation of pyramidal cells is followed by widespread inhibition which is mediated by a class of interneurones, probably the basket cell of Cajal. Following this inhibition of the pyramidal cells, there is a rebound excitatory phase during which the probability of firing is higher. In Horowitz's experiment on the cat (Horowitz 1972), the time course of this oscillation was too short to provide a 'natural resonant frequency' in the theta range, but this may have been due to the specific conditions of the experiment. In other experiments the post-excitatory inhibition can last up to several hundred milliseconds (e.g. Andersen *et al.* 1964a,b). Horowitz suggests that the rebound excitation is due to a reduction in firing rate of the inhibitory interneurones which results from the temporary loss of afferent drive from the (inhibited) pyramidal cells. Alternatively, the oscillation may be due to inherent membrane properties of the pyramidal cells. In addition to inhibition of the soma of pyramidal and granule cells, there is also probably inhibition of the dendrites during part of the theta cycle. Whether this is mediated by the basket cells or by some other group of interneurones is not known. Winson's mapping studies suggest that, in the rat at least, part of the dendrite is inhibited while, at the same time, another part is in an excitable state. One reason why theta activity is seen in CA1 and fascia dentata, and not in CA3, may be the location of the inhibitory interneurones. In CA1 and fascia dentata these are located in stratum oriens and the polymorphe zone, far away from the main zone of inhibition in the apical dendrites, such that the oscillating dipoles set up by the depolarization of the interneurone soma and the hyperpolarization of the projection cell soma and apical dendrites would add to each other. In CA3, on the other hand, the interneurone soma is located in the apical dendritic region and the dipoles set up by the interneurones and the projection cells would largely cancel each other out.

The oscillations of the intrinsic hippocampal circuitry within this natural frequency band are synchronized by an external generator in the medial septum/DBB. The septal afferents from this generator probably make direct synaptic connections with the basket cells as well as influencing them indirectly via the pyramidal and granule cell collaterals. In a subsequent section we shall discuss this generator and its inputs in greater detail.

4.3. Large-amplitude irregular EEG activity (LIA)

A second characteristic EEG pattern recorded from the hippocampus of animals such as the rat and cat is an irregular large-amplitude wave with dominant frequencies lower than that of theta (see Fig. 12). In some electrode placements in the rat, LIA features a large primarily negative spike of duration 50-100 ms. As we shall see in a subsequent section, the

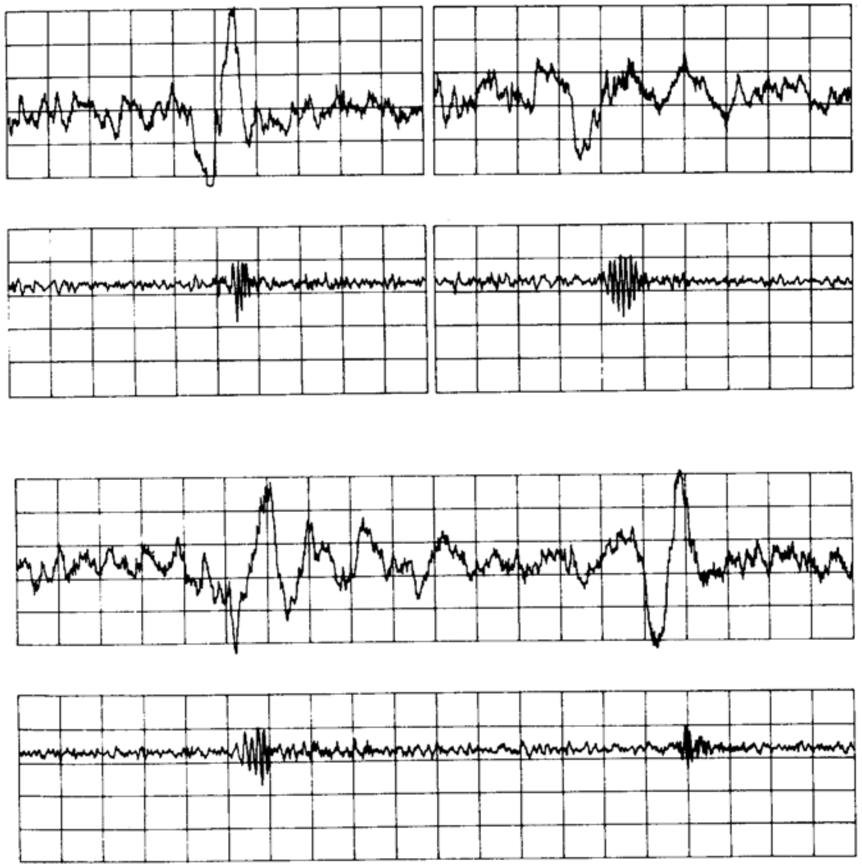

FIG. 14. Examples of hippocampal ripples and their relationship to the EEG in the freely moving rat. The ripples were recorded by a microelectrode in the pyramidal layer of CA1 (bandwidth 80-800 Hz), while the EEG slow waves were recorded by another microelectrode 300 μm posterior and 175 μm below the pyramidal layer in the apical dendrites of CA1 (bandwidth 0.6 Hz-6 kHz). Calibrations: each division is 50 μV for the ripples and 73 μV for the EEG slow waves. Time: each division is 50 ms. (From Dostrovsky and O'Keefe, unpublished.)

LIA pattern only occurs during automatic, non-displacement behaviours such as sitting quiet, slow-wave sleep, eating, drinking, and grooming. The EEG spikes occur most frequently during slow-wave sleep and quiet sitting, less frequently during eating and drinking, and least of all during grooming. They are often inhibited by arousing stimuli, even when no movement occurs.

The large negative slow wave is intimately associated with two other neural events: a sinusoidal ripple consisting of 4-10 waves with periods of 4-8 ms (see Fig. 14), and a burst of firing in the theta[*] units located in the stratum pyramidale and oriens. Less frequently there are a few spikes from other units. Fig. 15 shows an example of an LIA spike associated with bursts in both a theta unit and complex spike units. In many of these non-'theta' units this firing during the LIA spike is the only activity recorded for many hours.

In our laboratory (Dostrovsky and O'Keefe, unpublished) we have

FIG. 15. Example of a smaller theta unit and larger complex spike units bursting in relation to the slow-wave EEG spike from the same microelectrode. Upper trace, bandwidth 600 Hz - 6 kHz; lower trace, 0-6 Hz - 6 kHz. Up is positive in both traces. Time calibration, 100 ms. (From O'Keefe, unpublished.)

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[*] See pp. 196-201 for a description of the units in the hippocampus of the freely moving rat. Molnar, Arutyunov, and Narikashvili (1971) and Molnar (1973) have recorded spike bursts in the CA3 stratum oriens of the paralysed cat which probably represent the same phenomenon.

recorded the changes in amplitude of both the EEG spike and the ripples with depth in CA1. The maximum amplitude of the ripples occurs in the stratum pyramidale, or just below it, while the maximum amplitude of the EEG spike occurs several hundred microns deeper in the stratum radiatum. The EEG spike is positive or positive/negative in the stratum oriens and negative or negative/positive in the stratum radiatum. The inversion point usually occurs within the stratum pyramidale.

The nature of the causal relationships between the EEG spike, the ripples, and the burst of spikes in the theta units are not clear at this time. Standard volume-conduction theory suggests that the EEG spike represents an active depolarization of the apical dendrites, perhaps via a synaptic input. Whether this 'EPSP' is the cause or consequence of the bursting in the theta units cannot be said. The ripples may be summated abortive spikes either in the proximal dendrites or in the soma of the pyramidal cells. They look very similar to the abortive spikes recorded by Yamamoto (1972) and Ogata (1975) in *in vitro* hippocampal slices bathed in a chloride-free medium.

Andersen (1960a) has provided evidence for the occurrence of dendritic spikes in the apical dendrites of CA1 neurones from extracellular records. He showed that the onset of the spike elicited in the apical dendrites by stimulation of various pathways had a shorter latency in the stratum lucidum than above or below this point and that it did not reverse its polarity within the hippocampus. Spencer and Kandel (1961b) also found evidence for non-electrotonic propagation of activity into the soma from the dendrites. A large number of their spikes arose directly from a fast prepotential (FPP) without a preceding EPSP. Hyperpolarization of the soma sometimes exaggerated the break in the inflection between the FPP and spike or left only the FPP component without the spike. They concluded that the FPP is a dendritic spike as seen from the soma. Other studies which have provided evidence for dendritic spikes in hippocampal pyramidal neurones are Broggi and Purpura (1969), Cragg and Hamlyn (1955), von Euler, Green, and Ricci (1958), Fujita and Nakamura (1961), Fujita and Sakata (1962), Gloor, Sperti, and Vera (1963), Gloor, Vera, and Sperti (1963), Purpura (1967), Purpura *et* al. (1966), Renshaw, Forbes, and Morison (1940). Some of these studies show pictures of putative dendritic spikes which resemble the ripples in Fig. 14. Fujita and Nakamura (1961) demonstrated that a dendritic spike could be inhibited by a dendritic IPSP generated by electrical stimulation of stratum oriens and pyramidale (see p. 146).

From the physiological point of view LIA appears to represent a state during which the theta mechanism is inactive. Theta, on this view, represents an active inhibitory process which locks off the soma and dendrites of most of the pyramidal cells, allowing only a few to fire at any given time. In its absence, there is a synchronous activation of the dendrites of large numbers of pyramidal cells, although in most cells this activation fails to trigger a full soma spike. Several reasons can be offered for adopting this view.

(1) There is always a period of at least a few seconds between the onset of the behaviour related to LIA, such as lying down, and the onset of the EEG spike and ripples, suggesting the release from an active phenomenon.

(2) Place units,* whose place fields differ when the animal is active and thus never fire synchronously, will sometimes fire together on a ripple, recalling the synchronizing effect of the neocortical slow waves during slow-wave sleep. It would seem unlikely that much information could be conveyed by this synchronous activity.

(3) Lesions of the fornix at the level of the ventral hippocampal commissure do not decrease the frequency or amplitude of the ripples; if anything they are increased (O'Keefe, unpublished observation).

Before turning to a discussion of SIA, we should mention one possible source of confusion between LIA and theta. Sometimes during LIA several EEG spikes occur rapidly in succession and resemble a short burst of high-frequency theta. There is no way of distinguishing between the two from the low-frequency EEG alone, but the presence of ripples on the LIA spike makes it possible to do so at higher frequencies.

4.4. Small-amplitude irregular EEG activity (SIA)

The desynchronized arousal pattern (SIA of Vanderwolf 1969) is sometimes seen in the rat hippocampal EEG, but only for a short period. It is more common in the cat and rabbit. In several studies (Anchel and Lindsley 1972, Brugge 1965, Stumpf 1965a, Parmeggiani 1967, our own unpublished records) lesions of the septum or fornix have been shown to have no effect on this desynchronized activation of the hippocampus. Thus, Stumpf (1965b) suggested that it was mediated by fibres entering through the entorhinal cortex. On the other hand, stimulation experiments which have studied the brain-stem pathways responsible for this pattern suggest that the pathway runs from the dorsolateral tegmentum through the lateral hypothalamus (MFB) and lateral preoptic area. Furthermore, high-frequency stimulation of the septum and fornix will also produce SIA. These findings suggest that there might be two SIA patterns identical in their EEG waveform but differing in their underlying physiological basis. One SIA would reflect an inhibition of the theta mechanism (with perhaps a cessation of firing in theta units) and would be mediated by the entorhinal pathway. The other SIA might reflect an overactivation of the theta system resulting in continuous hyperpolarization block of pyramidal dendrites and a loss of slow-wave amplitude. The theta-related units would

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* See pp. 196-201 for a description of the different types of hippocampal unit.

fire at a high uniform rate during this SIA which could be considered as one end of the theta continuum. There is no way of distinguishing between these two SIA patterns without resorting to unit recording.

4.5. External circuits involved in hippocampal theta and desynchronization

In one of the earliest studies on hippocampal theta Green and Arduini (1954) found that it depended on the integrity of a neural circuit originating in the mesencephalic reticular formation, extending through the hypothalamus to the septum, and thence via the fornix to the hippocampus. Subsequent studies have traced this circuit in greater detail, focusing in particular on the role of the septum as a pacemaker for theta. Attempts to implicate areas outside this basic circuit have been less successful, although, as we shall see at the end of the section, there is some evidence for the involvement of the medial thalamus.

4.5.1. BRAIN-STEM STRUCTURES INVOLVED IN HIPPOCAMPAL THETA AND DESYNCHRONIZATION

The general consensus of those studies (see Table A4) which have tried to delineate the brain-stem pathways involved in the production and regulation of the hippocampal EEG is that there are two principal systems, a medial theta system and a more lateral desynchronizing (SIA) one. The theta system arises in at least five different areas: nucleus pontis reticularis oralis, locus coeruleus, the large cell area of pontine tegmentum, portions of the mid-brain tegmentum, and the ventrolateral periaqueductal gray (Macadar, Chalupa, and Lindsley 1974). The fibres from the more caudal areas gather together in the region of the mid-brain reticular formation and follow a medial course rostrally through the posterior hypothalamus, ventromedial hypothalamus, medial preoptic area, and into the medial septum. From there, they reach the hippocampus via the fornix-fimbria pathway. Electrical stimulation along this pathway produces theta; lesions along this pathway eliminate the theta produced by stimulation of more caudal brain sites or peripheral nerves as well as that occurring naturally in the freely moving animal (Green and Arduini 1954, Mayer and Stumpf 1958, Jouvet and Mounier 1962, Corazza and Parmeggiani 1963, Brugge 1965, Stumpf 1965b, Donovick 1968, Gray 1971, Anchel and Lindsley 1972). There are several theoretically important facts concerning the relationship between electrical stimulation of brain-stem sites and the hippocampal theta it produces (Stumpf and his colleagues, cited in Stumpf 1965b, Macadar *et al.* 1974). The same voltage and frequency of stimulation in different brain-stem areas yields different frequencies of hippocampal theta. For example, stimulation in the periaqueductal gray yields 3-5 Hz theta, while the same stimulation applied to reticularis pontis oralis gives 5 Hz. At any given site of stimulation, increases in *voltage* produce increases in the initial frequency of theta; after the initial response theta settles down to much the same frequency regardless of the stimulating voltage.* Increases in stimulus strength also prolong the length of time theta continues after stimulus offset. On the other hand, changes in the *frequency* of stimulation above the threshold rate (usually c. 50 Hz) have no effect on theta frequency but are sometimes reported to yield decreases in theta amplitude. With some medial placements stimulation above a certain voltage causes the hippocampal response to switch from theta to desynchronization, or a mixture of the two. This latter effect may be due to current spread to the more lateral desynchronizing system. We shall discuss this lateral system next, and then return to the role of the medial septum in the production of theta.

Electrical stimulation at sites lateral to the medial theta system results in desynchronization (Table A4). Arising in the raphe nucleus and nucleus reticularis pontis caudalis (Macadar *et* al. 1974), this system runs through the mesencephalon in close proximity to the medial theta system and then diverges to course more laterally through the hypothalamus, probably as part of the medial forebrain bundle (Anchel and Lindsley 1972). The threshold voltage for activation of SIA is usually higher than that needed for the production of theta from the medial pathway. Lesions of the lateral pathway eliminate the SIA pattern elicited by more caudal stimulation. Although high-frequency stimulation of the septum can produce the SIA pattern, SIA is still present in animals with septal (Brugge 1965, Stumpf 1965a) or fornix (Anchel and Lindsley 1972) lesions. Stumpf (1965a) has suggested that SIA might be mediated by the entorhinal input to the hippocampus; however, we have already seen that the serotonergic lateral pathway from the raphe nucleus enters the hippocampus via the fimbria-fornix system (pp. 126-7). The possibility that there might be two types of SIA has already been noted (pp. 153-4)

Paiva, Lopes da Silva, and Mollevanger (1976) have argued against the separation between medial synchronizing and lateral desynchronizing systems. In their studies, performed on awake, unrestrained cats, stimulation in both systems elicited synchronization, with desynchronization only resulting from more intense stimulation; they did, however, replicate the finding that voltage increases lead to frequency increases in the elicited theta. On the other hand, Coleman and Lindsley (1975) and Lindsley and Wilson (1975) present strong supporting evidence for the existence of two separate systems. As in the earlier work, these studies on freely moving cats showed that medial stimulation elicited synchronization and lateral stimulation elicited desynchronization. Further, close correlations were observed between the behaviour of the animal and its hippocampal EEG

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* Hippocampal theta during movement shows a similar effect; during jumping or the initiation of running there is a good correlation between theta frequency and velocity of the movement. This effect diminishes or disappears with continued movement (see p. 179).

(see below), and between the behavioural and EEG effects of brain stem stimulation. Finally, Lindsley and Wilson discuss work showing that stimulation in the two systems has opposed effects on cells in the medial septum, in accordance with the notion that medial stimulation drives cells in bursting patterns which then mediate theta, while lateral stimulation disrupts the bursting pattern of these cells and thus elicits hippocampal SIA. The apparent discrepancy between Paiva *et al.* (1976) and other work might relate to the fact that they defined desynchronization in a restrictive manner, as a flat frequency spectrum. Thus a marked decrease in EEG amplitude would not count as desynchronization if there was a peak in the theta range. The discrepancies in behaviour observed to be elicited by medial and lateral stimulation are not so easily dismissed. While Coleman and Lindsley found that medial stimulation elicits behaviours typically associated with hippocampal theta, such as orienting and exploration, and lateral stimulation elicits a brief orientation followed by fixated attention, Paiva *et al.* failed to observe any difference in the behavioural effects of the two types of stimulation. Aside from noting the different electrode placements used in these studies, we can offer no explanation for the discrepancy they present.

4.5.2. OTHER THETA PATHWAYS

In addition to the brain-stem–septal pathway, it has been suggested that the medial thalamus and the entorhinal cortex might be involved in the generation of hippocampal theta. Eidelberg, White, and Brazier (1959) found that medial thalamic lesions abolished hippocampal theta elicited by natural stimuli or tegmental stimulation in the rabbit. Other studies have found a reduction in theta (Kawamura, Nakamura, and Tokizane 1961, Azzaroni and Parmeggiani 1968, Corazza and Parmeggiani 1963). On the other hand, Green and Arduini (1954) failed to find any effect of thalamic lesions. Azzaroni and Parmeggiani (1968) have also reported that hippocampal theta could be elicited by brief bursts of electrical stimulation to the anterior thalamus and the cingulum bundle. Although there are no known fibres from the anterior or medial thalamus to the septum, there are substantial projections from both the septum and the subiculum to the anterior thalamus (see p. 135); the possibility of retrograde effects must be kept in mind. Azzaroni and Parmeggiani (1968) suggest that the role of the thalamic–cingulum pathway is regulatory rather than one of direct elicitation of theta. An alternative possibility is that theta is not a unitary phenomenon and that different thetas with different frequency ranges, are generated from different brain-stem or thalamic areas. We shall discuss this notion in greater detail in the section on the behavioural correlates of hippocampal theta.

The involvement of the entorhinal area in the generation of hippocampal theta is more controversial. While many studies have found no effect of lesions of this area, two report the abolition of theta following such lesions (Carreras, Macci, Angeleri, and Urbani 1955, cited in Stumpf 1965b; Chronister *et al.* 1974). The findings of this latter study are particularly suspect. It is not clear that their hippocampal EEG contained very much theta activity. The slow time-base used in the records shown makes it difficult to tell, but the presence of the large-amplitude spikes characteristic of LIA are clear in several records. The electrodes were placed in the CA3-4/F.D. area, where uncontaminated theta is not easily recorded with large electrodes, while the behavioural testing stiuation was conducive to sleep, a state when LIA predominates: the rats were habituated to a small, uninteresting box and recordings were only taken for the last 5 of 20 min in the box. The restriction of the loss of theta to the same lamella as the lesion suggests that the effect may be vascular. It is imperative that in these types of lesion studies controls be employed to show no change in LIA or SIA. We can conclude, then, that although the entorhinal area might be involved in the generation of hippocampal theta the evidence is not very strong at present.

4.5.3. THE PACEMAKER ROLE OF THE MEDIAL SEPTUM

Why do increases in the stimulus strength applied to the brain-stem theta pathway result in increased frequencies of the initial theta? There must be a mechanism somewhere in the pathway for converting change in total neural activity into variations in theta frequency. Since the conversion occurs with stimulation applied to points along the pathway up to but not including the septum, there is *prima facie* evidence that the transformation occurs there. This position is most clearly associated with the Viennese group of Petsche and Stumpf and is outlined in Fig. 16(a). It states that circuitry within the medial septum and diagonal band of Broca oscillates at the theta frequency when stimulated by a continuous afferent barrage. The pulsing output of this circuit is then transmitted to the hippocampus where it paces the hippocampal cells at the theta frequency. Other possibilities which must be kept in mind are that the pacemaker is located (1) in the brain-stem (Fig. 16(b)) or (2) in the hippocampus itself (Fig. 16(c)). A variant on this last model states that theta arises from a loop involving both the hippocampus and septum (hippocampus →lateral septum→medial septum→hippocampus) (McLennan and Miller 1974a, 1976, Calvert and Miller 1975). Let us examine some of the available data to see how good a fit each model provides.

The first important piece of information is the existence of units in the medial septum and diagonal band of Broca which burst in phase with the hippocampal theta waves in a fashion similar to the hippocampal theta-related units (Gogolak *et al.* 1967, 1968, Apostol and Creutzfeldt 1974, Macadar *et al.* 1970, Morales *et al.* 1971, Petsche, Gogolak, and van Zweiten 1965, Petsche, Stumpf, and Gogolak 1962, Ranck 1975b, Lindsley

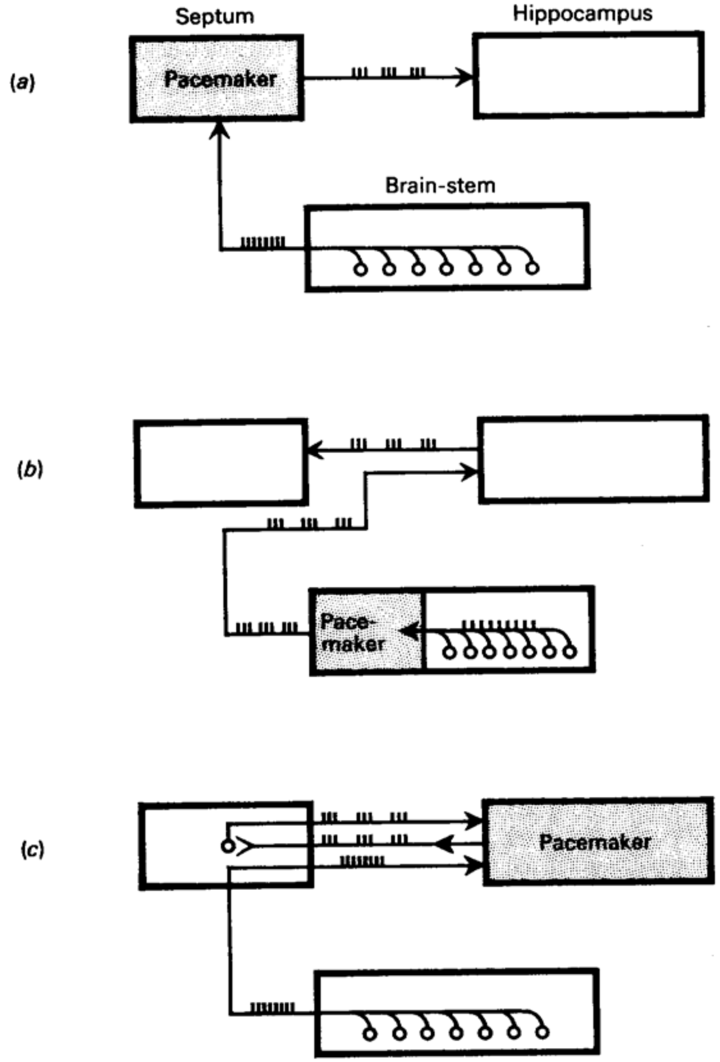

FIG. 16. Three models of theta generation: (a) septal pacemaker; (b) brain-stem pacemaker; (c) septo-hippocampal interaction.

and Wilson 1975). According to Petsche et al. (1965) groups of units within a 300 μm diameter area burst in synchrony with the same phase relationship to hippocampal theta, but each group is related to a different part of the theta wave. All cells in the dorsal diagonal band of Broca either fired in the theta mode spontaneously or could be provoked to do so by reticular stimulation or eserine. Interestingly, there was no uniform effect of reticular stimulation on unit firing rate, some units increasing their frequency, others showing a decrease (Gogolak *et al.* 1967). Similarly, there was no consistent relationship between theta frequency and unit firing rate. Increases in theta frequency were associated with increased rates in some units, but decreases in others. Aside from the constant phase relation of the onset of the septal unit burst, the only other reliable relationship was that between theta frequency and unit burst duration. Length of burst duration was inversely correlated with theta frequency, so that, in addition to starting at the same phase in the theta cycle, the burst would cease at the same phase irrespective of theta frequency.

Komisaruk (1971) has recorded theta-related units in the lateral hypothalamus of rats, and Harper (1973) has found similar units in the thalamus. Since in the first study lesions placed between the septum and lateral hypothalamus abolished hypothalamic theta but not septal theta, it is likely that the former was driven by the latter, and not *vice versa,* as suggested by the model of Fig. 16(b). The consistent finding that lesions of the medial septum abolish theta (see p. 154) would seem to make postulation of a pacemaker in the hippocampus itself unlikely, since it should still be activated by other inputs.

The other possibility to be considered is the model of McLennan and Miller (1974a,b, 1976) which proposes that theta results from an interaction between the septum and the hippocampus (Fig. 16(c)). This model would predict the existence of theta-related units in both the septum and hippocampus and the abolition of hippocampal theta following a septal lesion. It would also suggest that a fimbria/fornix lesion should abolish the rhythmicity of the medial septal units, and this result has been reported by McLennan and Miller in urethane-anaesthetized rats. They also found that single shocks at a frequency up to 6 or 7 Hz produced an activation/inhibition response in lateral septal neurones in their acute experiments. In contrast, stimulation at higher rates (7-12 Hz) caused a selective loss of the inhibitory component and a concurrent loss of rhythmicity in the medial septal cells. Although McLennan and Miller interpret these findings as support for their model (see Fig. 16(c)), we suggest that a different interpretation is more plausible. The frequency of rhythmicity which they recorded (2-7 Hz) is lower than that usually found in the freely moving rat (see pp. 163-90), which suggests either that urethane has drastically slowed the frequency or that they are studying the low-frequency non-movement theta and not the more predominant movement-related theta.[*] This analysis is supported by the recent finding of Kramis, Vanderwolf, and Bland (1975) that the movement-related theta is abolished by urethane.

Other evidence against the McLennan-Miller thesis that all theta is generated from a hippocampal-septal interaction comes from the demonstration by Stumpf, Petsche, and Gogolak (1962) of a dissociation between

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[*] See p. 166 for a discussion of these two types of theta and their differential sensitivity to drug treatments.

the rhythmicity of septal units and hippocampal theta in the rabbit. They found that septal theta-related units often continued their rhythmical firing for several seconds after the onset of a hippocampal seizure before being disrupted, and quite often returned to normal during the post-ictal phase when the hippocampus was still electrically silent. Furthermore, certain drugs, such as LSD, urethane, and, at least initially, nicotine, abolished the hippocampal theta rhythm but had no effect on the bursting of the septal units. Other drugs such as scopolamine abolished both, but no drugs were found to influence the septal units without also affecting the hippocampal theta rhythm.

Finally, a preliminary report on the immobilized cat (respirated with nitrous oxide) by Wilson, Motter, and Lindsley (1975) is worth mentioning. They blocked the fimbria with a reversible cold block and studied the effect on septal bursting units. In 10 of 23 units there was no effect on the rhythmical activity. Of the remaining units two showed a decrease in firing rate but maintained their rhythmicity, two fell totally silent, and nine continued to fire but lost their rhythmical pattern. These results strongly suggest that, in addition to an afferent drive onto the septal bursting cells from the hippocampus, there is also an afferent drive from the brain-stem, and that some of these units can continue to exhibit bursting patterns in the absence of any hippocampal influence.

4.5.4. CONCLUSION

The most likely model on current evidence is that the pacemaker circuit is wholly contained in the medial septum and diagonal band of Broca. This pacemaker receives several sources of afferent drive including inputs from the hypothalamus, lateral septum, hippocampus, reticular formation, and other brain-stem structures. Looking ahead to the next chapter, it is likely that one or more of these five sources provides information about the animal’s ongoing and intended motor movements, while others signal mismatches between stimuli expected in a place and those that are experienced there. This latter information could derive from the hippocampal misplace system itself. It would appear that it is this input to the pacemaker which McLennan and Miller have studied in their urethane-anaesthetized rats. We shall discuss the septal pacemaker system and its role in building and modifying hippocampal maps after we have looked at studies of hippocampal EEG in freely moving animals.

4.6. Psychological correlates of the hippocampal EEG

In the previous section we considered those studies which provided information about the anatomical and physiological mechanisms underpinning the different states of the hippocampal EEG. Here we shall deal with those studies which have sought to determine the relationship between the EEG and events or processes in the psychological domain. First,

however, it will be helpful to set down our notions about the methodology of these experiments. We shall start with a sketch of the different logical types of experiments and then discuss some of the more obvious factors which ought to be, but frequently are not, controlled.

4.6.1. METHODOLOGICAL CONSIDERATIONS

Experiments on the relationship between hippocampal EEG and psychological variables can be roughly classified into three types: *(a)* correlational; (b) analytical; and (c) legislative. The *correlational* study seeks to establish the classes of behaviours or mental states which correspond to hippocampal theta, LIA, and SIA. Typically, the animal is placed in a situation which elicits several different behaviours to be recorded and correlated with the EEG. The situation may be a relatively unstructured one, such as a novel environment, or it may be highly structured, as when the animal is taught to press a lever in a Skinner box to obtain a reward. These studies can reveal general relations between brain activity and behaviour. Thus, for example, it might be found that theta occurs during lever pressing and moving from the lever to the food cup, while LIA occurs during eating. On the basis of these findings one might be tempted to venture a generalization about the conditions of occurrence of hippocampal theta. Unfortunately any such formulation is vitiated by the high intercorrelations between the various generalizations or hypotheses offered. Thus where one experimenter sees theta related to amount of learning, another sees it as a concomitant of increased speed of lever pressing or running in an alley, while a third focuses on the habituation of the orienting reflex or exploration.

The next logical step is an *analytical* experiment. This seeks to dissociate the various hypotheses by either holding one possibility constant and varying all the others or, conversely, varying one and clamping the rest. In the lever-pressing example it should be possible to distinguish amongst hypotheses relating theta to attention, specific movements, or expectation of reward. The animal could be required to press the lever with its nose instead of its forepaws, to vary its attention to the lever by lighting it at different levels of illumination in a discrimination paradigm, and so on. An alternative analytic approach to the relationship between hippocampal EEG and behaviour would be to vary the EEG by some independent means (usually by lesions or electrical stimulation of the central nervous system, more rarely by behavioural conditioning techniques) and observe the effects on behaviour.

The analytic technique can be repeatedly applied to narrow down the EEG/behaviour relationship. For example, if it turned out that the previous analytic experiments pointed to a correlation between movement of the forelimbs and theta, a subsequent analytical experiment could be designed to discriminate amongst the various aspects of the movement: the velocity, acceleration, force, patterning of muscles, etc.

Once the analytic experiments have pinned down the correlation, the stage is set for the *legislative* experiment. This establishes the quantitative laws governing the relationship by assessing the systematic changes in one variable as a function of another. To continue our example of the lever-pressing experiment, let us assume that the analytic experiments had shown that force of the lever press was the crucial variable. Then the legislative experiments would involve training the animal to press the lever with different forces while measuring the frequency and amplitude of theta; alternatively the animal could be operantly trained to produce theta of different frequencies and any correlated changes in force of lever pressing measured as a function of the frequency. Data from the legislative experiment provide the information with which to build a theory, and our present task would be simpler if there were enough of these kinds of experiments. Unfortunately, the field has not yet progressed to this stage, so that in general the majority of experiments to be reviewed are correlational; of the few analytical studies, the majority have been done by Black and his colleagues (e.g. Black 1975).

Before we look at the data we have several comments on methodology, first on the problem of classification and then on the inexcusable paucity of control data in many of these experiments. Often it is easy to assign an EEG waveform unambiguously to one of the three classes; however, in some instances this is difficult, in particular when there are high frequency components mixed in with the slow waves or when the EEG amplitude is small. Vanderwolf and his colleagues (e.g. Whishaw and Vanderwolf 1973) have emphasized this point in their discussion of the differences between the correlates of theta they find in the rat and those found by Grastyan and others in the cat (Grastyan *et al.* 1959). Some authors seem to have done studies ostensibly on theta, in which it is not clear that they recorded any theta at all. As we pointed out earlier (p. 157) the figures of raw data presented by Chronister *et al.* (1974) do not seem to contain very much theta. The most difficult discrimination to make is between a short burst of theta and a series of LIA spikes with approximately the same period (see e.g. Fig. 12 and Fig. 23,1). The latter often occurs in the rat just as the animal settles down after vigorous activity. In our experience the two waveforms can only be distinguished by looking at the concurrent higher-frequency waves (ripples, see pp. 151-2) or at 'theta' units. In CA1 ripples always occur on the LIA spikes and never during theta; the 'theta' units fire in a different pattern during the two EEG states (see pp. 199-201).

A similar problem may occur with SIA. As we have pointed out (pp. 153-4), it is possible that there are two distinct SIA patterns with different underlying physiological mechanisms but the same gross EEG. One is an interruption of an ongoing theta pattern and is accompanied by a cessation of 'theta' unit activity; the other is actually a form of high-frequency theta where the amplitude has dropped to a very low level because of the failure of the underlying cell membranes to repolarize. A similar suggestion has been made by Kramis *et al.* (1975). Again, the difference can only be discerned at the unit level.

A different sort of ambiguity arises when authors refer to changes in the 'amount of theta' (e.g. McFarland, Teitelbaum, and Hedges 1975). In this paper the authors studied differences in theta during different speeds of running, and found no difference in frequency but an increase in amplitude with speed. 'Amount of theta' probably ought not to be used.

Another problem concerns the accuracy of measurements of theta frequency. Many authors give the frequency to 0.1 Hz, some to 0.01Hz. Since many of these measurements are made from EEG records written out on polygraph machines, we have severe doubts whether an accuracy of 0.1 Hz is possible. Let us make some assumptions and work through the numbers. Assume a paper speed of 6o mm s^{-1} and an accuracy of measurement of ±0.5 mm: This yields an accuracy of ±8 ms. Since 7 Hz theta has a period of 143 ms, we have an accuracy of approximately ± 5 per cent or ± 0.3 Hz. The difference between 7.0 Hz and 7.1 Hz is a period of 3 ms!

Our final methodological comment concerns the lack of control recordings in many of these EEG studies. In the Chronister *et al.* (1974) study it is claimed that there is a decrease in theta activity following entorhinal cortical lesions, but there is no control record of LIA or SIA; thus, it is possible that there is a general decrease in all EEG patterns, and not merely a selective effect as implied by the authors. Few of the studies purporting to show changes in theta over time with, for instance, learning of a specific task incorporate a control condition to show that these changes are not due to electrode/tissue reactions, aging of the animal, etc. If it is imperative to provide such controls in within-animal experiments, even more so are they necessary in between-animal comparisons. The possible reasons for differences between the EEG recorded in different animals are too numerous to detail. Size of electrodes, placement in the brain, and personality of the animal are but a few. Control data which showed the same EEG from the two animals in a task uncorrelated to the main study would go a long way towards stilling this disquiet.

4.6.2. OVERVIEW OF THE BEHAVIOURAL CORRELATES OF THE HIPPOCAMPAL EEG

For a few behaviours there is general agreement for all species about the hippocampal EEG correlates; for other behaviours there is relatively good agreement within a particular species but discrepancies amongst different species; and finally there are many behaviours for which no consensus emerges, even within a particular species.

In all species theta dominates the hippocampal EEG during information-gathering behaviours elicited by novelty or discrepancy: exploration of a novel environment, the orientative reflex elicited by a novel stimulus and general searching behaviour (Table A5). During sleep the slow-wave phase is associated with LIA in all species (Table A11(a)) while the low-voltage fast or rapid eye movement phase is characterized by theta (Table A11(b)).

Aside from a few discrepant reports from Bennett's laboratory (e.g. Bennett 1969), most studies find theta of a high frequency during motor behaviours which change the animal's position in the environment and of a low frequency during motor behaviours which do not, including the most intensively studied, lever pressing (Table A6). In contrast to this uniformity, there is much less agreement about the state of the hippocampal EEG during most other situations. In some cases this lack of uniformity can be attributed to species differences, while in others it cannot. Thus, for example, even within one species, the rat, there is no consistent relationship between the effects of electrical stimulation of different brain areas on the hippocampal EEG and the rewarding properties of these stimulations (Table A12). On the other hand, attention to species differences does introduce some order into the studies of hippocampal EEG during consummatory behaviours such as eating and drinking (Table A7) and during alert immobility (Table A8(b). Consummatory behaviours such as eating and drinking are consistently associated with LIA in rodents but may be correlated with low-frequency theta in the rabbit and dog (Table A7). Similarly alert immobility in the rat and gerbil (except in the special case where it precedes jumping) is usually correlated with LIA, whereas a similar behaviour in the rabbit and cat can be associated with low-frequency theta (Table A8). Discrepancies such as these have led Winson (1972) to suggest that there is a species difference in the behavioural correlates of theta:

> 'there may be a correspondence between the theta-correlated behaviours and an important mode of natural behaviour of each species, such natural behaviours being the moving, exploratory tendencies of the rat, the readiness to run upon any provocation of the rabbit, and the stalking behaviour of the cat. Thought of in these terms, theta would be associated with a behaviour which has proven to have special survival value for the respective species' (p. 13).

Before we accept the strong conclusion that theta reflects a different function of the hippocampus in these different species, we should investigate some other possible explanations for the discrepancies.

The first possibility is that different electrode placements within the hippocampus yield different EEG/behaviour correlations and that there are systematic species differences in the placement of electrodes in the hippocampus. This explanation has been invoked by Whishaw and Vanderwolf (1973) to explain Bennett's (1969) claim that walking is not necessarily correlated with theta in the cat. They have shown that walking in the rat is accompanied by theta in most placements but that smaller movements such as pellet manipulation or lever pressing are not associated with theta at electrodes in the fascia dentata where the theta during walking is not so pronounced and is contaminated by considerable fast activity. Whishaw and Vanderwolf claim that there is low-amplitude theta at these placements during non-spatial movements but that it is obscured by the fast activity. We shall argue in a later section (pp. 178-9) that it is possible that there is no theta from these placements. In line with their argument Whishaw and Vanderwolf maintain that, with optimal electrode placement, they record theta during walking in the cat. The amplitude of this theta, however, is not as great as that recorded during orienting but the frequency is higher. A similar finding is obtained in the rabbit. As we noted earlier the amplitude of theta is probably not monotonically related to the number and frequency of theta units associated with it, since at high frequencies of unit firing there might be a decrease of theta amplitude because of a failure of the underlying membranes to repolarize (see pp. 153-4).

The argument that Vanderwolf, Whishaw, Bland, and their colleagues

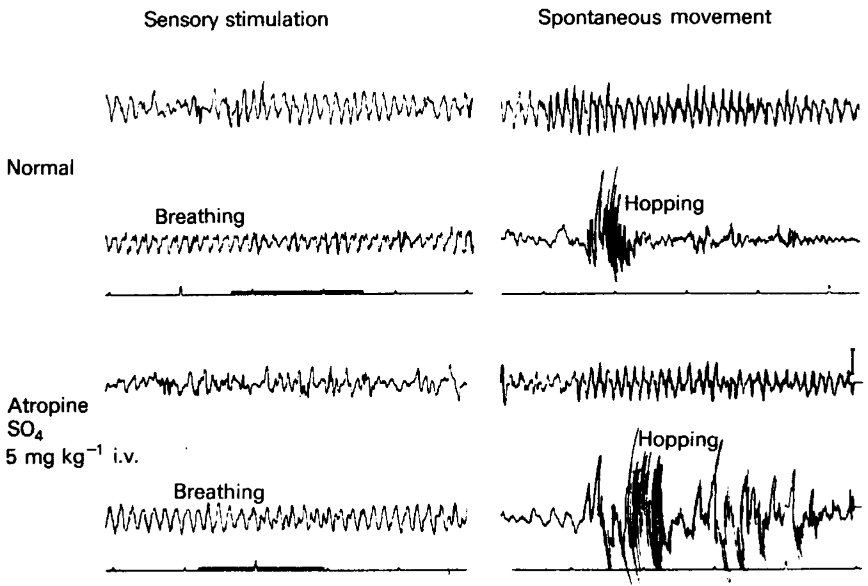

FIG. 17. Effects of atropine sulphate on hippocampal electrical activity in a rabbit during hopping and during sensory stimulation. In each panel, the top trace is the hippocampal EEG, the middle trace is a movement indicator which is sensitive enough to measure breathing, and the bottom trace is a time calibration which also marks the duration of sensory stimulation. Note that sensory stimulation (stroboscopic flashes) produced changes in respiration both before and after atropinization but did not elicit movement. Hippocampal rhythmic slow response to sensory stimulation is abolished by atropine while the rhythmical slow pattern accompanying hopping persists unchanged. Calibration: 1 s, 500 µV. (After Vanderwolf *et al.* 1975.)

have advanced, then, is that theta does occur in the cat and rabbit during rapid walking or running, but, because of its low amplitude, can only be detected with optimal electrode placements. These same workers (Vanderwolf 1975, Kramis, Vanderwolf, and Bland 1975) have suggested another possible resolution of the species differences. Instead of thinking of theta as a unitary phenomenon, they suggest that there are two types of theta, only one of which is related to movement. The behavioural correlates of this theta would be more or less the same across species. The second theta does not relate to movements and is more prominent in species such as the rabbit and cat than the rat. The main evidence for the two theta systems is pharmacological, though developmental data are also in accord with this idea. Intraperitoneal injections of atropine and other anticholinergic drugs block the non-movement theta but leave the movement related theta intact (see Fig.17); conversely, anaesthetics such as diethyl ether and urethane selectively abolish the movement-correlated theta.

As we saw in the section concerned with the physiological basis of theta (pp. 154-60) there are several anatomically distinct inputs to the medial septal pacemaker, including one from the hippocampus itself. The two-theta system formulation has the advantage of incorporating Vanderwolf's well-documented observations on the rat and at the same time allowing for clear species differences. Whether it means that theta has more than one function is a question to which we shall return. In the next two sections we consider studies of the hippocampal EEG during different behaviours in greater detail.

4.6.3. THETA DURING EXPLORATION

There is unanimity that the hippocampal EEG is dominated by theta when an animal is exploring or investigating some aspect of the environment (Table A5). Such investigation takes place when an animal is first placed in a novel unfamiliar environment, when a new stimulus is introduced into a familiar environment, when some aspect of a familiar environment is changed, for example when the reward is omitted during the extinction phase of a learning task, and during searching where the animal appears to be looking for something. Investigation in the rat always involves the animal approaching the object or place and whiskering and sniffing it with a characteristic motor pattern termed myostatial sniffing. This consists of a rhythmic pattern of synchronized head and whisker movements at a rate of 7-8 Hz (see Welker 1964). This sniffing pattern is always associated with hippocampal theta.

In the cat exploration of a novel environment usually involves gross bodily movements around the parts of that environment, but may also take subtler forms as when the cat remains in one place and visually searches its environment. Brown (1968) has reported that during the early phase of exploration of a novel environment, when the cat is actively moving about, its hippocampus shows theta which is irregular and widely ranging in frequency (3-6 Hz); during the later sedentary phases when the cat sits quietly in one spot making lots of active eye movements the theta is more marked and regular in frequency (4.3-4.7 Hz).

This theta response to novelty can also be seen during the early stages of conditioning when the conditioned stimulus is first presented. Grastyan and his colleagues have studied this phenomenon and suggested that it points to a role for the hippocampus in the inhibition of the orienting response. In Grastyan's early work (Grastyan *et al.* 1959) the cat sat in a box and could reach up onto a shelf when a sound, emanating from below the shelf, signalled the presence of food. During the early learning trials of this task the cat spends a considerable amount of time investigating the speaker while the sound is on before reaching up onto the shelf. With continued training this orientation reflex drops out and the approach to food becomes stereotyped. The hippocampal EEG shows theta activity during the orientation reflex to the cue, and a desynchronized (SIA) pattern during the short approach to food. As the orienting reflex drops out with continued training, so does the theta, until finally, when the task is well learned, the hippocampal EEG shows only desynchronization. On the basis of this finding Grastyan *et al.* (1959) argued that the hippocampus has an essentially inhibitory role, the main target of its inhibition being the reticular formation. Their argument goes roughly as follows. The orienting response is a learned reaction called forth by a familiar stimulus whose meaning, at that moment, is uncertain. This is supported by several observations. First, the initial presentations of the stimulus do not elicit an orienting response. Karmos *et al.* (1965) looked very carefully at the effect of an unfamiliar stimulus on the cat's behaviour in its home cage and found that on the first presentation such a stimulus produced only general activation or consummatory (e.g. eating, defecating) behaviours. Second, the most effective stimulus for eliciting the orientation reflex is the presentation of the stimulus in an unexpected place. In an apparatus in which one sound originating from below a feeding device served as an alimentary CS while another sound coming from an elevated shelf signalled an avoidance trial, the 'most expressed orientation reactions were obtained ... by reversing the original sites of the conditional signals' (Karmos *et al.* 1965, p. 136). It is also clear, they argue, that conditioning proper cannot proceed until the orienting response has been inhibited. Since lesions of the hippocampus, in their view, retard this inhibition process, the primary role of the hippocampus must be to inhibit those structures responsible for generating the orienting response, probably the reticular formation. Further, hippocampal stimulation directly inhibits the orienting response. Thus they argue that the hippocampus must be inoperative during the early trials and actively inhibiting the reticular formation during subsequent trials. Since the orienting response is accompanied by hippocampal theta, and its absence by hippocampal desynchronization, it follows directly from their argument that theta represents an inactive state of the hippocampus. As we have already seen, recent hippocampal physiology indicates that this conclusion is almost certainly wrong.

In more recent work Grastyan and Vereczkei (1974) have used a runway situation in which the start of a trial is cued by a sound. When this sound source was placed near the goal, or just before it, effects similar to those reported above were seen, except that the approach to the goal was accompanied by high-frequency (6-7 Hz) theta. The initial orientation to the cue, and the concomitant lower-frequency theta, rapidly diminished as the reflex became established. Changing the location of the cue elicited renewed orientation and marked theta, as well as confused behaviour which we shall discuss at greater length in a later section (p. 266).

Gray (1971), working on the rat, has also concentrated on the theta which occurs during exploratory sniffing and in particular that which occurs in response to the absence of expected reward. He and his colleagues (Gray and Ball 1970, Gray 1972, Gray, Aranjo-Silva, and Quintao 1972) recorded the hippocampal* EEG while the rat ran down an alley for water reward. There was continuous theta throughout the trial with frequencies of 7.5-8.5 Hz in the start box, rising to 8.5-10 Hz during the run down the alley, and then slowing to 6.0-7.5 Hz in the goal box as the animal drank. During non-reward trials the theta during the myostatial sniffing in the reward area had a frequency of 7.5-8.5 Hz instead of the usual slower 6.0-7.5 Hz. Gray suggested that the theta band is not a continuum but is divided into three discrete sections. At the lower end frequencies of less than 7.5 Hz are associated with consummatory behaviours such as eating and drinking, while at the upper end frequencies of greater than 8.5 Hz relate to approach to reward or avoidance of punishment. Theta in the narrow band between 7.5 and 8.5 Hz is

> 'responsible for the suppression of ongoing-behaviour upon receipt of signals of novelty, punishment, or omission of anticipated reward and for processing of new information under these conditions' (Gray 1971, p. 196).

Additional evidence for the independent identity of the 7.5-8.5 Hz band was adduced from physiological experiments in which electrical stimulation of the medial septal nuclei at low frequencies produced hippocampal theta at the same frequency with each theta wave phase locked to each stimulus pulse (theta driving). Gray and Ball (1970) found that the stimulation current necessary to produce this effect varied as a function of frequency and reached its minimum around 7.7 Hz, i.e. within the 7.5-8.5 Hz band. Furthermore, low doses of amylobarbitone selectively raised the threshold for theta driving in this band, leaving other bands relatively unaffected. Gray put all these disparate bits of information together with the fact that amylobarbitone has effects on an animal's response to non-reward during partial reward schedules or extinction and argued that the drug effect might be due to its action on 7.5-8.5 Hz theta, and that the hippocampus is involved in the frustrative response to non-reward.

To test this, he and his colleagues performed two experiments in which they tried to alter the normal relationship between the hippocampal EEG and reward or non-reward to see if this would affect the subsequent behaviour of the animals. In the first study (Gray 1972) rats were trained to run down an alleyway and were given a water reward on every trial. After reaching stable running speeds the water was omitted and extinction begun. Animals which received theta driving at 7.7 Hz throughout each extinction trial extinguished faster than animals not receiving stimulation. In contrast, giving amylobarbitone during extinction prolongs extinction.

In the second experiment (Gray *et al.* 1972) rats were trained on a 50 per cent partial reward schedule but the 7.5-8.5 Hz theta which usually occurs on the non-reward trials was blocked by high-frequency septal stimulation. Thus the hippocampus might be fooled into thinking the rat had received reward on every trial. In keeping with this interpretation, the prolonged extinction which usually results from partial reinforcement was not shown by the stimulated group. These are complicated experiments which are plagued by methodological difficulties and which are open to several alternative interpretations. For example, low-frequency theta driving from stimulation of the septum slowed the animals down in the runway which might have provided the opportunity for the development of alternative behaviours. The high-frequency disruptive stimulation appeared to be aversive and often caused seizures which would involve neural structures outside the hippocampus. It would have been much more convincing if the authors could have shown a selective effect of theta driving at 7.7 Hz, but not at 6 or 9 Hz. Nevertheless these experiments do suggest a possible role for theta during the searching behaviour elicited by non-reward.

A selective effect of producing 7.5-8.5 Hz theta has been reported by Glazer (1974a) in a lever-pressing experiment which he interpreted as further evidence for the role of hippocampal theta in responses to frustrative non-reward suggested by Gray. Three groups of rats were initially given training on a schedule requiring five lever presses for reward (fixed ratio 5; see pp. 321-2). Then one group was operantly conditioned to produce 7.5-8.5 Hz in the same box, a second group was conditioned to produce lower frequency theta, and a third group acted as a yoked control, receiving reward whenever the first group did regardless of their behaviour or EEG pattern. After this, further fixed-ratio lever pressing was allowed, followed by extinction. Resistance to extinction was increased in the group which

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* The recording electrodes were actually in the dorsal subiculum medial to the CA1 field which might explain the unusual finding of low-frequency theta during consummatory behaviours in the rat.

had received 7.5-8.5 Hz theta conditioning. Glazer suggested that the increased resistance to extinction was a function of the 'counterconditioning' of frustration, as reflected by the presence of theta; according to frustration theory (cf. Amsel 1958) this would lead to longer extinction. Although we have no ready explanation for this finding, two points are worth noting. First, during the operant conditioning of theta or LIA the rats rewarded for theta sniffed and reared, while the LIA group were inactive; this agrees with Black's work in dogs (see pp. 174-5). Second, the conditioning of theta developed more slowly than that of LIA, suggesting that during the time they were not lever pressing in this situation the rats had a tendency to remain inactive. It is possible that some 'counter-conditioning' of this inactivity occurred during the time when theta was being rewarded and that this led to the increased resistance to extinction.*

Two recent studies have addressed themselves specifically to the question of a theta role in frustration effects. As a direct test of the hypothesis Kimsey, Dyer, and Petri (1974) tested rats on a go/no-go alley task in which reward was only available on alternate trials (see p. 331). In this task rats learn to run slower on the trials following reward, showing that they have noticed the alternating pattern of reward and non-reward trials. Mean frequency of theta in the goal box during reward consumption was about 6.5 Hz, while during the exploration elicited by non-reward it was about 8.0 Hz. To test the role of the anticipation of non-reward, the investigators included some probe trials on which the rats were rewarded when they expected non-reward, and vice-versa. On these trials the hippocampal EEG reflected the rat's behaviour (either eating or sniffing) and not its expectancy of reward or non-reward.

Another test of the Gray-Glazer hypothesis has been performed by Morris and Black (personal communication). They recorded hippocampal EEG during various behaviours while the rat was in a free environment and constructed frequency spectrograms for each behaviour. They then recorded the animal's behaviour in a non-reward situation similar to Gray's, and computed the proportion of time spent at the different behaviours. They reasoned that if the EEG reflected nothing more than the animal's behaviour (e.g. sniffing) during non-reward, their frequency spectrograms could be generated simply by adding together weighted spectrograms from each of the component behaviours. The recorded EEG matched the predicted EEG derived in this way quite well, suggesting that the former reflects the animal's behaviour and not some internal state of frustration elicited by non-reward.

A more general criticism of the Gray thesis is its failure to take into account the non-specificity of theta. While it is true that 7.5-8.5 Hz theta occurs during those behaviours elicited by non-reward or during exploration, it also occurs during a short burst of walking, during swimming, and during wheel running (Vanderwolf et al. 1973).

One question that will continually recur throughout this chapter is whether correlations between the hippocampal EEG and behaviour must be confined to gross behavioural descriptions such as exploration or whether they can be related to the fine details of the behaviour. In the case of exploration it has been suggested that there is a one-to-one correlation between individual myostatial sniffs and theta waves in the rat. Similarly the theta during investigation in the cat might be associated with eye movements.

Komisaruk (1970) reported that during myostatial sniffing there was a one-to-one phase locked correlation between each sniff and a theta wave. In addition there were also correlations between sniffing and bursts in the hippocampal multi-unit activity, and between heart rate and theta. It was not clear whether Komisaruk was suggesting that hippocampal theta only occurred during myostatial sniffing or that it could occur during several different behaviours but during myostatial sniffing there was a phase locking.

Whishaw and Vanderwolf (1971) criticized the former view and emphasized the dissociability of sniffing and theta. They showed examples of behaviours where there was good hippocampal theta without rhythmical whisker movements (swimming), good whisker movements without theta (sniff with head still), and good whisker movements with good theta, but with no constant phase relation between the two. A resolution of this issue has been suggested by Macrides (1975) in what appears to be an important study. He repeated Komisaruk's study in the hamster and found that, as Whishaw and Vanderwolf stated, there usually was no constant phase relationship between sniffing and hippocampal theta. However, every once in a while the two rhythms would become phase locked and remain so for up to 10 successive sniff cycles. That this entrainment was not just a chance phenomenon was strongly implied by his finding that in any particular rat the two rhythms always became phase locked with the same phase shift. For example, in one animal the hippocampal theta might lag behind the sniff cycle by 90° during entrainment. The phase difference was characteristic of each rat, but differed from rat to rat. Another interesting finding was that entrainment was often preceded by a brief acceleration or deceleration in the frequency of the sniff rate, or by a discontinuity in the sniffing rhythm such as a skipped beat. It was as if there was an active mechanism bringing the two rhythms into phase. We should remind the reader that lesions of the medial septal pacemaker, which eliminate theta from the hippocampal EEG, also abolish myostatial sniffing in some animals, although the effects are dissociable (Gray 1971). In Macrides' study no difference in the theta-sniffing relationship was seen during the

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* We discuss the role of the hippocampus in frustration later (pp. 348-50) and conclude that the evidence from lesion studies argues strongly against the hypothesis that the hippocampus, and a specific theta band, are central to frustration effects.

sniffing of various odours, strengthening the suggestion that the relationship is with the motor rather than the sensory aspects of sniffing.

In the case of the cat there is some evidence that theta may be related at least in part to the eye movements which occur during exploration and visual searching. Recall that Brown (1968) found a clearer, more consistent theta during visual searching in the relatively immobile cat than during the earlier more active phase of exploration. Sakai, Sano, and Iwahara (1973) studied the relationship between eye movements and theta in cats during low-voltage fast sleep and waking and reported a good correlation. This correlation remained when the animal was paralysed with curare and the ponto-geniculate potential was used as an index of intended eye movements. Finally they noted that the reflex eye nystagmus produced by caloric stimulation was associated with theta. Similarly, Costin, Bergmann, and Chaimowitz (1967) found theta during nystagmus produced by rotation on a table. It might be interesting to study the relationship between theta and eye movements in cats trained to track visual stimuli, to locate visual stimuli whose position is unpredictable, and to make eye movements in the dark.

As we have mentioned Whishaw and Vanderwolf have suggested that the results in the cat may be due to less than optimal electrode placement; they found theta during running and walking as well as during searching and exploration, but found that the frequency was higher and the amplitude smaller. As we have pointed out (pp. 153-4) there is probably not a linear relationship between the amplitude of theta and the number and rate of the underlying displace units. Thus a low-amplitude theta does not necessarily signify a lower level of activation of the theta mechanism. Another factor contributing to the large amplitude of theta during exploration might be the involvement of the misplace units of CA1. As we shall see in the next section on the activity of single units in the hippocampus of the freely moving rat (pp. 201-11) there are units in CA1 which fire maximally when the animal explores a part of the environment. These misplace units fire in synchrony with the theta waves while the animal is investigating the appropriate place. Thus during exploration of a place the firing of these units might summate with the theta mechanism to produce greater amplitude potentials during exploration than during walking in a familiar place. We shall also suggest in a later section (pp. 228-30) that one of the functions of CA1 is to signal a mismatch between what is expected in a place and what is found there and thus serve to initiate exploration.

4.6.4. MOTOR PERFORMANCE AND HIPPOCAMPAL EEG

There is good agreement for all species except the cat that during motor behaviours involving spatial displacements there is high-frequency (> 8 Hz, rat and rabbit; 5-7 Hz, dog and cat) theta in the hippocampal EEG (Table 6(a)); during non-consummatory motor behaviours not involving spatial displacement there is usually low-frequency (6.7 Hz, rat; 3.5-5.5 Hz, dog and cat) theta (Table A6(b)). Consummatory behaviours such as eating and drinking are associated with LIA or low-frequency theta, with the notable exception of sexual behaviours in the rat where high-frequency theta has been reported (Table A7).

There are two ways to interpret this pattern. The first, put forward by Vanderwolf (1969, 1971), states that theta is a continuum and is related to voluntary movements. Different frequencies of theta, according to this notion, relate to different parameters of the movement such as its acceleration or, to some extent, the amount of vigour involved. The alternative interpretation of this pattern of EEG-behaviour correlates was first suggested by Adey (1967) and by Gray (1971). This is that theta is not a single continuum but subdivides into different frequency bands, each of which has a different behavioural correlate. We shall discuss Adey's ideas below. As detailed above (pp. 168-9) Gray thought that low-frequency theta in the rat was associated with consummatory behaviours, intermediate theta with exploration and reactions to the omission ‘of expected reward, and high frequency theta with approach to reward or avoidance of punishment. Vanderwolf (e.g. 1975) has recently also advocated a variant of the multi-band thesis on the basis of the differential susceptibility of theta during different behaviours to pharmacological agents. We shall first consider the evidence for the view that theta forms a continuum related to voluntary movement and then discuss the notion that there is more than one type of theta.

Vanderwolf, Black, and their respective associates have stressed the relationship between the animal's motor behaviour and the hippocampal EEG. Vanderwolf, Bland, Whishaw, and their colleagues have concentrated on correlation studies, attempting to relate all of the animal's behaviour to one of the three states of the hippocampal EEG (see pp. 141-3 for a description of these three states). Black and his colleagues have concentrated on analytic studies which dissociate movement from other variables such as attention and learning.

Basing his assertions on studies done primarily, but not exclusively, on the rat, Vanderwolf (1969, 1971) reports the following correlations between hippocampal EEG and behaviour.

> *RSA (theta):* walking, running straight ahead or backing up, turning, rearing, jumping, climbing, struggling when held, swimming, head movements, postural changes (such as shifting weight from one foot to another, getting up, lying down), manipulation (such as handling a large food pellet, or pressing a lever in a Skinner box), and digging in sawdust.
>
> *LIA:* behavioural immobility (whether standing still or hanging from a support), licking, chewing, chattering of the teeth, chewing without anything in the mouth, salivation, piloerection, urination, defecation, pelvic

> thrusting and ejaculation during coitus, face washing, licking and biting the fur on any part of the body, scratching with a hind foot, vocalization (squealing in rat, barking in dog), shivering, and stamping on the ground with the hind feet (in gerbil, Kramis and Routtenberg 1969).
>
> SIA: drowsy rat startled, leaps to feet but does not run; rat jumps out of avoidance box and almost goes over the side (requiring sudden halt of forward movement); rat in Skinner box, thrusting head into food cup and stopping abruptly to seize a food pellet which had just been delivered; rat in motor-driven wheel where behaviour consisted in alternately jumping (RSA) and clinging to the wheel (SIA).

According to Vanderwolf, within the RSA (theta) category there are further correlations between behaviour and the frequency and amplitude of theta. Roughly, 'the amplitude of RSA is related to the gross amount of concurrent motor activity. Frequency increases, on the other hand, are associated with the initiation of movement' (Vanderwolf 1971, p. 92). More specifically

> '. . . RSA amplitude is related to the extent (i.e. possibly the total number of motor units activated together) of muscular activation while frequency shifts are related to the acceleration with which movement is initiated. However, frequency is also increased during large as compared to small movements and the present data do not show clearly the degree to which RSA frequency and amplitude are independently related to behaviour' (Whishaw and Vanderwolf 1973, p 481).

Black, Dalton, and colleagues, in a series of well-designed analytic experiments, have provided strong support for the hypothesis that hippocampal theta is correlated with some aspect of motor activity. In one set of experiments (Dalton and Black 1968, Black and Young 1972a) dogs were trained to avoid shock by pressing a lever in the presence of one stimulus and by refraining from movement in the presence of a second. Measurement of hippocampal theta in the two conditions showed more theta during the lever press than during immobility. Since the dogs successfully learned to avoid shock in both conditions, the difference in brain activity must be attributable to the movement and not to the degree of learning, attention, or motivation.

In a second series of studies (Black 1972) the converse procedure was employed. Brain activity was conditioned and behaviour measured as the independent variable. Dogs were operantly trained to avoid shock by generating hippocampal theta in the presence of one stimulus and by refraining from producing theta in the presence of another. Simultaneous recording of behaviour showed that more movement occurred during the theta condition. Earlier experiments (Dalton 1969) had shown that theta could be operantly conditioned in the paralysed dog, and the experiment just described was also performed under paralysis. After conditioning, the dogs were tested without paralysis and found to move more during the theta condition than during the non-theta condition. As Black has pointed out (e.g. Black 1975), this finding rules out the possibility that theta is related to movement-produced feedback from the periphery.

In a final experiment (Black 1975) an attempt was made to condition high-frequency theta in rats allowed to move or forced to remain immobile. Only the group allowed to move could generate theta with frequencies above 7 Hz. Theta in the immobile group was restricted to a frequency of less than 7 Hz. Thus there seems to be an ineluctable connection between movement and higher-frequency hippocampal theta.

In addition to those of Black and his colleagues, several studies of theta during lever pressing for food have been reported (Table A6(c)). In general, there is good agreement that, in the rat, the hippocampal EEG shows theta activity during the lever press and LIA during the consummatory act. There is less agreement in other species, in particular the cat. In a study in which a fixed ratio schedule was used (see p. 321) Pond and Schwartzbaum (1972) showed that there was a greater amount of theta in the hippocampal EEG during the first eight lever presses than during the last eight; in this situation the rat had to press the lever 16 times to obtain reward.*

Most studies report that theta persists during lever pressing, no matter how long training is continued. There is evidence, however, that the theta during highly overtrained, high-ratio lever pressing has a smaller amplitude than that recorded earlier in training, and that there is considerably more LIA in the record (Frederickson 1974a). In contrast, Feder and Ranck (1973) reported LIA during lever pressing on a highly overtrained fixed-ratio schedule requiring 50 presses for a reward. Furthermore, theta units (see below) had the same rate during lever pressing as during the consummatory act. This latter is a very sensitive index of the difference between theta and LIA. As we discuss shortly, it is likely that the rats in this study had learned to use some automatic behaviour to press the lever repetitively.

The presence of hippocampal theta during lever pressing in the cat and dog is more controversial. Black and his colleagues (Black 1975) have found that there is a higher incidence of theta during lever pressing than during immobility in both species. Others (e.g. Lopes da Silva and Kamp 1969, Yoshii *et al*. 1967) have reported no theta during lever pressing.

We can only suggest in considering these contradictory data that, although theta has been reported during lever pressing in all species, there are enough negative reports to indicate that the actual morphology of the

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* We have criticized the use of 'amount' of theta earlier (p. 163). It involves triggering a window discriminator when the theta amplitude reaches a certain voltage, and counting the pulses so generated. As such it cannot discriminate between changes in amplitude and frequency. As we mentioned earlier (p. 139), this study also showed a correlation between the amplitude of a visual evoked response in the neocortex and the amount of hippocampal theta.

response, or some other variable, may be an important deciding factor. In the rat both the frequency and amplitude of lever pressing-related theta are low, making detection somewhat difficult.

An obvious question is: To what aspect of movement is theta related. As pointed out in the introduction to this section, correlation studies can suggest possible answers to questions of this nature, but in the final analysis they can only be settled by analytic experiments. One thing seems clear from the correlational studies; theta is not uniquely related to any simple aspect of movement such as the activation of a specific muscle or muscle group, or the movement of a particular joint. For example, the same theta occurs whether the rat turns its head to the left or to the right in response to a stimulus. The theta frequency during two such disparate movements in the rat as running and swimming are approximately the same (7.5 Hz), as are the amplitudes (Whishaw and Vanderwolf 1973). In spite of this some authors have suggested a relationship between a specific movement and theta. As we have noted above Komisaruk (1970), for example, noted that theta in the rat occurred at a similar frequency to certain rhythmical behaviours, in particular the myostatial sniffing which occurs primarily during exploration. He reported that there is sometimes a one-to-one, phase locked correlation between the theta waves and the myostatial muscles, but it was clear that the phase locking, if it existed, was not a constant one.

If theta cannot be related to some simple peripheral feature of movement such as the contraction of particular muscles, then one must try to relate it to some more global or molar aspect of behaviour. On the basis of his observations on the rat, Vanderwolf (1969) originally suggested that theta (RSA) was associated with voluntary behaviours, LIA with automatic species-typical behaviours, and SIA with the abrupt cessation of ongoing movement:

> '... the rhythmical activity produced by the hippocampus may be a reflection of the activity of some of the complex circuitry which appears necessary for voluntary movement' (p. 415).

But what is voluntary movement? In Chapter 1 we discussed the Helmholtz/Poincare theory that locale space could be built up as a result of the organism's observations of the effects of its voluntary movements on its perceptions. In the classical philosophical sense 'voluntary' movements are those initiated by, or at least accompanied by, an act of the will. Neurologically, one difference between voluntary and involuntary movements is that the former are accompanied by a corollary discharge from the motor systems which enables the sensory systems to 'predict' the outcome of the movement and thus stabilize the world (von Holst 1954).

Vanderwolf (1971) has tried to specify the differences between voluntary and automatic behaviours. First, a voluntary behaviour can be used to

accomplish different goals, while automatic behaviours are associated with one particular motivational state. Thus an animal can walk or press a lever to obtain food or to avoid punishment, but usually only drinks when it is thirsty. A second difference is that the sequence of movements in a species typical behaviour is relatively fixed, whereas that of a voluntary behaviour is less so. Once a rat has groomed one side of its body it is highly probable that it will go on to groom the other side. Finally, voluntary behaviours appear to be organized at a different (higher) level of the nervous system than are automatic behaviours, as shown by the fact that animals with high decerebration (removing all structures above the mesencephalon) can still lick, chew, groom, and shiver, but cannot walk or perform other 'voluntary' behaviours (Woods 1964; see Vanderwolf 1971 for a good discussion of these points).

There is no doubt that Vanderwolf's voluntary/automatic distinction captures a good deal of the difference between theta-related and LIA-related behaviours. Nevertheless, enough difficulties with the classification have arisen that Vanderwolf now plays down the use of the terms voluntary and automatic in his writings (e.g. Vanderwolf et al. 1975), and refers to the two classes of behaviour by the theoretically neutral but mnemonically hopeless terms type I and type II.

In terms of the rigidity of the pattern of the behaviour, or the transition probability between it and preceding behaviour, there are several behaviours in the rat which do not fit the above classification. The transition from grooming one part of the body to another, postural adjustments, and the righting reflex elicited by placing the animal on its back or dropping it upside down would all normally be described as reflexive or automatic, can only be used to accomplish one end, and have high transition probabilities from the preceding behaviours. Yet, they are all accompanied by hippocampal theta, not LIA. Conversely, such behaviours as jumping in the rat, which can be used to satisfy several different motives, are nevertheless rather rigid sequences of muscle activations. In fact, rather than thinking of the voluntary/automatic distinction as a rigid one with sharp boundaries, it is more likely that one is dealing with a continuum extending from the reflex arc at one extreme to walking and lever pressing at the other.

Certainly, so-called automatic behaviours, such as licking, can be employed by the rat as operants, that is, as a means to accomplish some other goal. For instance, Black and Young 1972b) trained rats to lick a water tube to avoid shock. The hippocampal EEG showed LIA as it also does during the superstitious or displacement drinking induced by certain lever-press schedules (e.g. Haines and Ranck 1975). LIA also predominates when rats are operantly trained to hold still in order to receive rewarding brain stimulation (Paxinos and Bindra 1970).

There is one report of a putative voluntary behaviour (lever pressing on a fixed-ratio schedule in the rat (Feder and Ranck 1973)), during which LIA was recorded from the hippocampus. As we have seen this last study is an exception rather than the rule; most lever-pressing studies find a considerable amount of theta. There have been suggestions that in the Feder and Ranck study the rats were acting differently than in other studies, perhaps calling into service some automatic behaviour such as shuddering or shivering. If so, this would be another example of an automatic behaviour used as an operant, accompanied by LIA in the hippocampal EEG, and a further proof of the irrelevance of the motive behind a behaviour in determining the state of the hippocampal EEG.

A final objection to the original Vanderwolf notion that hippocampal theta only occurs during voluntary movements is the occurrence of theta in the absence of any movement. Even in the rat, theta is seen during immobility before a jump (Table A8(a)) and during some presentations of a stimulus which had previously been paired with inescapable shock. Theta is even more frequently observed in the immobile cat and rabbit (Table A8(b)). Originally, Vanderwolf proposed that in these circumstances the central motor programmer, and the related theta, was activated but the programmed movement was not triggered. A good example of such a situation is low-voltage fast sleep during which there is considerable neural activity in motor areas which fails to reach the effectors because it is blocked at spinal-cord levels. More recently, as we have seen, Vanderwolf and his colleagues have suggested that there are two types of theta, one related to voluntary movements in the manner previously described and the other unrelated to movement.

If there is more than one type of theta, separable on the basis of frequency or pharmacological sensitivity, then the question arises as to the behavioural correlates of each theta. Vanderwolf's original classification scheme must be re-examined since it was based on the assumption of a unitary theta system. In particular, some doubt surrounds the inclusion of small non-spatial movements such as isolated paw movements in the same category as the larger displacement movements rather than with immobility theta or the low-frequency theta which occurs during consummatory behaviours in rabbits. In the first place the frequency of these small movements tends to be in the same low range as those seen during immobility. Furthermore, there are several observations which suggest that the theta during non-spatial movements has different characteristics from the theta associated with displacements. First, Vanderwolf (1975) showed that injections of atropine abolished immobility theta seen before jumping but left the theta during jumping or walking intact. Interestingly:

> ' .. the RSA accompanying small head or limb movements, which normally has a lower frequency and a smaller amplitude than RSA accompanying gross movements, appeared to be more severely depressed by atropine than the RSA accompanying gross movements (e.g. walking or struggling)' (p. 308).

suggesting that it may be part of the same system as immobility theta.

Second, Whishaw and Vanderwolf (1973) reported that electrodes in or near the CA1 field record large-amplitude, high-frequency theta during spatial displacement behaviours such as walking or jumping and low amplitude, low-frequency theta during smaller non-spatial movements. In contrast, deeper electrodes in the dentate area record theta only during the spatial behaviours and not during non-spatial movements such as lever pressing. Whishaw and Vanderwolf argue that the greater amount of high frequency activity at these deeper placements obscures the low-amplitude, low-frequency theta which occurs during lever pressing. It is equally possible that the two thetas have a different distribution within the hippocampus with the theta associated with lever pressing and other small movements occurring only in the CA1 field. An experiment mapping the distribution of the two thetas in the rat is needed. Winson (1976b) has mapped the distribution of theta during low-voltage fast sleep, sensory stimulation, and voluntary movement in the rabbit and finds them all to be the same. However, it is not clear if his voluntary movements were spatial movements.

Finally, Bland and Vanderwolf (1972a) have shown that posterior hypothalamic stimulation produces spatial movements such as walking and running, and associated theta in the hippocampus. Increases in the strength of stimulation increase the rate of running. Furthermore, stimulation synergizes with naturally occurring spatial behaviours and strengthens them. For example, stimulation during jumping increases the height that the rat jumps. In contrast, stimulation in the posterior hypothalamus inhibits lever pressing. In summary, spatial and non-spatial behaviours appear to be organized differently in the brain stem, and the theta associated with them may have a different distribution in the rat hippocampus and may be sensitive to different pharmacological manipulations.

Elimination of small voluntary movements such as these from the class of atropine-resistant behaviours leaves a set of behaviours behind which can best be described as those which translate the rat's position in space. We shall suggest that one of the functions of theta is to shift the focus of excitation within the map from those cells representing the animal's present position in the environment to those which represent the position it will occupy as a result of movement. In order to accomplish this end, some aspect of theta must signal either the velocity of movement through the environment or the distance which the movement translates the animal within the environment.

In order to test this prediction from the theory, we (Morris, Black, and O'Keefe 1976) chose to study jumping in the rat, a ballistic movement during which all the muscle activation is packed into a short launch period. After take-off there is little that the animal can do to correct any major mistakes in its trajectory. Vanderwolf and his colleagues (Vanderwolf 1969, Whishaw and Vanderwolf 1973) had reported that prior to jumping there is continuous theta and that the frequency increases sharply at the time of launch. Furthermore, a jump to a height of 22 in. was accompanied by a higher frequency at launch than one to a height of 11 in. Finally, there was the tantalizing suggestion in one of their review papers (Vanderwolf *et al.* 1973) that placing weights on the rat's back had no effect on theta frequency during jumping. Since this latter manipulation could be expected to alter many aspects of the jump, it might enable one to isolate the crucial variable related to the frequency increase.

In our study rats were trained to jump either for food or to avoid shock. In the shock experiment the animals were required to jump vertically to the top of a false internal wall, the height of which could be varied between 20 and 60 cm. In the food-motivated experiment the hungry rats ran down an alley and then jumped onto a shelf where food was located. During the jumps we measured the force exerted by the animals on the floor. As Vanderwolf and colleagues reported, there is continuous theta while the rat prepares to jump and there is an increase in frequency of theta during the launch. In our experience the maximal changes in frequency occur either during the exertion of force or during the flight itself. There is a good correlation between the frequency of theta during the jump and the distance jumped. No effect of distance jumped is seen in the theta prior to the jump. Fig. 18 shows the best height to theta frequency relationship obtained thus far from an animal required to jump accurately to 10 different heights. To ensure accurate jumping at each height, a second wall was brought down to within 2 cm of the top of the false wall.

In contrast to this relationship between height and the period of the theta waves, adding 50 g to the animal's back had no effect on the period of the theta waves for a jump of a constant height. An analysis of the effect of the additional weight on the animal's behaviour will help us to decide what the theta period is related to. In order to reach a given height the rat must attain a certain velocity at the moment it leaves the surface. It does this by generating a force over a short period of time *(impulse).* Greater heights require greater take-off velocities, and thus greater impulses for a given mass. Increasing the rat's mass by placing weights on its back forces it to produce a greater impulse to reach the same height. Since only the higher, and not the heavier, jump is associated with a higher-frequency theta, impulse can be eliminated as a candidate for the correlate of theta period. Similar arguments can be applied to force and acceleration. Given that an animal must increase its impulse at take-off, it can do so by one of two means. Either it can increase the peak and average forces exerted for the same time period, or it can exert the same amount of force (or even less force) for a longer period of time. Some animals appear to choose the former strategy, but others, such as the animal whose data are shown in Fig. 18, opt for the second strategy. For this rat increases in impulse associated with increased mass were accomplished by exerting a decreased force for a longer time, while increases in impulse associated with increased distance were accomplished by exerting an increased force for the same time. For this animal, then, there was a clean dissociation between theta frequency during the jump and the force. Since acceleration is a direct function of force and an inverse function of mass, addition of the weights to this animal caused a large decrease in acceleration; thus we can eliminate acceleration as a candidate.

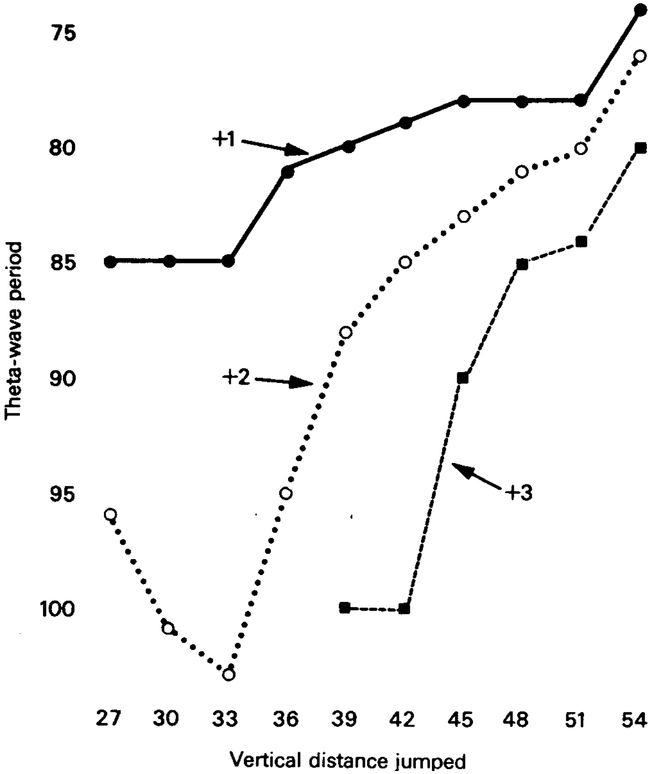

FIG. 18. Relationship between vertical distance jumped by a rat (abscissa) and periods of the three hippocampal theta waves following take-off (ordinate). In this animal little relationship was seen below jumps of 33 cm. Above 33 cm there is a good relationship between theta frequency and the distance jumped. (From Morris, Black, and O'Keefe, unpublished.)

Comparable analyses enable us to eliminate all other physical variables aside from take-off velocity, distance jumped, and the acceleration of and the force acting on the head. These latter are independent of the body for the initial part of the jump and have not been measured yet. The changes in pattern of the force curve with the addition of weights was related to a significant reorganization of the temporal activation of the various muscle groups during the jump, and we suspect that these changes can also be ruled out although it will be necessary to record electromyograms (EMG) from the muscles involved to do so.

These preliminary findings strongly suggest that during a ballistic movement such as jumping, the frequency of theta is related to velocity of the movement or to the displacement which the movement produces. As we have already seen, during a continuous movement, such as running on a treadmill or in a running wheel, the initial frequency of theta is a function of the velocity of the moving surface, but with continued movement the frequency tends to settle down to the same value irrespective of velocity of movement. Since there is a difference in the pattern of movement during running on a treadmill and running across an immobile surface (Wetzel *et al.* 1975), one must take care about generalizing these findings to natural running. One must also ask how a rat or cat normally runs around an environment. Does it run in fits and starts, with a constant acceleration, constant velocity, or what? Until we have such information we shall have to be somewhat cautious about the use of these data in our models of hippocampal function. Nevertheless, it appears that the frequency of movement-related hippocampal theta is a monotonic function of the speed with which a ballistic movement is initiated or the distance that it translates the animal in its environment.

4.6.5. HIPPOCAMPAL EEG DURING SENSORY STIMULATION

Table A9 lists those studies which have recorded hippocampal EEG during sensory stimulation, while Table A10 lists those in which the sensory stimuli have been used as conditioned stimuli either in Pavlovian or operant tasks. The tables are difficult to interpret, since in many studies the animals were not required to hold still nor were they reported to do so. Therefore the theta associated with sensory stimulation might have been due to the associated orientation or investigation. Nevertheless enough studies have taken this into account to enable generalizations to be made. In the rat sensory stimulation produces LIA unless the animal orients towards it (Whishaw 1972, O'Keefe, unpublished observations). On the other hand, stimuli used as cues in aversive tasks do produce low-frequency theta in immobile animals (e.g. Pickenhain and Klingberg 1967, Whishaw 1972, Schwartzbaum 1975). Theta also occurs in an immobile rat prior to jumping (Table A8(a)) whether to obtain food or avoid punishment (Morris, Black, and O'Keefe, unpublished). In his earlier papers (e.g. Vanderwolf 1971) Vanderwolf suggested that this non-movement theta was related to the activation of a central movement programmer prior to the movement itself. It would be interesting to see if theta was elicited by a stimulus which signalled the onset of rewarding ESB where the rat was required to remain immobile. The results described by Black (1975) in which immobile rats could be trained to produce low (but not high) frequency theta for rewarding electrical brain stimulation suggest that this should be possible.

If there is low-frequency theta in the rat which is not connected with movement, why is it not seen more frequently? One hint comes from a lesion experiment by Robinson and Whishaw (1974). Posterior hypothalamic lesions in rats drastically reduced their spontaneous motor movements. Robinson and Whishaw (1974) reported spontaneous occurrences of low-frequency theta during immobility in these akinetic rats. Perhaps in normal rats the situations which elicit low-frequency theta also often elicit spatial movements and the higher-frequency theta associated with these overrides the low-frequency theta.

In contrast to the rat, the rabbit hippocampal EEG shows abundant theta activity in response to any arousing or attention-attracting sensory stimuli (Figs. 17 and 19). This arousal response was first reported by Jung and Kornmuller (1939) and was documented by Green and Arduini (1954) in their classic paper on theta in the rabbit hippocampus. They did not make it clear whether or not an orientation towards the stimulus was necessary for the theta response. Several recent studies on the rabbit (Klemm 1970, 1971, Harper 1971, Kramis, Vanderwolf, and Bland 1975) have confirmed that theta (usually low frequency, 4-7 Hz) does occur during immobile arousal.

Green and Arduini suggested that hippocampal theta was a sign of arousal analogous to neocortical desynchronization. In support of this notion Klemm has recorded electromyograms and multiple-unit activity in the mesencephalic and medullary reticular formation, and found a good correlation between phasic increases in EMG and reticular activity on the one hand and the occurrence of hippocampal theta on the other. Kramis *et al.* (1975) dispute this correlation and cite instances of hippocampal theta activation without concomitant EMG activation. They further claim that a rabbit which is highly alert need not show theta in its hippocampus. We shall return to the question of how useful such concepts as arousal and attention are at the end of this section.

In the cat the hippocampal EEG response to a sensory stimulus is not as clear as it is in the rabbit. As we have seen, considerable theta occurs during exploration of a new environment, but here there is movement to consider. Several authors have insisted that theta can occur in the cat in the absence of gross bodily movements (Brown and Shryne 1964, Brown 1968, Bennett 1969, Whishaw and Vanderwolf 1973, Kemp and Kaada 1975). This theta seems to be best elicited by moving stimuli, or by a reflection of the animal in a mirror. There have been suggestions that, at least in part, theta in the immobile cat may be associated with eye movements (see above, p. 172).

Bennett and his colleagues (Bennett 1970, 1975, Bennett and Gottfried

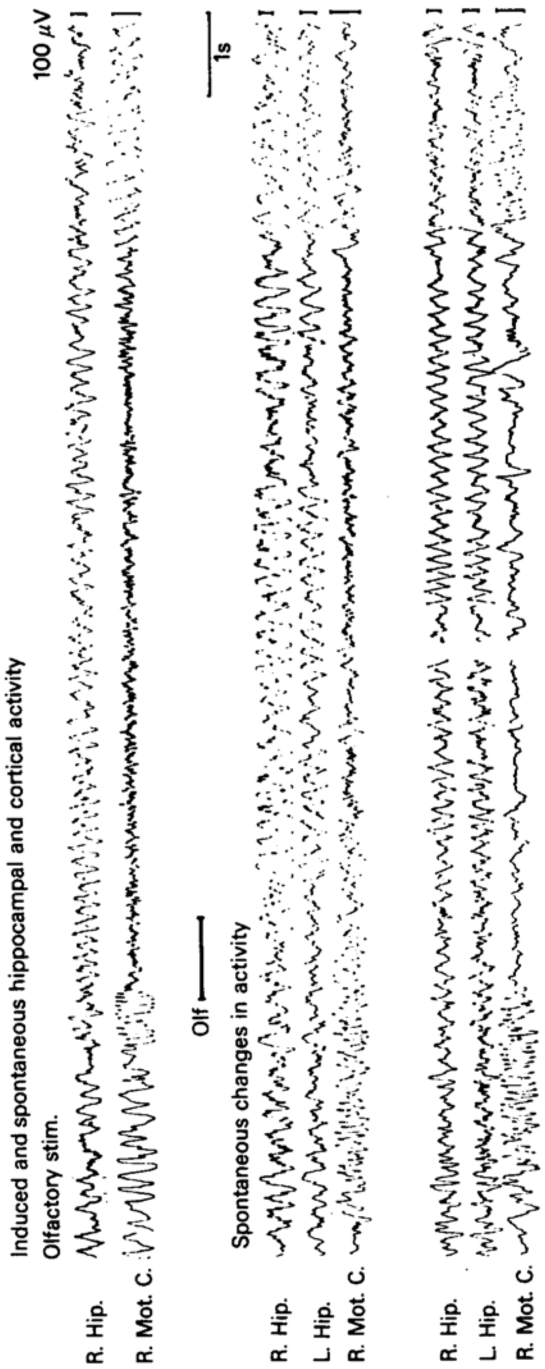

FIG. 19. Acute preparation, rabbit. Abbreviations: R. Hip., right hippocampus; R. Mot. C., right motor cortex; L. Hip., left hippocampus. The first pair of records shows the effect of an olfactory stimulus (olf). Examples of inverse relationships between neocortex and paleocortex are seen in the second and third groups of records. Another group of records (not shown) showed the absence of this inverse relationship. (Modified from Green and Arduini 1954.)

1970, Bennett, Herbert, and Moss 1973) have recorded hippocampal EEG from rats and cats during a lever-pressing task in which the animal is only rewarded if it waits a certain period of time between each lever press (the DRL schedule, see p. 323). In the early studies on cats they found an absence of theta during lever pressing, and a difference in theta incidence during the waiting period dependent upon the presence or absence of a cue signalling the end of the delay. There was a high incidence of theta with the cue (Bennett *et al.* 1973), while SIA predominated without the cue (Bennett and Gottfried 1970). It was concluded that theta was a correlate of attention to environmental cues, while SIA related to attention to proprioceptive cues. Later work in the rat showed that theta occurred during lever pressing and during the waiting period, with or without a cue (Bennett 1975). While Bennett now allows that the correlate of theta in the rat dorsal hippocampus is something like voluntary movement, he maintains that theta in the cat is solely a function of attention to external cues. We would suggest a somewhat different interpretation of these data. First, it is possible that Bennett is concentrating solely upon the low-frequency theta which, as he suggests, may be related to attention. Second, the differences observed in the cats tested with and without the cue could reflect differences in behaviour in these two situations during the waiting period. Bennett (1975) reported that in the absence of the cue all cats mediated the delay by going to the opposite end of the apparatus and lying down for some time. The dominance of SIA in the hippocampal EEG for these cats is consistent with the view that high-frequency theta is a function of displacements. On the other hand, the rat appears to mediate the delay period by engaging in active behaviours (see p. 324); this would explain the presence of theta in Bennett's rat experiment in the situation producing SIA in the cat. Unfortunately, Bennett (1975) did not provide protocols of the cats' behaviour during the waiting period when a cue was present, so it is impossible to know if the theta seen during that time was paralleled by movement or whether it was a function of immobile attention.

To summarize, then, theta can easily be elicited by sensory stimuli in the rabbit, is more difficult to elicit in this way in the cat, and can only be seen under exceptional circumstances in the rat in response to such stimulation. Following Green and Arduini, various authors (e.g. Bennett 1971, Klemm 1970, Kemp and Kaada 1975) have suggested that theta is related to arousal or attention. Vanderwolf *et al.* (1975) have questioned the usefulness of such concepts partly because they are notoriously difficult to define and partly because there is no reason to assume that such psychological concepts correspond to delimitable brain processes. As we have pointed out elsewhere (Nadel and O'Keefe 1974), such vague hypotheses are only useful if they lead to experiments which sharpen the boundaries around the phenomena they describe, increase the precision of the terms they employ, and most importantly provide links with other domains such as behaviour. In the case of the hypotheses under consideration, that theta relates to arousal or attention, one can expect to see analytic experiments which will dissociate the stimulus-processing notion of attention from the more motor-related ideas of arousal or intention to move. Further questions which these well-controlled experiments will answer include: How does theta frequency and amplitude vary (if it does) as the amount of attention demanded by a given task is systematically varied?* What properties does an attended stimulus possess that an unattended stimulus lacks? Can an immobile rabbit discriminate between two stimuli better when it is allowed to generate low-frequency theta than when it is prevented from doing so? Can it acquire some types of discriminations and not others? Up to this time there has been a marked unwillingness on the part of the proponents of the arousal-attention hypothesis to perform these, or similar, studies.

4.6.6. HIPPOCAMPAL EEG DURING LEARNING

We suggested earlier that conditioning paradigms might be useful as analytic tools for dissociating the various possible correlates of theta. As we saw, Black and his colleagues have used the operant conditioning of movement or immobility to test the hypothesis that theta relates to movement and not to other processes such as attention or motivation. Conditioning and discrimination experiments can also be used to generate a series of behaviours with which the hippocampal EEG can be correlated. Alternatively they can be used as a means to study the EEG correlates of the learning process itself. It is in these latter two ways that most investigators have used learning experiments. When we look at these studies it is important to bear in mind the likelihood that many aspects of an animal's behaviour vary during learning in addition to those which are concentrated upon by the investigators.

In several studies hippocampal EEG was recorded during the elaboration of a conditioned reflex. A previously neutral stimulus (CS) is consistently and unavoidably followed by an aversive stimulus (US) such as shock or an attractive stimulus such as food. After training the CS elicits some of the components of the response to the US. Theta has been reported during the CS in aversive-conditioning paradigms in all species tested, but it does not occur as readily in the rat as in other species such as rabbit.

Powell and Joseph (1974) have provided some details which give us clues to the correlates of theta in this situation. They conditioned the corneo-retinal potential in the rabbit, using a shock to the eye as the US and one stimulus (CS+) to signal shock while another (CS–) signalled no shock. In addition to the corneo-retinal potential they recorded heart rate and, in a separate study, movement, neck, and leg EMG. Their measure of theta incidence was the number of trials during which the period of CS presentation was dominated by theta; they did not report theta frequencies. During the early stages of learning, before the reflex had been established, there was a high incidence of theta to both CS+ and CS–; after differential conditioning had occurred, when the CS+ but not the CS– elicited the reflex, there was a considerably higher theta incidence to the CS+. During the early trials there was also a heart-rate decrease to the CS+ without movement or EMG change. Subsequently, when the reflex was conditioned, the heart-rate response to the CS+ involved inhibition followed by acceleration. At this time the EMG indicated considerable movement of the head, but not the legs. These findings indicate that in the rabbit theta is related to some aspect of the conditioned stimulus during the early trials, but increases in incidence during the motor activity of the later trials. These data are consistent with the two-theta proposal noted above; provision of frequency data might have shown that the early theta was primarily low frequency, while the later theta was high frequency.

Preobrazhenskaya (1974) found a similar high incidence of theta during conditioning of leg lifting in the dog. Heart rate also increased to the CS used in this study, and there was good correlation between the amplitude of theta and heart rate. Increases in heart rate during conditioning are usually associated with movement (e.g. Black and de Toledo 1972). However, alimentary conditioned reflexes, which elicited no significant change in theta, were associated with little or no change in heart rate. The most striking increase in theta amplitude was found when a compound discriminated conditioning paradigm was used. The same CS signalled either an aversive or alimentary US, depending on the presence or absence of a second stimulus (the room fan). This association of theta with an uncertain situation has been seen to recur in several studies.

Adey and his colleagues (Adey, 1962, Adey, Dunlop, and Hendrix 1960, Adey, Walter, and Lindsley 1962, Elazar and Adey 1967, Holmes and Adey 1960, Porter, Adey, and Brown 1964, Radulovacki and Adey 1965) studied the hippocampal and entorhinal EEG during the learning of runway discrimination tasks. They found that different patterns of theta were related to different aspects of the task. For example, Radulovacki and Adey (1965), on the basis of the analysis of power-spectra data, discerned three states of the hippocampal EEG: (1) 3-7 Hz, wide spectrum, animal alert but non-performing without overt aspects of orienting behaviour; (2) 4-5 Hz, some variability, animal orienting during discrimination task; (3) 6 Hz regular, animal performing smooth discrimination. These data were obtained from cats tested in a Y-shaped maze on a visual discrimination task. One goal arm was marked with a positive (rewarded) visual stimulus and the other with a negative (non-rewarded) stimulus.

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* The reader will recall that there is no change in the frequency of theta preceding a jump as the distance to be jumped increases (p. 180).

The animal was confined to a start area at the beginning of the trial, during which time a conditioned stimulus was presented; the dominant frequency in the hippocampal EEG increased from 4 to 5 Hz. During the subsequent approach behaviour it moved up to 6 Hz, returning to 4-5 Hz in the goal box. The lower-frequency theta in the start area may have been related to the programming of a movement generator. Holmes and Beckman (1969) have shown that the occurrence of theta during a CS waiting period is a good predictor of whether or not a cat would run when the start door was opened; this was in a task where running was only rewarded on some of the trials.

Although in earlier studies (e.g. Adey, Dunlop, and Hendrix 1960) there was no difference in theta frequency while the animal ran down the alley between correct and incorrect trials, such a difference was reported by Elazar and Adey (1967). Correct trials were associated with 6 Hz theta and incorrect trials with 5 Hz. Whether this was related to successful information retrieval, as they seem to imply, or to some other variable is not clear. Thus they noted 'a confused state with exaggerated reactions denoted an unsuccessful performance from the beginning of the /incorrect/ trials' (p. 232).

In earlier studies (e.g. Adey *et al*. 1960) it was found that the phase relations between theta from an electrode in the entorhinal cortex and another in the dorsal hippocampus changed as a function of learning. These various frequency and phase shifts are taken by Adey as evidence for a hippocampal role in learning. More specifically, he seems to think that the theta wave itself is the carrier of information, producing a state in the hippocampus which enables other structures to store and retrieve memories. He concludes that the hippocampus is

> 'not the repository of memory traces in the sense of a bank or store of such engrams. Through its connections with the diencephalon and more caudal brain stem, it appears to influence, and to be influenced by, activity in sensory systems; and to be responsible for the establishment in these extrahippocampal structures of the physiological "set" that is requisite for storage of information therein. It is a challenging notion that these systems may transact information without the ability to effect its storage in the absence of appropriate interrelations with the hippocampal system' (Adey 1967, p. 228).

This 'deposition of a memory trace in extrahippocampal systems may depend on such wave trains /theta/ and subsequent recall on the stochastic re-establishment of similar wave patterns' (p. 25). P. M. Milner (1971) adopts a similar position when he suggests that the hippocampus is an operator, necessary but not sufficient for trace encoding and retrieval. Unfortunately Adey never spells out what sort of information is contained in the hippocampus and how it can accomplish its purpose without itself storing some kind of information. A more substantial criticism of Adey's

position is that changes in frequency and phase may be less related to learning than to the organism's behaviour *per se.* Differences in theta frequency between correct and incorrect trials are mirrored by differences in behaviour. Similarly, the phase relations between theta recorded from different hippocampal electrodes in the cat are continuously varying and seem to be related to frequency changes more than to anything else (Brown 1968).

4.6.7. HIPPOCAMPAL EEG DURING LEARNING MOTIVATED BY REWARDING BRAIN STIMULATION

In several studies hippocampal EEG was recorded during learning motivated by rewarding or aversive brain stimulation. In one such study in the cat, Grastyan *et al.* (1966) found that rewarding hypothalamic stimulation was associated with hippocampal theta while punishing stimulation produced desynchronization. Different voltages at the same site could produce the two different states. They argued that hippocampal theta is a reflection of a positive approach ('pull') state, While hippocampal desynchronization indicates an avoidance ('push') state.

In contrast, those studies done in the rat have consistently failed to find a relationship between hippocampal EEG and the rewarding properties of ESB (Table A12). For example, Paxinos and Bindra (1970) found that positively rewarding stimulation of the lateral hypothalamus could be associated with theta or LIA depending on whether the rat was reinforced for moving or holding still. Although we found only one report of negatively reinforcing ESB associated with LIA or SIA in the rat (Gray *et al.* 1972) there is no reason to suppose that the appropriate experiment would not reliably produce this combination.

4.6.8. CONCLUSIONS

In summary, then, it is clear that there are two types of theta which can usually be separated roughly (although not exclusively) into high and low frequency bands. In the rat and rabbit low frequency is below 7 Hz and high frequency above 8 Hz. In cat low frequency is 3-5 Hz, while high frequency is above 6 Hz. High frequency, atropine-resistant theta in rat, and probably in other species as well, correlates with spatial displacements, i.e. active movements which, under normal circumstances, translate the animal relative to the environment. Low-frequency theta occurs in all species and is sensitive to intraperitoneally administered atropine. Vanderwolf *et al.* have suggested that one source of this theta, especially in rat, is the activation of a movement programmer in the brain stem without overt movement. The suppression of movement could occur because of a conflict between the intended movement and some other tendency, such as freezing to an aversive stimulus. This pre-programming of movement occurs quite clearly during jumping behaviour. In species other than the rat, low-frequency, high-amplitude theta seems to be related to attention and exploration, although not necessarily to movement. There is a possibility that it is related to eye movements, reflecting the fact that exploration can take place through visual scanning, as well as overt movement, in these species.

High-frequency theta then relates to displacements in the environment; initial theta frequency is a function of the velocity of the displacement. This mechanism functions within our model of the mapping system to shift the focus of excitation from the place representation corresponding to the animal's position at the start of the movement to one whose coordinates fit the distance covered by that movement. The low-frequency, non-movement-related theta might be generated by the misplace system itself, through its connections to the septum (possibly via the subiculum, see pp. 133-8). When there is a mismatch between expected and experienced stimuli in a place, the output of the CA1 field triggers the septum into rhythmical activity which, in turn, drives the CA1 field in the theta mode.* In the rat this is generally masked by the theta triggered by movement, while in other species misplace detection does not necessarily trigger active movements and low-frequency theta is more obvious.

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. In the latter situation the animal is in one place using the map to anticipate stimuli in another place (cat and rabbit) or to guide its behaviour to that place (rat). Without theta input an existing map cannot be modified, but it could be used to distinguish one environment from another and to locate the animal within an environment.**

4.7. Single neurones in the hippocampus of the freely moving animal

There are two fundamentally different approaches to the study of single-unit activity in the freely moving animal. These roughly parallel the two different approaches to the study of behaviour which we outline below (pp. 237-9) and have close affinities with the different types of experiment of hippocampal EEG (see pp. 161-2). The first can be called *neuropsychological* and the second *neuroethological.* The neuropsychological approach draws its inspiration from the methodology of behavioural psychology which has

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* As we shall see there are misplace neurones in CA1 (of freely moving rats) which fire at the theta rhythm when the rat goes to a place and does not find stimuli there which it expects.

** Partial functions such as these could ultimately lay the basis for an understanding of the effects of localized lesions within the hippocampus, thus addressing the question of 'differentiation' of function within that structure. As we will see, there is not sufficient information at present to warrant a detailed analysis of such differentiation.

dominated animal experimental psychology in most countries for the past 30 years. The experimenter addresses himself to a limited number of preordained questions (usually one) about unit activity, and seeks to design a well-controlled study which will answer them. For example, he might want to know whether the unit responds to a particular stimulus or not, whether that response decrements when repetitively elicited, or whether changes in the animal's behaviour or attitude towards a stimulus are paralleled by a change in the unit's responses. Often the stimulus and situation are chosen so as to be devoid of biological significance to the animal at the start of the experiment. The underlying assumption appears to be that the animal's biological constitution and its ecological niche are incidental, or irrelevant, to the behaviour of its neurones. Instead, interest centres on such abstract psychological concepts as learning, habituation, memory, and so on. The units in a particular neural area are treated as a group. 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.

Neuropsychological experiments into the physiological basis of learning are often conducted using tasks from the Pavlovian or Skinnerian literature in the belief that these 'simple' learning tasks reduce the variables associated with learning to a minimum and permit a greater degree of control over these variables than is afforded in other situations. In a thoughtful and thought-provoking article Thompson (1976) has spelt out some of the methodological reasons why his group has chosen one of these tasks, the classical conditioning of the rabbit nictitating-membrane response, as a paradigm for the study of the physiological changes associated with learning. Briefly, the response consists of the movement of the nictitating membrane over the cornea in response to a noxious stimulus in the region of the eye such as an air puff or an electric shock. If this response is preceded often enough by a neutral stimulus such as a tone or a light flash, then that conditioned stimulus begins to elicit the nictitating-membrane response prior to the onset of the noxious stimulus. Some of the advantages listed by Thompson for this preparation include the following: the response can easily be measured and the motoneurones generating it identified; aside from this response the animal is essentially motionless, facilitating electrophysiological recording; the stimulus is well controlled and quantifiable; learning is easy to distinguish from other types of changes: the conditioning stimulus does not elicit the response before conditioning begins and there is no pseudoconditioning or sensitization as a result of unpaired presentations of the tone and the air puff. Thompson and his co-workers have begun to work out some of the physiological properties of this simple learning. We shall mention some of their findings on hippocampal neurones shortly. Here let us briefly examine the use of such preparations in the study of learning.

The first assumption which must be questioned is that the response selected for measurement is the only one being emitted or programmed. There are several reports of animals which have been conditioned in these Pavlovian paradigms to perform simple salivary responses, revealing quite complex behaviours when they were freed from their harness and allowed to. Lorenz (1969) cites an anecdote in which Liddell set free one of Pavlov's dogs after it had been conditioned to salivate to the change in frequency of a metronome.

> 'The dog at once ran to the machine, wagged its tail at it, tried to jump up to it, barked, and so on; in other words, it showed as clearly as possible the whole system of behaviour patterns serving, in a number of Canidae, to beg food from a conspecific' (1969, p. 47).

Earlier, Zener (1937) had reported similar findings. His dogs had been trained in a Pavlovian paradigm with either food or acid as the unconditioned stimulus and salivation as the response measured. When freed, the dogs tended to approach the food-related stimuli and to avoid the acid-related one. More surprising, when the dogs were tested with the same conditioned stimuli but on another platform in another part of the same room, two of four left the new position and went to the old table to look for food during the first presentation of the food-related stimulus but not during the acid-related stimulus.

One must surely surmise that, at least for some of the animals in these simple paradigms, the learning which takes place and the responses programmed (even if not emitted) may be considerably more extensive than what is measured by the experimenter. In terms of the model advanced elsewhere in this book (pp. 89-101) we suspect that a localized conditioned stimulus acts as a guide or a nemesis (see also Hearst and Jenkins 1974) and that locale hypotheses are also activated. Restriction of the animal's movements or responses does not prevent this, it only masks it. The general methodological principle here is that the learning situation must be structured so as to allow the animal to emit all of the responses which might be programmed and to enable the experimenter to probe the situation to test for the different hypotheses which could be generating a particular behaviour (see pp. 237-9). The complexity of the testing situation should be matched to the brain systems underlying the behaviour and not to the behaviour itself.

A similar objection can be raised against the assumption that a stimulus with a simple physical characterization is simple from the point of view of the nervous system. Lorenz's interpretation of Liddell's dog as begging food from a conspecific is not far-fetched, and one would not be surprised to find that neurones in that animal's brain which were normally involved in identifying conspecifics could now also be activated by the conditioned stimulus.

After conditioning, then, neurones influenced by the conditioned stimulus could range from ones related to the sensory properties of the stimulus to ones conferring valence on the stimulus, or identifying it as a conspecific, or locating it in a cognitive map of the environment.

Both Ranck and Vanderwolf have discussed this problem of interpreting neural changes which occur during learning in the absence of a good deal of information about the units (or EEG) being studied. In a privately circulated paper Ranck (1972) has suggested that many changes which will occur during learning will be incidental to the main learning process:

> 'if one were to record from a neurone in the striate cortex while the rat learned a tone-shock avoidance, no doubt some changes in the neurone could be recorded, if for no other reason that the rat might well learn a habit of gaze during avoidance. If the learning occurred, this change in firing would no doubt change systematically as the visual response of the animal systematically changed. Pseudoconditioning, habituation, and extinction controls would probably not conflict with the interpretation that this was real learning since the visual behaviour during the controls would probably be different from the learned case' (p. 10).

Vanderwolf has been labouring the same point for several years now. In exasperation, he has invented the intrepid Professor Omega:

> 'Consider the case of Professor Omega, an imaginary researcher who suspected that the somatic muscles had a direct role in mental processes. Omega began his research by recording electromyographic activity (EMG) from m. pectoralis major during behaviour in animals. His first finding was that on exposure of an animal to an unfamiliar Skinner box, EMG activity was high initially but declined progressively during continued exposure. Presentations of tones and flashing lights led to temporary increases in EMG activity. These findings suggested a role of the muscle in habituation and attention. When the animal was trained to press a lever in the box, EMG activity rose to a high level. Extinction procedures resulted in a decline to pretraining levels. These facts suggested a role for the muscle in conditioning and learning. Further research showed that EMG activity rose during food deprivation and fell after feeding, suggesting a role in motivational phenomena. Amphetamine increased EMG activity sharply, but anaesthetics abolished it. Tranquillizers had an intermediate effect. Omega concluded that the pectoralis muscle was probably the site of action of many drugs.
>
> To his surprise, Omega had great difficulty in publishing his results in scientific journals. In response to criticisms of his work, he pointed out that the methods and experimental design he had used were identical to those adopted by many researchers who had published papers purporting to show that the slow wave or unit activity of the neocortex, the hippocampus, and many other parts of the brain is related to attention, learning, motivation, etc.' (Vanderwolf 1976).

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. When we first venture into the unknown, we need not the incisive beam of the proud penetrating laser, but the gentle diffuse illumination of the humble torch. There will be plenty of time later for detailed investigation of the nooks and crannies; first we must find the mountains. The neuropsychological approach is too myopic for this job. At the rate of one or two questions per experiment, it will be many years before we hit upon the correct question, even with a good bit of luck and insight.[*] We 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.

The neuroethological approach differs from the neuropsychological one in several respects. First, it seeks to study the activity of single units in as naturalistic a setting as possible, in the belief that an animal's behaviour in its natural environment maximizes the possibility of producing changes in unit activity that are meaningfully related to that unit's function. It thus embodies the reasonable assumption that the brain of a particular animal is built to operate in a specific environment. At the very least, this entails an environment to which the animal is accustomed and within which it can move about, eat, drink, groom, establish a territory, and occasionally encounter conspecifics. At best, it entails recording units in the animal's natural habitat.

A second principle of neuroethology is that, unlike the neuropsychological approach, it seeks not to ask a pre-ordained set of questions about a population of units, but instead treats each unit as an individual and attempts to describe the full range of its behaviour in as many different situations as possible. Once the range of a unit's activity has been defined, its specific correlate can be worked out using a set of probes similar to those to be described later for work in lesion experiments (pp. 237-9). There is a pious hope that some commonality of behaviour will emerge for the units of one neural area as opposed to those of another.[**] Since this is the ultimate goal it is important that the investigator address each new unit of a neuroethological experiment with a healthy scepticism. He must temporarily purge his mind of as many preconceived notions about the function of that particular unit as possible. He must be willing to entertain the possibility that the unit could be responding to anything from a very specific event, such as the contraction of a single muscle or changes in illumination on a part of the retina, at one end of the continuum to very abstract psychological processes, such as attention or thinking, at the other. This is not to say that he should not hold a general model of how the nervous system works or even what function is subserved by the structure being studied; the Popperian scepticism (Popper 1959) is necessary because, in order to demonstrate that a unit's function is related to some abstract concept, it is necessary first to rule out less exotic possibilities such as muscle contractions (see Vanderwolf's point above, p. 193). As we noted in the last section, a similar imperative exists in, but has rarely been employed with, the work on the correlates of the hippocampal EEG.

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[*] It is worth observing that, in so far as information does exist to guide such experiments, it seems to be ignored. Thus, many unit experiments use task situations which are not affected by lesions in the hippocampus, such as incentive conditioning and habituation to sensory stimuli.

[**] The search for this commonality would be facilitated if paradigm units exist. These display the salient features of all units in an area in a particularly clear and uncomplicated way. On the other hand, the belief that one has found a paradigm unit can produce premature mental closure about the function of an area and introduces a strong source of bias in the testing of subsequent units.

4.7.1. A NEUROETHOLOGICAL STRATEGY

The strategy used in our own work on single-unit activity in the hippocampus of the freely moving rat leans towards the neuroethological, rather than the neuropsychological, approach. The following is a general outline of this procedure. Let us assume that one has isolated a stable unit and has observed that it increases its firing rate several-fold during some behaviours but not others.[*] The next step is to ask a series of questions in the order dictated by a branching-tree structure. In such a structure the answer to each question leads one down one branch of the tree rather than another and thus determines all subsequent questions. The questions are arranged in the order of more general to more specific, much in the manner of the twenty questions game (animal, vegetable, or mineral). The first question is whether the unit is responsive to something in the environment or to something which travels with the animal such as body movements, internal body states, autochthonous thoughts, etc. In general, the latter will occur independent of the animal's position in an environment. For example, neck proprioceptors will fire during several different types of head movements and will fire whenever the rat makes those head movements, irrespective of its position in the environment. Of course, this distinction between environmental and personal sources of input is not absolute, nor are these classes mutually exclusive. A unit might only fire when the rat actively turns its head to the left *and* stimulates a portion of its retina with a vertical line. Once the role of proximal or internal sources of input has been defined (or excluded) the nature of any distal influences can be investigated by systematically excluding first vision

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[*] At this stage in our study of unit activity in limbic and other brain areas, we should concentrate on large changes in unit activity and ignore subtle effects which require averaging or other computer manipulation for their demonstration. A quick perusal of the important papers in the field of unit recording in the last decade does not reveal any responses which were not clearly detectable by ear and eye. On the contrary, such phenomena as habituation, learning, and the influence of the animal's behaviour on sensory inputs would strongly militate against any premature data reduction. Once we know the major correlates of a unit we can use this knowledge to aid in the study of lesser influences. For example, the knowledge that a unit fires during eye movements enables one to control this variable while studying the influence of various sensory inputs on the unit.

(lights out), audition (ears plugged), and proximal olfactory or tactile inputs (change floor). If one finds that the unit response is abolished by turning off the lights, this enables one to conclude that the visual input is necessary for activation; in order to demonstrate that it is sufficient, one must then eliminate all other modalities and try just the visual inputs. This, of course, is much more difficult, since it is possible that there are many types of stimuli of which the experimenter is unaware or which he is not usually in a position to exclude (e.g. geomagnetism). The alternative is to try to narrow down the location of the source by blocking off parts of the stimulus field. In the case of the visual field this could involve blocking off successively smaller parts of the visual field using curtains. At this point the research strategy could switch to the classical sensori-motor technique of looking for receptive fields in the secure knowledge that only the relevant stimulus or stimuli is being studied.

One of the important advantages of this method which must be stressed is that it makes no assumptions about the source of the unit activation. As we shall see later in this section, hippocampal units do not respond to individual stimuli but appear to signal that an animal is in a particular place. It follows that an exhaustive search through all of the stimuli in the universe might fail to activate these units. Although a further search through all of the possible spatial combinations of stimuli, time permitting, might locate one to which the unit responds, the search would have to continue indefinitely in order to reveal the full complexity of these units.

Note that with the use of this strategy the units in an area will not have been treated equally unless they are all identical. Each one would have been followed down a different branch of the logic-tree structure. It is therefore not possible to construct tidy tables of the number of units responding to one stimulus as opposed to another. On the contrary, the function of a neural structure must be inferred from the similarities between the logic trees. This may turn out to be a similarity near the top of the tree, e.g. all units respond to visual stimuli, or it might be lower down, e.g. all units respond to stimuli from a particular part of taxon space, or it might be something about the structure of the tree itself, e.g. all units are multi-modal or require a stimulus plus a body movement, or, finally, it may be necessary to abstract a property from the commonality amongst the tree structures, e.g. all units respond to ethologically significant stimuli or to abstract concepts such as places.

4.7.2. TYPES OF UNITS IN THE HIPPOCAMPUS OF THE FREELY MOVING RAT

In our experience (O'Keefe and Dostrovsky 1971, O'Keefe 1976; O'Keefe and Conway 1976) the units in the CA1 field of the hippocampus of the freely moving rat fall into two general classes: *place* units and *displace* units. Roughly speaking, the displace units are those whose firing pattern relates

to the behaviour of the animal, irrespective of where it occurs in an environment, while place units are those whose firing pattern is dependent on the location of the animal in an environment. These definitions will be made more precise towards the middle of this section. In particular, the definition of a place unit will be made to include the fact that it does not respond to any single stimulus in an environment.

For some place units the rat's location in an environment is a necessary, but not sufficient, condition for unit firing; in addition, the animal must be behaving in a specific way or receiving a specific stimulus. Included in this last category of complex place units are *misplace* units which fire maximally when the animal engages in myostatial sniffing in a place either because the object usually there (e.g. reward) has been removed or in some cases because there is a novel object there.

4.7.2(a). Physiological differences between place and displace units. The two general classes of units in the CA1 field, place and displace units, can be distinguished not only on the basis of their behavioural/physiological correlate but also in terms of such fundamental parameters as the rate and pattern of spontaneous activity, width of the action potential, etc. Ranck (1973) has listed many of these differences between what he has termed complex-spike units and theta units. All of our place units show the complex-spike configuration.* A complex spike is a burst of several spikes within a brief period (inter-spike interval 1.5-6 ms) where successive spikes have differing (usually decreasing) amplitudes. On the other hand, displace units never fire with a complex spike and are clearly identical to the theta units described by Ranck (1973) and Feder and Ranck (1973). Table 4 lists the differences between complex-spike units and theta units.

As Ranck has emphasized, it is possible on the basis of these physiological characteristics to decide within a few seconds of encountering a unit to which class it belongs. The differences clearly suggest that the two types of units are generated by two different anatomical elements. The close correspondence between the distribution of complex-spike units and the soma of pyramidal cells on the one hand, and between the theta units and the soma of the basket cells of Cajal on the other, has led Fox and Ranck (1975) to assert the identity of the two. Although our own unquantified impressions about the spatial distribution of the two types of unit in CA1 agrees with the careful observations of Fox and Ranck and leads us to accept their conclusion, in the absence of more direct evidence one must keep other possibilities in mind. In order to make a conclusive argument for identity of complex-spike units with pyramidal cells, and theta units with basket cells, one would need either selective antidromic activation of the complex-units, but not the theta units, from the projection sites of the pyramidal

* Babb, Carr, and Crandall (1973) have recorded units with this complex-spike configuration in humans.

TABLE 4

Differences between theta cells and complex-spike cells in the hippocampus

		Theta cells	Complex-spike cells
1	*(a)* Complex spikes	Never	All have some
	(b) Simple action potentials	Always	All have some
2	Duration of extracellular negative spike (distorted)	All 0.15-0.25 ms	All 0.3-0.5 ms in single spikes and spikes of complex spikes
3	Rate of firing most of the time awake and SWS	Almost all > 8/s	All < 12/s, most < 2/s, many off *
4	Maximum rate of firing	29-147/s, sustained for many seconds	All < 40/s, most < 20/s sustained for less than 2 s *
5	Patterns of firing	Comparatively regular	Irregular
6	During theta rhythm in slow waves in paradoxical sleep or awake		
	(a) Rate	At maximum rate if and only if theta rhythm is present	No simple relation usually < 1/s *
	(b) Phase relations	Most have clear phase relation	Most have clear phase relation
7	Relation to LIA spike	Almost all fire with bursts	Sometimes fires
8	Spike heights	Usually <200 µV (mean = 164 µV)	Larger than theta units (mean = 267 µV)
9	Anatomical location		
	in CA1	Stratum pyramidale Stratum oriens	Stratum pyramidale
	in CA3	Stratum pyramidale Apical dendritic layers	Stratum pyramidale
	in F.D.	Stratum granulosum Hilus of F.D.	Stratum granulosum

* A complex spike is counted as a single potential. From Ranck (1973) and Fox and Ranck (1975).

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cells (the subiculum in the case of CA1) or an intracellular dye mark of the two types of unit. Andersen (personal communication) has done the antidromic experiment in anaesthetized rabbits and found units with close relations to EEG theta which project out of the hippocampus and cannot, therefore, be interneurones. If these observations were repeated in the unanaesthetized rat they would create extreme difficulties for the Fox and Ranck hypothesis. It should be noted, however, that phase locking to the hippocampal EEG is not a strong criterion for distinguishing between complex-spike units and theta units, since the former can show strong phase relations to theta under certain circumstances (see, for example, Figs. 24 and 25). Frederickson (1974b) has suggested that the spatial distribution of the theta units in the fascia dentata would fit the distribution of the terminals of septal afferents. It is possible that these have a special morphology which would enable their activity to be recorded by a microelectrode in much the same way as a soma spike.

4.7.2(b). Displace units. The displace units have the same general relationship to behaviour as has been described for the hippocampal EEG by

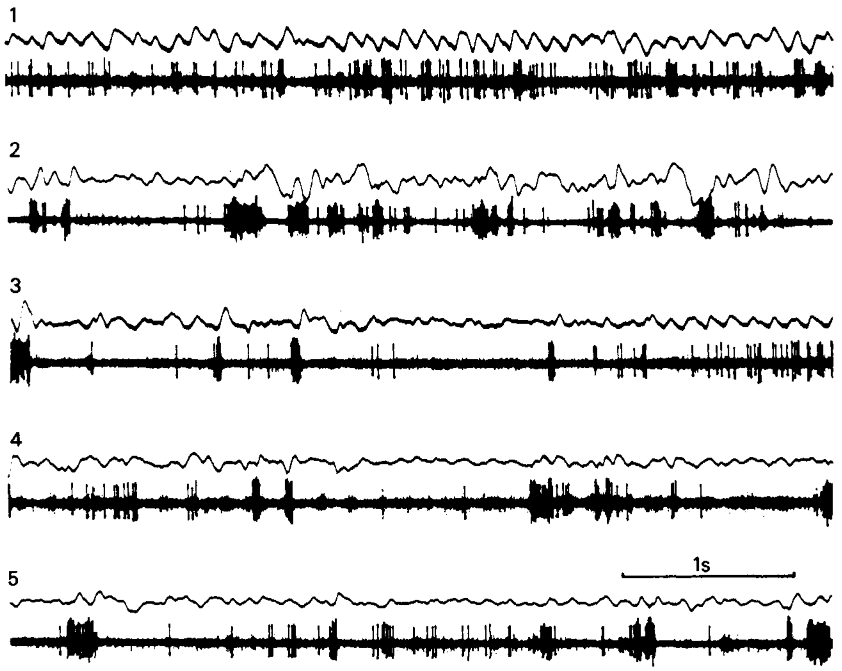


FIG 20. A displace unit with poor phase relation to the theta recorded from the same microelectrode: 1 exploratory sniffing, 2 lying down awake, 3 drinking water. Towards the end the animal sniffs around the water bowl, 4 eating a large food pellet, 5 face washing. Up is negative for both units and slow waves in this and subsequent figures. (From O'Keefe 1976.)

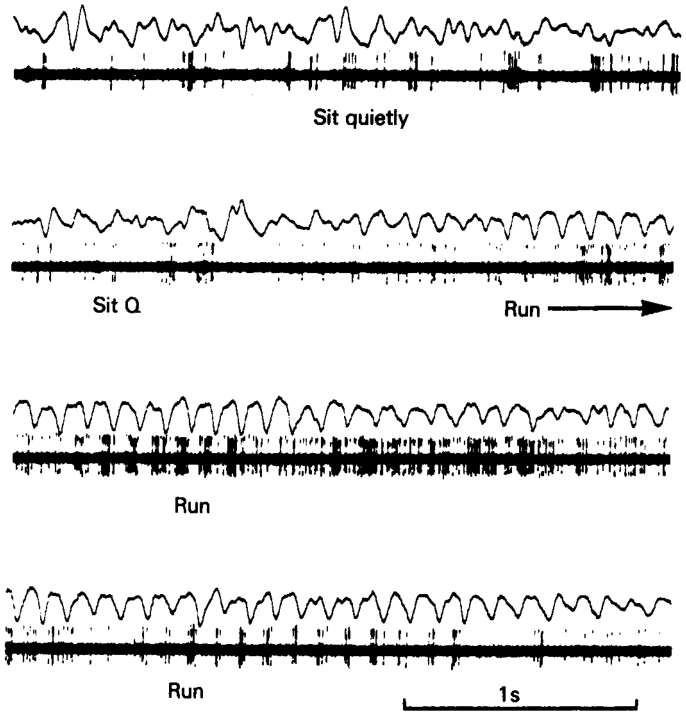


FIG. 21. A displace unit with good phase relation to the theta recorded from the same microelectrode. The third set of traces is a continuation of the second. (From O'Keefe, unpublished.)

Vanderwolf and his colleagues (see pp. 173-4). Their relation to the EEG slow wave and to the animal's behaviour have been described extensively by Ranck (1973) and Feder and Ranck (1973). During behaviours when the hippocampal EEG is dominated by the LIA pattern (e.g. eating, drinking, grooming, sitting quietly) the displace units fire randomly at a low rate (Fig. 20, 2-5). Interspersed with these random responses are high frequency bursts associated with the EEG spike often seen in the LIA pattern (see pp. 150-3). Much the same rate of firing is recorded during all these different behaviours.

In contrast, there is an increase in overall firing rate during behaviour associated with theta activity in the hippocampal EEG (Fig. 20, 1). As noted earlier, in the rat these are primarily behaviours which change the animal's position relative to the environment, such as exploration, sniffing, rearing, walking, and jumping. Furthermore, during displacement behaviours, many of these units exhibit a marked bursting pattern, each burst synchronized to a particular phase of the concurrent slow-wave EEG theta. Fig. 21 shows a good example of this phase locking. As can also be seen

from this figure, there are periods when the unit fails to phase lock to the EEG theta and may cease firing altogether. It is not clear, at present, under what circumstances this occurs. If some displace units continue to burst while others momentarily cease to do so, this could be the primary means by which information is transmitted in these neurones. As the firing frequency of these units increases, the phasing of units to slow waves becomes nothing more than a slight pause in firing at a certain part of the wave. Ultimately, with very high frequencies, some units fire continuously and no phasing is seen. Some displace units show only slight phase relation to the EEG theta but still markedly increase their firing rates when theta is present. During behaviours associated with high-frequency firing in displace units, the amplitude of the EEG theta may actually drop. As suggested earlier, the amplitude of theta is not uniquely related to the number of spikes within a burst or the number of active displace units. Some units fire with the positive-to-negative phase of theta from the same electrode, while others are off during this same time. As we pointed out in a previous section, if the displace units are interneurones it would seem unlikely that these two varieties are both projecting to the same parts of the same pyramidal cells, since their activity would tend to cancel out. The most likely alternatives are that they project to different pyramidal cells or to different parts of the same pyramidal cells.

4.7.2(c). Place units. The second class of units in the CA1 sector of the dorsal hippocampus are those whose firing is dependent on the rat's location in an environment. The part of the environment where the unit fires, or fires maximally, is called the *place field.* Some units fire maximally when the rat is in the place field and, in addition, performing a specific behaviour, such as sniffing, or receiving a specific stimulus. Some of these units are almost certainly identical to the units which Ranck (1973) has called approach-consummate-mismatch cells. It is also possible that his approach-consummate units are place units, since they have such a close resemblance to place units which fire best when the rat runs past a place. Most of our place units fire at a rate of considerably less than 1/s when the animal engages in a theta-related behaviour outside of the place field. During LIA behaviours the rate increases to about 1/s, irrespective of the animal's location. Ranck (1973) has emphasized his finding that for many units there is a greater firing rate during one of these LIA behaviours than during theta. In our work we have not found striking differences between firing rates during eating and drinking in the same place, and have concentrated on the increase in firing related to the animal's position in the environment.

In our original experiments the rats were tested on a platform open to the environment in order to maximize the chance of finding units responsive to environmental influences. Figs. 22 and 23 show two units

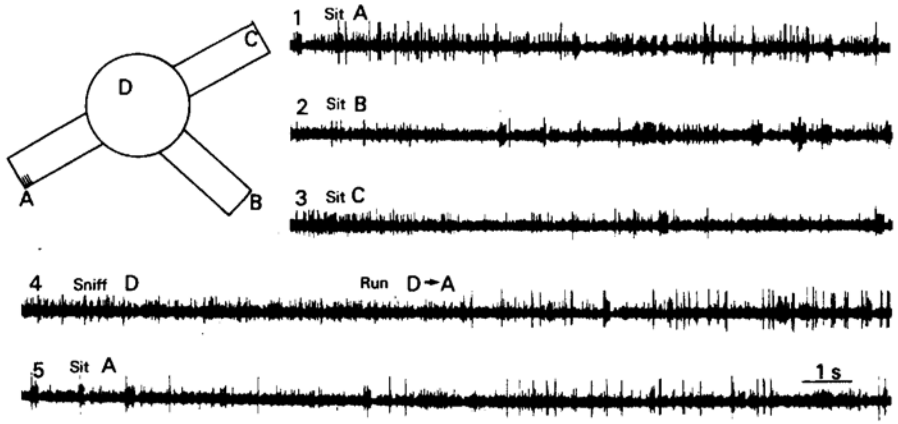


FIG. 22. Place unit. The letters in this and subsequent figures refer to the place on the maze where the behaviour occurred. In this experiment the rat had been made hungry and thirsty, and trained to run to a small porcelain dish wherever it was placed on the elevated T-maze. When placed in arm A, the dish contained three or four 45-mg food pellets, in arm B, water and in arm C, sweet condensed milk. The maze had no walls, maximizing the amount of spatial information available to the animal. Sit does not mean that the animal was totally immobile. The field indicated on the maze is the location of the rat's head during maximal unit firing. Unless there are indications to the contrary (as in 4), the behaviour described at the beginning of the trace continues for the duration of the trace. In this figure there is a smaller displace unit in the background. The record in 1 was taken 45 s after the record in 4; 5 shows unit activity while the rat sits at A with a new arm substituted for the usual A arm and the room darkened. (From O'Keefe 1976.)

from these studies, one which fired whenever the animal went to a part of the environment, the other when the rat ran past a part of the environment. Fig. 24 shows a more complicated type of place unit, one which fired maximally when the rat did something in a place. In this case the unit fired to some extent when the rat sat in the place field, but only reached its maximal rate when the rat sniffed off the edge of the maze without making contact with the object towards which he was sniffing. Many of the complex place units fire maximally during myostatial sniffing elicited in a place, either by the absence of an expected object or by the presence of an unexpected one. We have called these units *misplace units,* although as yet there is no conclusive evidence that they are actually signalling a mismatch between an internal representation and an input. Perhaps some of them are merely signalling that the rat is sniffing in a place. Note, however, that the behaviour of the unit shown in Fig. 24 is *not* amenable to this description since the firing depended not only on the place and the myostatial sniffing but also on whether contact was made with the object.

We have concentrated on determining what the place units are influenced by in the environment. In the original experiments attempts to answer this question were aimed at the exclusion of inputs from a whole

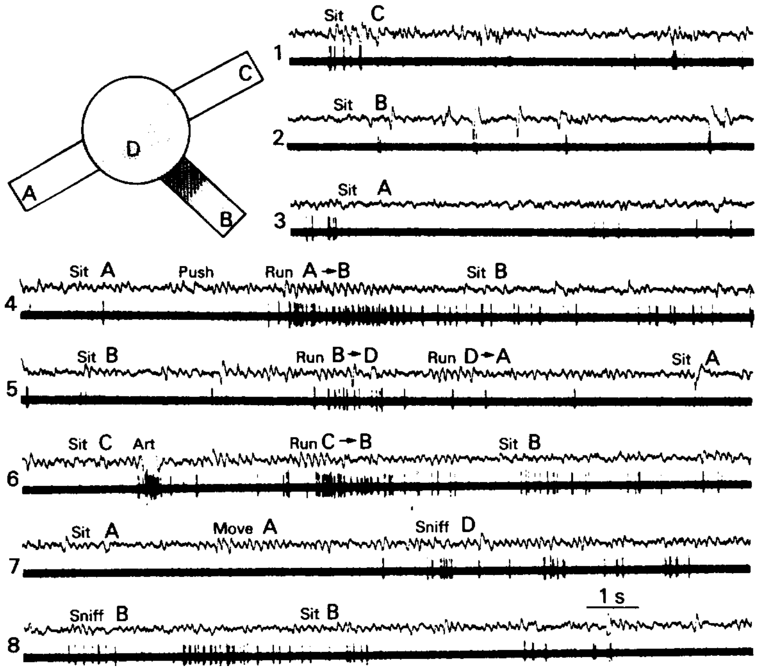


FIG. 23. Another place unit. There may be two units with the same field here, one of smaller amplitude than the other. Maximal firing was obtained when the rat ran rapidly into arm B from either arm A (4) or from arm C (6). Less firing occurred and the field was smaller if the animal ran slower or out of arm B into one of the other arms (5). Maximal field is shaded; minimum cross-hatched. 7 and 8 show the firing on the second trial after a new arm had been substituted for the B arm and the animal slowly sniffed its way from A to B. Notice the continued firing in B when the rat enters it after crossing the place field (6). This firing ceased after about 10 s in this unit. Art at beginning of 6 is artifact due to a vigorous headshake. (From O'Keefe 1976.)

modality at a time by, for example, turning off the room lights or by changing the intra-maze stimuli. For many units it was clear that the place response persisted even when the visual and local olfactory/ tactile cues were removed or altered. The testing situation did not allow for inputs in other modalities to be adequately tested, but there were several observations which suggested that the place of response was not defined by some simple stimulus. First, several place units were studied while the rat was held in the appropriate place after the maze arm had been removed. In no case did the unit fire, suggesting that 'being on the maze' was part of the definition of the place. A second observation was that for several units the response in the place field was lost when the room lights were turned off, but reappeared on all subsequent tests without light if the animal was given further experience in the light (Figs. 22, 5 and 24, 5,6). This suggested that, under normal conditions, the unit was strongly influenced by some

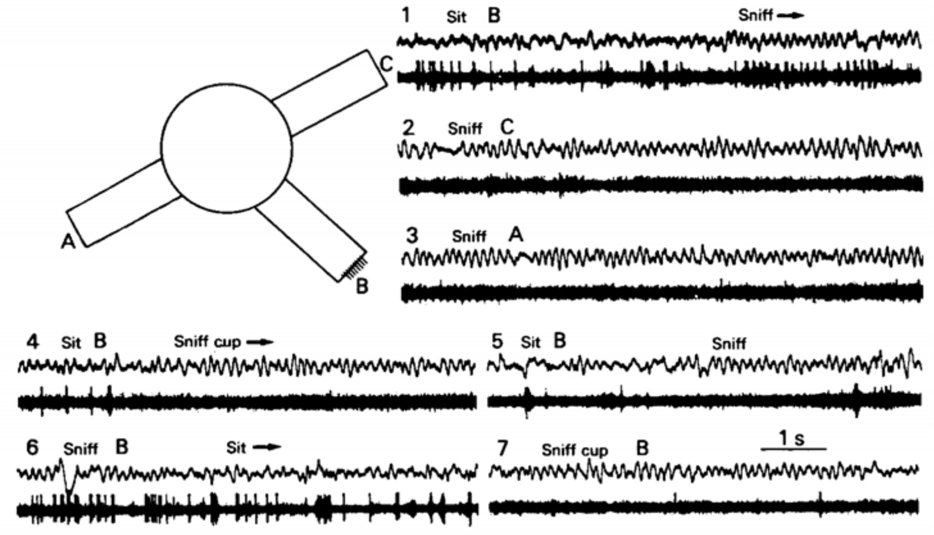


FIG. 24. Misplace unit. 1-3, Sniffing at empty food cup held off arms B,C,A respectively without making contact. Note the lower rate of firing in B when the rat sat without sniffing (begin 1). 4, Sniffing off arm B and contacting the cup; unit is silent. 5-7, Effect of turning room lights out on unit firing in B arm. 5, First-time lights out, no firing during sitting in B or sniffing off arm B without contacting cup. 6, Second time (and subsequently) lights out, normal firing during sitting in B or sniffing off arm B without contacting cup. 7, Absence of unit firing during sniffing off B and making contact with cup, lights out. (From O'Keefe 1976.)

visual stimulus in the environment, but that when that source of information was lacking it could use inputs from other modalities to identify its place field. Finally, in some units alterations in the environment, such as shutting off the lights or replacing the maze arm, resulted in changes in the place field which could not readily be interpreted as being due to the removal of a single excitatory stimulus. For some units there was an increase in the size of the place field, for others, a new field opened up in addition to the old one, and finally in one unit there was a subtle but reliable shift in the location of the place field. This last unit has been described extensively (O'Keefe 1976) since it offers insights into the formation of the place field; the reader is referred to the original paper for a full description of this unit. Here, we shall only describe those aspects of the field which are pertinent to the present discussion. The unit had two fields on the maze. One was located at the end of the arm where the animal usually received food pellets; this arm faced a large window (Fig. 25, top). The unit fired when the animal sat at X, but not when it sat at Y, W, or V (Fig. 25, 1,2,3). When the window blind was drawn, there was a shift in the field from X to W and the opening up of a new field on the

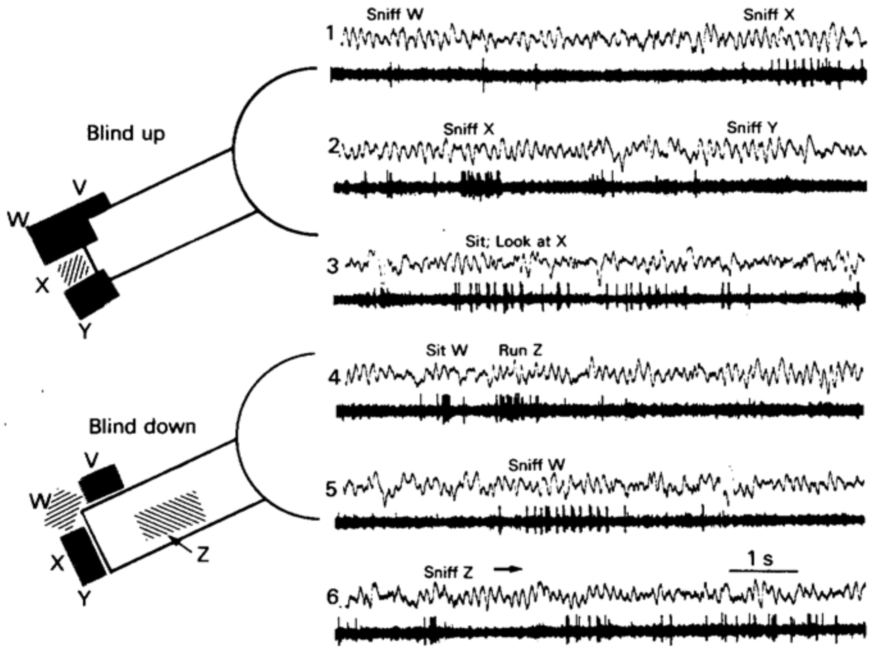


FIG. 25. Place unit, changes in place field in arm A as a result of drawing the window blind. Traces 1, 2, 3 the blind open; 4, 5, 6 blind closed. 6, Room lights out and new arm substituted for A; unit responds to whole arm. The hatched area shows place field; the gridded area shows portion of maze adjacent to the place field where the unit did not fire. (From O'Keefe 1976.)

same arm at Z (Fig. 25, 4,5). When the lights were turned off, and a new arm substituted for the original, the unit fired to the whole arm (Fig. 25, 6).

Of course, all of the above observations are at best suggestive, and it is clear that one can never prove that the response of the place units is not due to some specific sensory stimulus on the basis of an inability to find that stimulus. What is required is a demonstration that these place units will fire in places in an environment where the stimuli are controlled and limited. Preliminary results from such a study are now available (O'Keefe and Conway 1976).

The environment consisted of a 7 ft square set of black curtains within which was set a T-shaped maze. On the walls formed by the curtains were four stimuli: a low-wattage light bulb on the first wall, a white card on the second, and a buzzer and a fan on the third and fourth, respectively. Throughout the experiment the location of the goal arm of the T-maze and the four stimuli maintained the same spatial relationship to each other, but all other spatial relations were systematically varied. From trial to trial we varied (1) the spatial relation of the four stimuli and the goal to the external world (2) the physical arms serving as start, goal, and non-goal, and (3) the body turn required to reach the goal.

Rats were taught a place discrimination in this environment. They were made hungry and taught to go to the goal arm as defined by its relation to the four stimuli within the curtains in order to obtain food. After they had learned the task, place units were recorded. In order to relate the firing of these units to the animal's position in the environment, advantage was taken of the fact that these units have low spontaneous activity when the rat engages in RSA-related behaviours (such as running) outside the place field. Each action potential from the unit was used to trigger a voltage pulse which, when fed back to a light-emitting diode on the animal's head, produced a brief flash of light. A camera on the ceiling of the environment photographed the spots, recording directly the firing of the unit relative to the environment.

After the rat had mastered the place discrimination it was taught to run out of the goal arm into the non-goal arm and thence into the start arm, where it received an additional mouthful of food. This behaviour pattern resulted in the animal visiting the whole maze on each trial, thus ensuring that the unit had an equal opportunity to fire on all parts of the maze.

Preliminary results show that some place units have a place field in this environment, and that this field remains constant relative to the four stimuli within the environment and not to the external environment, nor to a physical arm, nor to the body turn which the rat makes before, within, or after the place field. This is not to say that there may not be an influence of the animal's behaviour on the unit, only that this is not sufficient of itself to elicit the response.

Fig. 26(a) shows a response pattern of a place unit recorded in this curtained environment. Notice in the top four pictures that the unit always fired in the start arm when it was opposite the light, irrespective of the position of the start arm relative to the external environment. This is revealed more clearly in Fig. 26(b) where these four pictures are superimposed upon each other in different ways. Only when all the mazes and stimuli have the same orientation do the dots superimpose (Fig. 26 (b)). The bottom two pictures of Fig. 26(a) show that the unit did not fire in the start arm when it was positioned on the side of the T closest to the light. This rules out the possibility that the unit was signalling that the rat was in the start arm or merely responding to the arm where it was placed down on the maze.

Finally, Fig. 27, A and B, showing the activity of the unit when the four stimuli were removed from the environment, confirms the dependence of the place field on these stimuli. Notice that this unit, like the one described above (Fig. 25), reacted to the removal of the stimuli by firing to a much larger area of the maze than when the stimuli were present. We do not have enough data at this time to know whether the apparent absence of firing

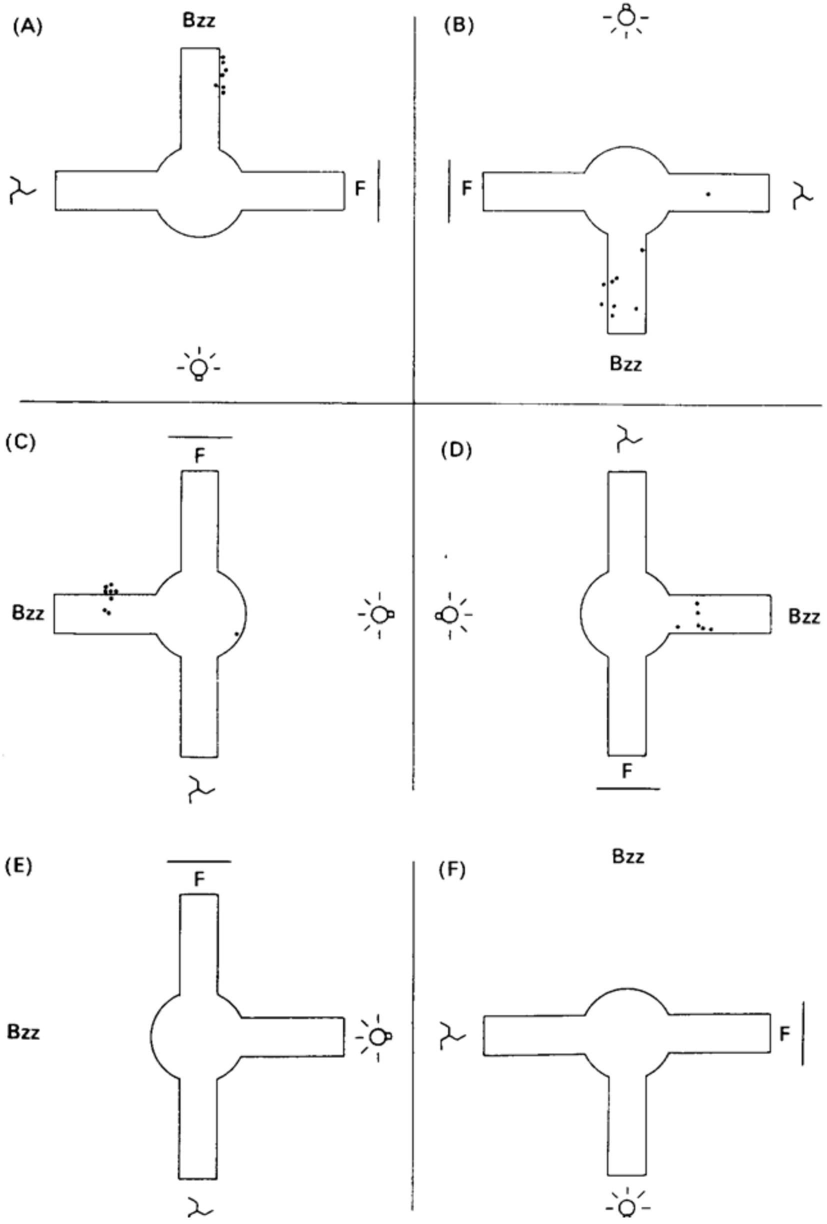


FIG. 26(a). The firing of a place unit when a rat is on the T-shaped maze inside the cue-controlled enclosure. Each dot represents one action potential. Four ground trials are shown in A-D in which the T-maze and the cues on the wall have four different orientations relative to the external world. The orientation of the external world is constant both in this figure and Fig. 27. Note that the unit fires when the rat is in the start arm when it is on the side close to the buzzer, regardless of the orientation relative to the external world. E and F show two ground trials with the start arm rotated 180° so that it is on the side close to the light. There is no unit firing in the start arm. (O'Keefe and Conway, unpublished)

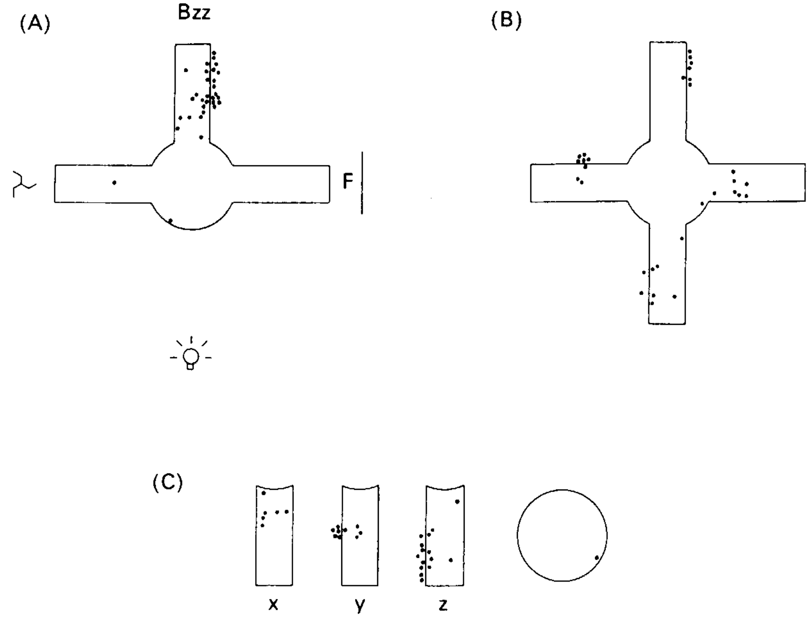


FIG. 26(b). The same unit as in Fig. 26(a), showing the results of trials A-D superimposed in different ways. A: pictures aligned with the same orientation to the controlled cues on the wall; B: pictures aligned with the same orientation to the external world; C: physical components of the maze separated and superimposed. Note that the z arm was used as the start arm *twice* while the other arms were used only *once* each. (O'Keefe and Conway, unpublished)

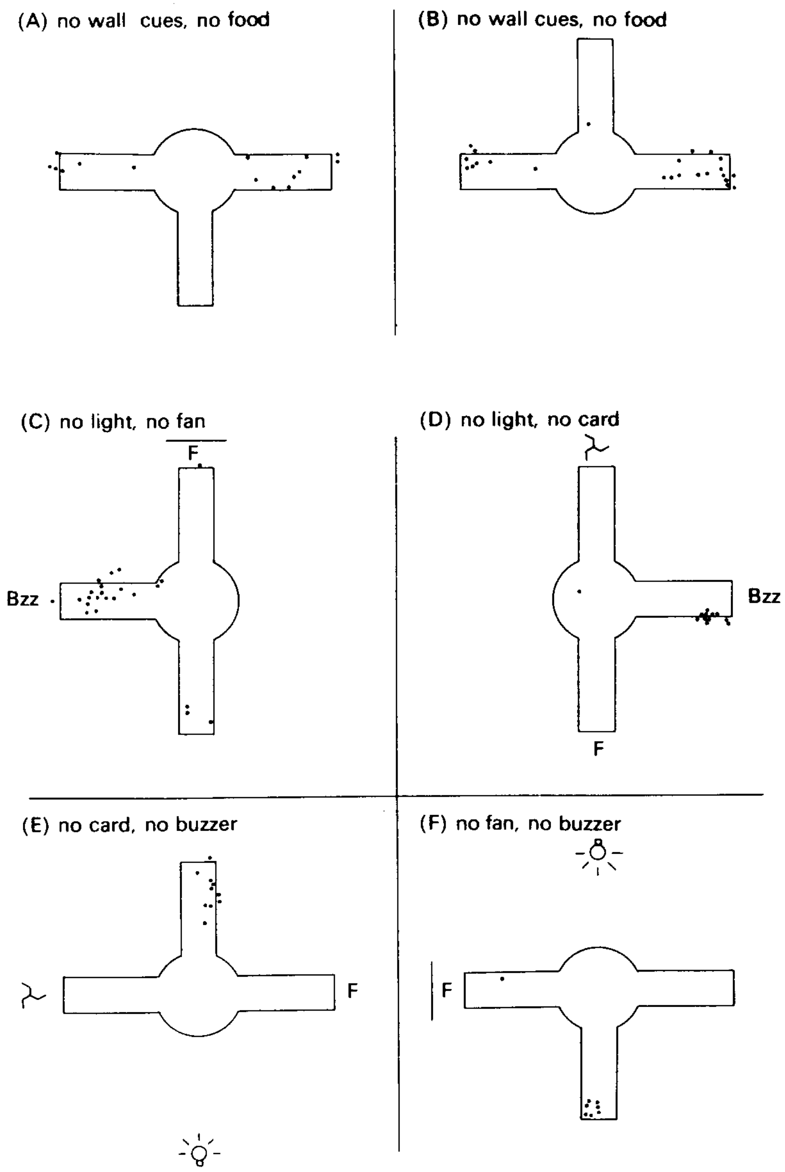
of the unit in the start arm, which was observed on these trials, will be found in the majority of units of this type. Nor do we know what was responsible for this feature of the unit's responsiveness; it is obviously of considerable theoretical interest. It should be noted that, although some units increase their field size and firing rates when the stimuli within the environment are removed, others simply stop firing altogether.

For some of these place units it has proved possible to ascertain whether the place field was dependent on any specific stimulus within the environment by removing each of the four stimuli during different probe trials. In some units two stimuli at a time were removed. In general, the units' place fields survived intact if the animal could perform the place discrimination. There have been enough exceptions, however, for us to feel confident that the place units are not merely reflecting the animal's behaviour. Almost all animals, and place units, behave normally after the removal of any single stimulus. This is the theoretically crucial finding, and demonstrates that the units are not responding to the position of one of the stimuli in egocentric space. When two stimuli are removed many animals have difficulty with the task and many place units begin to show changes in their field. Different units prefer different combinations of stimuli, but in general the light seems to be the most salient cue in this situation. Fig. 27, C-F show the effect of removing two stimuli at a time for the unit illustrated. Only when both the fan and the light were removed together was there a significant change in unit firing. We interpret this type of change (increased firing primarily in and around the usual place field) to mean that the cell still identifies the animal's position but also signals a change in the stimuli impinging on the animal in that place.

As we have seen, then, there exist two apparently different classes of place units: one which stops firing when sufficient cues are removed from the environment, and a second which increases its firing when the necessary cues are removed and begins to fire in parts of the maze where it was previously silent. We can speculate that for the first group of units the place field derives from the simultaneous convergence of any subset of a larger set of environmental cues in the appropriate spatial relation to each other.

For the second group of place units it appears that the field is generated by active inhibitory processes acting on neurones with a tendency to fire spontaneously or with a diffuse source of excitatory drive. This inhibition restricts the area of an environment where the place unit fires. It derives from environmental stimuli either directly or indirectly through the mediation of other place units. When the appropriate number of these inhibitory inputs converge on a place unit, a discrete place field in an environment results. When there are too few environmental inputs, the unit fires over a large part of the environment or in several parts of the environment. On the other hand, too many such inputs (which is likely the rule rather than the exception) will totally inhibit the place unit and result in the absence of a field in that environment. The misplace units which increase their firing when the rat goes to a place and fails to find a particular object or cue (i.e. a light or a reward) there are probably all drawn from this second class of place units. We shall have more to say about how these place fields in the two groups of units might be generated in the next section, where we describe a model of hippocampal function.

Ranck (1973), in his unit study of the hippocampus of the freely moving rat, has described two classes of units in CA3 and CA1 which have very similar properties to our place units: approach-consummate units and approach-consummate-mismatch units. The approach-consummate units, which were found primarily in CA3 and to a lesser extent in CA1, fired whenever the rat ran to a reward and continued to fire as it consumed the reward. Ranck has emphasized the fact that these units may only fire during one consummatory behaviour such as drinking and the approach to water. To what extent this selectivity is due to the spatial location of the water or food is not yet clear. It is possible that in addition to place information these units are coding information about



(A) no wall cues, no food

(B) no wall cues, no food

(C) no light, no fan

(D) no light, no card

(E) no card, no buzzer

(F) no fan, no buzzer

FIG. 27. The same place unit as in Figs. 26(a) and 26(b). A and B: two no-wall-cues probe trials in which both the wall cues and the food are absent; C-F: four probe trials in which only two of the four wall cues are present. C: no light, no fan; D: no light, no card; E: no card, no buzzer; and F: no fan, no buzzer. Note that the place field is maintained in all these trials but there is a marked increase in total spikes in C. (O'Keefe and Conway, unpublished)

specific reward. Against the suggestion that the approach component of the place units is connected to the reward are the observations that (1) many place fields are not altered by removal of the reward or by switching rewards, (2) many fields are not located on the route to reward (see O'Keefe 1976), (3) in unpublished experiments we have found place units in an environment in which the animal had never been rewarded, and finally (4) the preliminary experiments in the curtained environment, as described above, show that the place field is dependent on environmental stimuli. The approach-consummate-mismatch units, found primarily in CA1, fired maximally when the rat went to the reward site and failed to find reward. Ranck noted the spatial aspects of some of these units, which appear to be identical to our misplace units.

Ranck listed two other major categories of units in the hippocampus: appetitive units and motion-punctuate units. Appetitive units fire during orienting or approach behaviour, but not during the subsequent consummatory behaviour. The exact character of these units is not clear, since they did not fire during all approach or orienting towards a particular reward and also fired when the rat approached other things. Many of these units were found in the fascia dentata. In our experiments the fascia dentata units remained silent for long periods of time and proved difficult to activate. Some units were found to require specific inputs in order to fire. Examples are a unit which fired only when the rat sniffed in a corner of its home box, irrespective of its spatial relation to the environment, and another which responded during sniffing of a round metal can but not other objects. If the units here are selective for quite specific sensory inputs it would explain why they are silent most of the time and why they could not be activated consistently in Ranck's study. Motion-punctuate units fire at the end of an approach or orienting movement and appear to be firing on the rebound release from inhibition. We shall assume that they are some form of place unit which is momentarily released from basket-cell inhibition and that they are of no special theoretical significance.

4.7.3. OTHER STUDIES OF UNIT ACTIVITY IN AWAKE UNRESTRAINED ANIMALS

Other studies of unit activity have been more neuropsychological than neuroethological. They all involved confining the animal in a closed space where there was little of interest and nothing for the animal to do except attend to the pre-ordained stimuli or learning task set up by the experimenter or *go to sleep.* As we shall see, this latter possibility may provide a serious source of artefact in most of these studies.

Vinogradova (Vinogradova 1970, Vinogradova *et al.* 1970) has recorded from rabbits confined in a small, semi-dark, soundproof box. She studied unit responses to flashes of light, general illumination, pure tones, clicks, and air puffs. Two types of units were found in the dorsal and ventral hippocampus: one which was activated by all sensory stimuli tested (A units) and another inhibited by all sensory stimuli (I units). No specificity for any stimulus was observed, and the unit response to any given stimulus habituated with repeated presentations. The response could be reinstated by switching to a new stimulus. Once the animal had been habituated to a stimulus new units did not respond to it. Most of these findings would seem to be parsimoniously explained as due to arousal and the habituation of arousal. Unfortunately, no observations of the animal's behaviour, or records of hippocampal or neocortical EEG, were taken. As Ranck (1973) has pointed out, place (complex-spike) units decrease their firing rate if the rat is aroused from slow-wave sleep, and this arousal lasts for some time after stimulus presentation. As mentioned in the discussion of LIA (p. 151), arousing stimuli will block the LIA spike in the CA1 field, and this effect habituates. Interestingly, Black and de Toledo (cited by Black 1975) have shown that the LIA spikes are inhibited by the conditioned stimulus in a situation where the US is aversive. In contrast, theta units in the rat will increase their firing if there is any orienting movement towards the stimulus. Since theta occurs in the rabbit to sensory stimulation, without movement, it is likely that Vinogradova's type A units are mainly theta units. Thus, she reports average response frequencies of 15-30 spikes/s in dorsal hippocampus and 10-20 spikes/s in ventral hippocampus; all of her figures look like theta units. In one of her records (Vinogradova *et al.* 1970, Fig. 11, p. 208) an A cell is shown to fire vigorously during movement, as would be expected of a theta unit. Some units fire, at times, with bursting phase frequencies of 5-6 Hz (her Fig. 4, p. 148).

Mays and Best (1975) have looked at the response of single units to similar stimuli in the rat hippocampus during waking and sleep. They found much the same pattern as did Vinogradova when the stimuli were presented during sleep, but no effect when presentation occurred during waking. They suggested that some of Vinogradova's findings might have been due to arousal from sleep; Ranck (1973) raised the same possibility.

A study by Lidsky *et al.* (1974a,b) on the paralysed but unanaesthetized rabbit also emphasizes the dangers of failing to monitor closely the state of arousal of the animal. They found that hippocampal units (probably mostly theta units) decreased their firing rates to sensory stimuli which aroused the animal from slow-wave sleep as evinced by a concomitant change in the neocortical EEG from synchronized to desynchronized. The same stimuli, however, caused an increase in firing when presented to an already alert animal, resulting in a further increase in arousal. This latter was signified by a continued desynchronized neocortical EEG together with an increase in muscle tone and heart rate.

Other studies (Noda, Manohar, and Adey 1969a,b, Olmstead, Best, and Mays 1973, Vincent, Dufy, and Faure 1968) which have looked at changes in unit activity during sleep and wakefulness have not classified the units according to any other criteria, making comparisons difficult. Vincent *et al.* (1968) found two different types of unit in the hippocampus of the free chronic rabbit. The first fired with theta during waking and low-voltage fast sleep, while the second rarely fired during waking, was silent during low-voltage fast sleep, and increased rates during slow-wave sleep. These findings would fit nicely with those of Ranck (1973) if we could identify the first type with his theta units and the second with his complex-spike units. Noda *et al.* (1969a,b) reported that unit rates in CA2 and CA3 of the cat were highest during low-voltage fast sleep, were progressively lower during movement, sitting quietly, and shallow slow-wave sleep, and were lowest during deep slow-wave sleep. During slow-wave sleep there was a greater tendency for different units to fire synchronously than during aroused wakefulness or low-voltage fast sleep. Perhaps the most interesting finding of the Noda *et al.* study was the absence of a constant phase relation between the units and the slow waves (theta activity) recorded from the same electrode. This finding stands in contrast to reports in the rat and rabbit, and suggests a more complex basis for theta in the cat than in the other two species.

Olds and his colleagues (Hirano, Best, and Olds 1970, Olds 1965, 1967, 1969, Mink, Best, and Olds 1967, Olds and Hirano 1969, Olds, Mink, and Best 1969, Olds *et al.* 1972, Segal, Disterhoft, and Olds 1972, Segal and Olds 1972, 1973, Segal 1973a,b, Hirsch 1973) have recorded from the dorsal hippocampus and other structures in rats in a series of experiments which exemplify the neuropsychological approach in a relatively pure form. All recordings were done during the same classical conditioning paradigm, and each paper seeks to answer a slightly different question about unit activity during that paradigm. Units were recorded from microwires while the rats were conditioned in a small cylindrical (12 in. diameter) enclosure. No attempt was made to hold onto the same unit during the two or three days of the experiment and often new units were substituted for old ones lost during the study. Some of the microwires were clearly recording from more than one unit. One of the criteria for selection of units to be studied was that the spontaneous rate should be between 1 /s and 20/s. This criterion means that almost all of the units recorded in the CA1 field of the hippocampus were displace units. In addition, there might have been a small contribution from place units while the animal was quiet or asleep, or place units with place fields within the small enclosure. In a related experiment (Ito and Olds 1971) the rates of units are given as 6-64/s. The likelihood that most of these were displace units is strengthened by inspection of the records of raw data published in these reports. These units showed an absence of complex spikes and fired with high-frequency bursts of uniform amplitude spikes reminiscent of the LIA spike-related bursts (see p. 200; see e.g. Olds *et al.* 1972, Fig. 2, p. 204).

Most of the experiments were conducted in three phases. The conditioning phase of the experiment was preceded by a pseudo-conditioning phase of several hundred trials during which the two stimuli and the food delivery were presented randomly. During this phase the unit response to the habituated CS provides a comparison for the response to the CS after learning. During the conditioning phase the CS+ was consistently followed by food delivery, while the CS– was not. In some experiments the conditioning phase was followed by extinction during which the stimuli were presented without food. Data were grouped together in terms of those units which increased or decreased their firing above pre-stimulation rates and, in later experiments, the latency of the unit's response to the stimuli are given. Gross movement of the animal was recorded electrically, but the actual behaviour of the animal was not observed. In some studies the rat was required to hold still before and during presentation of the stimuli in order to minimize the influence of movement and movement-related feedback. No attempt to study any other properties of the units, which might help in classifying them or understanding their behaviour, was reported.

We shall try to isolate a few conclusions from these extensive, but not very informative, studies. In general, the units from the hippocampus are not particularly affected by the stimuli prior to conditioning. Some experiments report no change, while others report a slight increase or decrease in baseline rate. For example, Olds and Hirano (1969) found only one of 19 hippocampal units which showed an increment of 40 per cent or greater to the tone during the pseudo-conditioning phase. After conditioning, the percentage of responsive units is only slightly improved. In the Hirano and Olds experiment 5 of 19 hippocampal units responded to the tone after conditioning. In some studies the group claim that there were large rate increases in the small number of responsive hippocampal units, usually involving a change from a small decrease in firing rate during pseudoconditioning to a large increase after conditioning. Latency studies generally showed that the responses to the conditioned stimuli occurred earliest in the CA3 field and only later in the fascia dentata and CA1 field. If these are, in fact, displace units, then this finding might indicate that there is a different theta input to CA3 than to the fascia dentata and CA1 In one study (Olds *et al.* 1969) it was found that some units responded better while the animal waited for food than while it waited for water, but these results were not consistent from day to day, suggesting that the electrode had moved.

Three other studies have reported effects of learning on hippocampal unit activity (Sideroff and Bindra 1976, Thompson 1976, Zippel, Kolle, and Gabriel 1973). Thompson and his colleagues (Thompson 1976) report that hippocampal multi-unit activity begins to show an increase in response to the conditioned stimulus during the early stages of a Pavlovian

conditioning paradigm (see above) and that no such change is seen to the unconditioned stimulus or to the conditioned stimulus in a pseudoconditioning paradigm. Sideroff and Bindra (1976) trained rats to lick at a water spout in the presence of one tone but not another. Multi-unit activity in the CA1 field of the hippocampus showed a greater and more prolonged increase to the positive stimulus than to the negative one. This differential increase was maintained even when the rats were subsequently satiated and no longer drank to the tone. Zippel *et al.* (1973) trained rats to a negative stimulus in a shuttle-box avoidance task (see p. 301) and to a positive stimulus in an approach runway task. The response of single hippocampal units were then recorded in acute experiments. The high average spontaneous rate of their units suggests that many of them were theta units. The rates of some units were increased, while others were decreased, to one or the other or both stimuli. There was no difference in the number of units which increased or decreased to the negative tone between the trained group of animals and an untrained group, nor was there a difference in the number of units which were influenced by both stimuli. The only difference found between the two groups was that in the untrained group nine units were excited and two inhibited by the positive stimulus, whereas in the trained group the respective figures were three and six.

In summary, then, all these studies agree that there are units in the hippocampus which change their firing patterns during various types of learning paradigms. The *significance* of these changes, however, is difficult if not impossible to assess. Some of the same studies found that unit responses in other brain areas were also modified during training (e.g. Olds *et al.* 1972, Sideroff and Bindra 1976). Olds and colleagues have tried to make some sense of these embarrassing riches by looking at the latency of the unit responses. As Thompson and colleagues (Thompson 1976) have pointed out, there is no reason to assign greater importance to earlier responses since these might appear only during the later stages of learning and might in fact be dependent on, or derivative from, longer latency responses which began to appear earlier in training. The emphasis of the Thompson team on responses arising early in training strikes us as being only slightly less precarious. The moment one begins to question the single-channel linear model of nervous system function, the force of arguments derived from temporal measurements is severely vitiated.

As we have previously stressed, part of the difficulty in the interpretation of these studies stems from their assumption that the brain can be treated like a porridge; all cells have been created equal and identical unless proved otherwise. No attempt is made to characterize units with respect to any variable except those of the learning situation. We need to have much more information about biophysical variables (firing rates, spike heights, spike widths, etc.), anatomical variables (location within the hippocampus relative to other units), the range of stimuli to which the unit responds (not just the conditioned and unconditioned stimulus), and the range of behaviours during which the unit fires. Armed with this knowledge, we can study unit changes during learning with some hope of understanding them. Does the unit firing correlate with eye movements prior to conditioning? Yes, then take your choice: (a) monitor eye movements during learning to see if there is any change in the correlation, or (b) train the animal not to move its eyes during learning (but check subsequently that no eye movements have been programmed even if not executed), or finally (c) forget that unit and search for one which would appear more likely to be involved in learning on the basis of its pre-training characteristics.

In the experiments under consideration we suspect that many of the hippocampal units recorded were theta units because of the high average spontaneous rates reported. This implies that in the rat the unit changes were probably secondary manifestations of primary changes in the animal's motor behaviour. In the rabbit the theta units probably change their firing patterns in the absence of movement as well as during movement. Until the correlates of these units are worked out in the untrained animal, interpretation of the learning-correlated changes will be impossible.

P. Best, a former student of Olds, has suggested that some of the changes in unit activity seen in the learning experiments of the Olds group might be related to arousal from sleep. Recall that, like Vinogradova, Olds does not record neocortical EEG or watch his animals. Only a crude movement indicator is used and this would not discriminate amongst different states of arousal. Best and Best (1975) showed that the effects of a tone differed in the course of a conditioning experiment, depending on whether it was presented during sleep or not. When presented during sleep, the tone woke up the rats and produced a significant decrease in unit rates during the preconditioning phase, during conditioning, and during extinction. No such changes were seen when the tone was presented to an awake animal. Interestingly enough, if no preconditioning habituation to the tone was given, the only significant effect of conditioning was an increased firing during conditioning in the awake state. No attempt to classify the units into theta and complex spikes was made. It is possible that there were more complex spike units in that group which had preconditioning habituation than in the other group. As noted above, complex-spike units will decrease their firing rates when the rat is awakened from slow-wave sleep.

There is, at the present time, good evidence from unit experiments on the hippocampus of the freely moving rat for two classes of units, one related to the slow-wave EEG and related, in the rat, to some aspect of movement, probably the displacement produced by that movement. Some of the other group, the complex-spike units (particularly in CA1), are

related to the location of the rat in an environment* and can be shown to be controlled by sensory stimuli in that environment *but not to depend on any specific stimulus.* Some of these place units are simple, responding when the rat goes to a place or runs through a place. Others have more specific requirements such as sniffing in a particular place. Whether there are other complex-spike units which do not have a place influence but are related primarily to consummatory activities such as eating or drinking is not clear at this time. We can now turn to an outline model of the neural basis of the hippocampal cognitive map on the basis of what is known of the anatomy and physiology of this structure.

4.8 Neural model for a spatial map

In the final section of this chapter an attempt is made to sketch the outline of a neural model of the hippocampus which is consonant with the known anatomy and physiology, and which would act as a spatial map. We must hasten to note that on many of the important points there is simply not enough information, while on others the results of different studies are in conflict. Nevertheless, we will try to make the model as explicit as possible, in accordance with the strategy adopted in the rest of the book. Although this ensures that it will be far off the mark, and therefore hopefully modified or rejected in the near future, the exercise will, first, demonstrate that it is possible to construct a spatial mapping system from what is known about the hippocampus, and secondly, by its very concreteness and testability, generate a better class of data than would be prompted by a vaguer model.

All model building proceeds in two stages. In the first, one must select and interpret the data to generate a set of 'facts', and in the second stage one must build a model out of these facts. It will be useful to begin with a summary of the facts about hippocampal anatomy and physiology (the 'pieces and patches' as we have called them elsewhere, Nadel and O'Keefe, 1974) which we will use to build our model.

(1) The hippocampus is made up of three separate systems, the fascia dentata, CA3, and CA1. Each system consists of a large matrix of logically similar cells, the dentate granules and the CA pyramids, together with a set of interneurones, the basket cells, which control the excitability of the soma and probably also the dendrites of the pyramidal and granule cells. The main flow between the three levels appears to be in the direction from the fascia dentata to CA3

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* In addition to the evidence from our own laboratory, recent experiments by Best and Ranck (1975), Branch, Olton, and Best (1976), and Hill (1976) have confirmed the existence of place units in the hippocampus of the freely moving rat.

and thence to CA1. These projections are topographically precise, such that a narrow strip of granule cells send their axons to a narrow strip of cells in CA3; these pyramids in turn innervate a narrow strip of CA1. In some of these projections there appears to be an interesting shift in the terminations along the septo-temporal axis, such that fibres at one end terminate closer to the soma than do those at the other end. These connected strips, or lamellae, form the major organizing principle within the hippocampus.

(2) One of the major inputs to the hippocampus, the entorhinal cortex, projects to the hippocampus in accordance with this precise pattern, small strips of entorhinal cells projecting only to a restricted lamella. It is likely that these afferents convey information about stimuli and perhaps their location in egocentric space (their local sign).

(3) The other major input to the hippocampus, the medial septum, has a much more diffuse termination, ending in all fields of the hippocampus and dentate area, probably on the basket cells, as well as on the granules and pyramids.

(4) In addition to the forward movement of activity from fascia dentata to CA1 there is considerable 'feedback' in the reverse direction as well as connections between neighbouring lamellae. The CA3 pyramids project back to the dentate granule cells and (in the posteroventral region) to the entorhinal cortex; they also project to neighbouring CA3 cells via the longitudinal association pathway. The CA1 pyramids send their axons to the subiculum, which in turn projects to the entorhinal cortex as well as through the fornix to lower brain areas.

(5) Both the pyramids and the granules have long, extended dendrites; the different inputs to the cell synapse at different levels of the dendrite. Most of these inputs stream across the dendrites of many cells, making synapses *en passage* as they go. Physiological studies suggest that a high degree of convergence of input to the dendrites is necessary to fire the cell. In addition, the basket cells and other interneurones (which probably generate theta) have an inhibitory action on the soma and dendrites of granules and pyramids. These factors explain why the granules and pyramids have low 'spontaneous' firing rates, especially during theta-related behaviours outside of the place field.

(6) The theta activity appears to be relatively synchronous over large areas of the hippocampus. One of its functions, therefore, must be to lock together in simultaneous oscillation large volumes of spatially disparate tissue. We shall accept, on the basis of Ranck's findings, that there are theta units in CA3 and that a theta mechanism exists there in spite of the failure of Bland *et al.* (1975) to find theta in the CA3 field.* The theta mechanism provides a time gate during which the granule or pyramidal cells may be activated. We shall assume that the theta/displace cells are the basket cells, although it must be kept in mind that they may be some other neural element such as the specialized terminals of the medial septal afferents. Since different theta/displace and place cells in CA1 fire with different phases of the theta recorded from the same electrode, a further assumption will be that different theta cells control different parts of the dendrites of the pyramidal cells. This assumption would explain the finding of Winson that there is a gradual phase shift of the theta as one moves out along the apical dendrites of the CA1 cells. The absence of such a phase shift in rabbit suggests that the basket cells end at the same level of the dendrites in this species.

(7) Theta activity recorded from the hippocampus of the freely moving animal has two correlates: movements in space and some aspect of sensory processing such as attention. The latter is less well defined than the former. Species such as the rat, rabbit, and cat differ in the extent to which they exhibit the two kinds of theta. In the rat the sensory related theta is rare, most theta being related to a class of movements, in particular to the velocity of, or displacement caused by, the movement. In rabbits and cats the sensory related theta is more common, but the movement-related theta exists as well. The distribution of theta within the hippocampus differs in the rat and rabbit. One possibility is that non-movement theta is elicited by the output from the hippocampus itself, an extension of the McLennan-Miller hypothesis, or perhaps it is a sign of the activation of the movement generator which is being blocked from gaining access to the effector system. All the situations in the rat during which this non-movement theta has been recorded are conflict situations.

(8) Studies of single units in the hippocampus of the freely moving rat can be divided into two categories: on the one hand, those which allowed or encouraged the animal to move around in space and provided it with a large amount of spatial information, and, on the other hand, those which restricted the rat's movements and limited the stimulating environment. These latter studies also tended to focus on a small range of stimuli, such as flashing lights, tones, and so on, or to study changes during a classical conditioning

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* As we noted, the theta activity recorded must be dependent on the spatial locations of the inhibitory interneurones and their endings on the pyramidal cells. The location of theta units in the apical dendritic layers of the CA3 field might result in no overall rhythmical slow wave, in spite of rhythmical intracellular activity in individual neurones. Similarly, if theta units which are out of phase with each other synapse on the same part of the dendritic tree of different CA3 pyramids, the extracellular currents would cancel each other and no extracellular theta would be recorded.

paradigm. Studies done in our laboratory suggest that spatial factors play a large part in the responsiveness of CA1 neurones, some fired when the rat went to, or ran past, a place in the environment, and others fired even better when the rat sniffed in a place because of the absence of a usual item or the appearance of a new item. We have called these place and misplace units respectively.

(9) Unit activity recorded in the controlled curtain environment indicates that one type of place unit is built up from the convergence of excitatory inputs, all tending to fire the unit in a particular place. The other type has a place field constructed from primarily inhibitory inputs which prevent the unit from firing in those parts of the environment outside the place field. The misplace units are probably a subset of this second type.

(10) The firing patterns of other units are closely tied up with the slow waves recorded from the hippocampus, including the theta wave.

(11) According to Ranck there are a large number of approach consummate units in CA3. It is not possible to say whether these are the same as our place units which fire maximally when the rat runs past a place. We shall assume that they are the same, and postulate that the map begins in CA3.

(12) It is very difficult to find the adequate situation to fire units in the fascia dentata. This probably indicates that they are responsive to a highly specific stimulus or combination of stimuli.

4.8.1. THE MODEL

Our working assumption is that the mapping system contains three stages, one for each section of the hippocampus. The first stage, to be equated with the fascia dentata and about which we know the least, seems to be involved in organizing the environmental inputs transmitted through the taxon systems into a schema required by the mapping system. In addition, it is quite likely that some combining of simple stimuli into complex stimulus configurations occurs here, as well as a representation of the angle with which they conjointly impinge upon the animal. There is the remote possibility that the angle between two stimuli, irrespective of their local signs, is actually calculated here. The second stage, located in CA3, makes up the initial part of the map and represents places in an environment and the connections between those places. The final stage, in CA1, continues the map and contains the misplace system, which signals the presence of something new in a place or the absence of something old.

4.8.1(a). Fascia dentata. Units recorded from the fascia dentata are either theta units or poorly defined approach units, in Ranck's terms. In our limited experience these latter relate to specific sensory inputs and are not

place coded. Ranck found some units which fired during approach behaviour, but they were poorly characterized. On the basis of the anatomy of their primary inputs from the entorhinal cortex and, in particular, the convergence of inputs from different areas of this cortex onto the dentate granule cells, we shall guess that they-are responsive to complex configurations of stimuli. More specifically, it will be assumed that each granule cell is sensitive not just to the simultaneous presence of two or more stimuli, but to their occurrence in a unique spatial configuration. For example, a particular granule cell would fire if a light was present at 3 o'clock in taxon space at the same time that a buzzing noise impinged on the animal from 7 o'clock.* Another granule cell would fire to a light at 3 and a buzzer at 8, a third to a wind at 3 and a buzz at 8, a fourth to a wind at 3 and food at 12. ** ***

The orderliness of the projections from the entorhinal cortex to the fascia dentata and thence to the rest of the hippocampus suggests that there must be some systematic relationship characterizing the sensory information in neighbouring granule cells within the same lamella and within adjacent lamella. There is no information available at present which can help us here. We shall assume therefore that all of the granule cells within a lamella have in common one sensory input at one location in taxon space (e.g. a light at 3 o'clock) and that different cells in the same fascia dentata lamella are distinguished from each other by their other inputs. Similarly, neighbouring lamellae are dominated by different sensory inputs, and there is an orderliness to this pattern. Lamella A might be dominated by a light at 3, the next lamella B by a light at 4, and so on.

There are several anatomical schemes which might support this type of correlation process. Fig. 28 represents one which does not do gross injustice to what is known of the entorhinal-dentate projection. It suggests that each entorhinal cell, in addition to a projection which stays within the lamella and traverses all of the apical dendrites of the granule cells in that lamella (Fig. 28(B)), sends a collateral to neighbouring lamellae which traverses them in an oblique fashion making contact with the dendrites of only a few granule cells in each lamella (Fig. 28(A)). On this model the

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* Here and subsequently we are using a convention for identifying the location of stimuli in the taxon space of the rat in which the nose points to 12 o'clock, the tail to 6 o'clock, the right eye to 3 o'clock, and the left eye to 9 o'clock.

** We have also considered a model of the fascia dentata in which individual granule cells or groups of granule cells responded not only to combinations of stimuli occurring in particular locations in taxon space but to stimuli occurring with a fixed angle anywhere in taxon space. For example, a cell would respond not only to light at 3 and sound at 7 but any time that a sound occurred 120° clockwise to a light (light at 4, sound at 8; light at 8, sound at 12, etc.). Our feeling is that the data from unit and behavioural studies on the rat do not warrant the attribution of this much power to the fascia dentata at present.

*** We assume that the map receives inputs from proximal stimuli and reinforcers as well as distal stimuli but that it treats them in the same way. For methodological reasons in unit and behavioural experiments distal stimuli are emphasized, since it is more difficult to demonstrate that proximal cues are being used to define places and not as guidances.

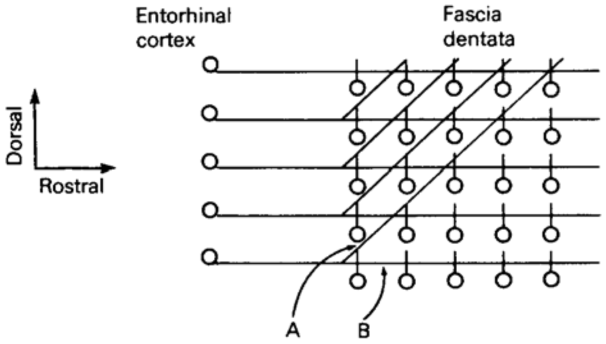

FIG. 28. Schema of the hypothesized projection from the entorhinal cortex to the fascia dentata. The axon of each neurone in the entorhinal cortex would have two branches: one (B) projects to all the granule cells in a lamella at the same horizontal level and the other (A) follows an oblique trajectory making contact with a different part of each neighbouring lamella.

fascia dentata is viewed as a simple cross-correlator representing all the conjunctions of stimuli, and their relations in taxon space, that an animal has experienced in an environment. Depending on the specificity of the stimuli to which it is sensitive, a given granule cell might respond to a large part of an environment or only to a small place. The role of theta activity in the fascia dentata, on this model, would simply be to impose a pulsation on the output from the fascia dentata which is superimposed over the entire extent of this structure. This is in line with the general finding that all the theta units in this area fire on the positive wave of the theta recorded from the same electrode. The frequency of the pulsing output would be a function of the speed of the animal's movement through the environment, at least during the initial phase of the movement. The output from the fascia dentata is sent exclusively to the CA4 and CA3 fields via the slowly conducting mossy fibres.[*] We shall assume that each pulsed output stays relatively synchronized as it passes the dendrites of the CA3 pyramidal cells.

4.8.1(b). The CA3 field. We are now faced with two possibilities as to the construction of the map. Either the map is constructed in the CA3 field and the CA1 field is primarily devoted to the misplace system, or the map itself is not constructed until CA1. On this latter view both the map and the misplace system are located in CA1; the CA3 field would then represent an intermediate stage between the compound-stimulus stage of the fascia dentata and the map of CA1. Part of the problem in deciding between these alternatives is our lack of information concerning the properties of the CA3 approach-consummate units. Our examination of similar units in the CA1 field shows that they are true place units, continuing to respond when any single stimulus is removed from our controlled environment (see pp. 205-9). Ranck (1973) has reported that, while a minority of units in CA1 are approach-consummate units, they represent the majority of units in CA3. What is not known yet is whether the CA3 units are also place units. It reminds us of a doctoral candidate in psychology we once knew. Having heard that his experiments had produced mixed results, some confirming his hypothesis and others failing to do so, we went to console him, only to find him close to ecstasy:

> 'If the results had come out as predicted, there would have been nothing left to say in the discussion. This way I can write a really good discussion' (privileged communication).

Regardless of where it is located, the job of the mapping machinery is twofold: (1) it must combine into a single place representation the various compound taxon spatial representations in the fascia dentata which correspond to those configurations the animal has experienced in one part of an environment and simultaneously to exclude those experienced in other parts; (2) it must connect these place representations together into an organized pattern which contains information as to the distance and direction between them. An important part of this latter process will be the allocation of cells to represent places in an environment where no stimuli have been experienced.

Part of the mechanism for constructing a place representation out of independent fascia dentata stimulus representations will be based on temporal contiguity; stimuli which impinge on an animal at the same time must be experienced in the same place. But this is clearly not sufficient of itself, since one of the properties of the map is that stimuli experienced in the same part of an environment at different times will also be connected to the appropriate place representation. This calls for a mechanism which will identify the appropriate representation of a part of the environment independently from the stimuli impinging on the animal at that time.[*] If each map were laid out in the hippocampus such that it was spatially isomorphic to the environment that it represented, then place representations which were not identified by environmental stimuli could nevertheless be activated by neighbouring place representations which had been activated by such stimuli. At present there is no hard evidence as to the morphology of the map of an environment within the hippocampus. CA1 place units recorded next to each other sometimes represent neighbouring parts of an environment, as often they do not.

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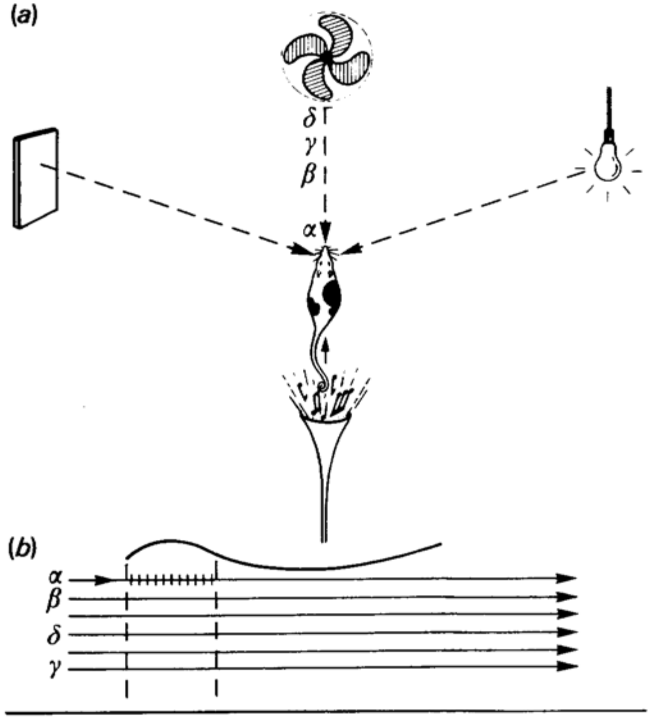
[*] No special function is assigned to CA4 in the model.

[*] This was, of course, Kant's fatal criticism of Berkeley's theory: that it could not identify which sequences of stimuli were attributable to the same sources being re-experienced and which were attributable to different sources. Only an absolute spatial system such as the one we are postulating can do this.

We shall postulate that the theta system is part of a mechanism for shifting the focus of excitation from one set of place representations to another within the map, not on the basis of sensory experience but on the basis of the animal's actual or intended movements. This is based on the clear evidence that atropine-resistant theta in the rat, and probably, other animals as well, is related to the animal's movements, and in particular movements which change its position relative to the environment. Furthermore, in unpublished experiments from our laboratory we find a monotonic relationship between the frequency of theta and the distance covered during a jump. Thus, at least for ballistic movements and probably for the initial phase of most continuous movements, theta provides one of the two pieces of information necessary for 'internal navigation'. As we have seen (pp. 157-60), the medial septum is probably a mechanism for translating programmed movements into displacement co-ordinates. An internal navigation system also needs information about the direction of movement. As we saw in our discussion of bird and human navigation, this could be provided by a compass mechanism which measured angles relative to some external landmark such as true North. Alternatively, it could be based on a calculation of the angles through which the animal actually turns. We have not seen clear evidence for either source of information in our unit studies yet, but will assume that at least the second, and perhaps also the first, source of directional information exists in the hippocampus.



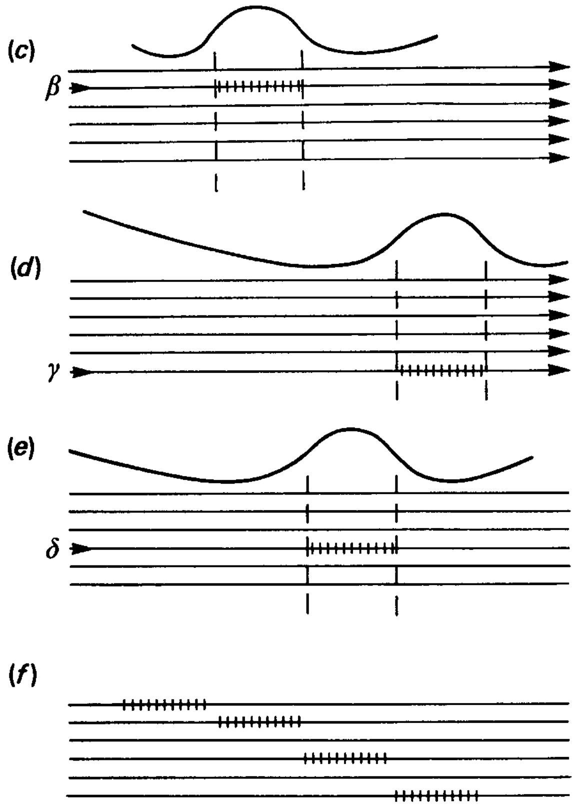


FIG. 29. Model of CA3 mapping system. Theta acts to locate inputs in CA3 on the basis of the speed with which the rat moves through an environment. (a) Environment with four stimuli: a fan, a light, a source of sound, and a large white card. (b)-(e) inputs at different times in a run from α to δ are deposited in different parts of the CA3 field. (f) Representation of the environment in CA3 after the rat has run from α to δ.

Let us consider how this theta system might serve to form a map in CA3. As we have already noted, the fascia dentata granule cells are gated by the theta mechanism and only allowed to fire at one phase of the theta cycle. Thus, in the fascia dentata one of the roles of theta must be to impose a synchronous bursting pattern on the granule cell output. This burst would act as a timing pulse, and the part of the CA3 lamella which fired in response to a particular burst would depend on its state of excitability relative to this pulse. How might this work? One possibility is that there are systematic phase and amplitude shifts along the CA3 pyramids from cycle to cycle of theta so that the peak of excitability would shift across the CA3 field. Recall that Adey *et al.* (1960) reported changes in the phase relations between the theta in hippocampus and entorhinal cortex as a result of training. The wavelength of the theta cycle relative to the length of CA3 is too large for this work if anything more than 10 per cent or so of the theta cycle represents the excitable phase of the pyramidal cells. Alternatively, the speed of conduction of the extremely thin, unmyelinated, mossy fibres might be much slower than has been measured. Synchronous electric activation of these fibres might result in ephaptic synergistic activation of neighbouring fibres yielding an overestimate of the velocity of individual fibres.*

The operation of such a system is schematized in Fig. 29. Let us assume for the sake of simplicity that the rat finds itself in a familiar environment consisting of four stimuli, a light, a white card, a fan, and a sound, and that the animal is located at α. Place α would be represented in the CA3 field by cells activated by dentate granule cells responsive to light at 2 and broad-band sound at 6; light at 2, card at 10; card at 10, wind at 12, etc. The animal generates low-frequency theta scanning across the CA3 field until the representation of place α (in the example towards the dentate end of CA3) is activated. In the rat this 'retrieval' theta is generally accompanied by small movements, but need not be so (as in jumping); in the rabbit and cat it often is not accompanied by movement. The output from the CA3 representation of α forms the go signal for the animal to move towards β and serves to synchronize the theta within the map. At each successive theta cycle the maximum amplitude of the theta excitation shifts a small distance within the CA3 field. Thus, sensory inputs entering any given lamella will be systematically deposited in different parts of the CA3 field on each successive theta cycle. Of course, as the animal moves along the path between α and δ the sensory inputs will change and thus the lamellae which are active will also change (large arrow in the figure). By the time the rat reaches γ the inputs experienced there might be deposited towards the CA1 end of the CA3 field (Fig. 29(d)). The representation of the path from α to δ might look something like the set of CA3 place cells shown in Fig. 29(f). The relationship between these representations is a function of the distance separating them in the environment and not of the particular behaviour which was used to get from α to δ. Recall that movements across greater distances are accompanied by theta of higher frequency, and that according to Petsche and Stumpf (see pp. 145-6) the phase difference between two points on the surface of the hippocampus remains the same regardless of the frequency. If both apply to the CA3 theta system, then a fast run from α to δ would activate the same place representations in the same order as would a slow run. Similarly, if no sensory inputs were represented at γ during one run from α to δ, a set of CA3 cells would still be set aside to represent that (empty) place. Stimuli noticed at γ on subsequent runs from α to δ would be allocated to these place representations and would stand in their proper spatial relationships to previously registered stimuli. We have not tried to incorporate information about directions into the model, but do not see any unsurmountable obstacles to doing so.

The CA3 neurones have three targets: (1) an extra-hippocampal one in the lateral septum; (2) an intra-hippocampal feedback to the fascia dentata granule cells; (3) an input to the CA1 field via the Schaffer collaterals. If the map is located in CA3, then information about places in the environment, which could be used to generate place hypotheses, would be transmitted via the fornix and fimbria to the lateral septum and thence to the brain-stem motor-programming circuits. We shall discuss shortly how such information could be used to control the animal's behaviour. Similar information would also be sent to the CA1 field where it would be used to build up the misplace system. We have no strongly motivated function to assign to the feedback from CA3 to the fascia dentata. We could suppose that it is involved in the circuitry which synchronizes the theta phase to the beginning of a movement in a particular place.

4.8.1(c). Output of the place system. There are many ways in which this place system could be used to control motor outputs. We shall discuss the wide variety of place hypotheses at many different points in the following part of this book. Here it will suffice to mention one. Let us imagine our animal has explored an environment containing food, while sated, and has built a map of that environment including the location of food. At a later time the animal, now hungry, finds itself again in the same environment. It knows it is in a familiar environment because it has a map for that environment. How might the map guide the animal's behaviour towards the food? Let us assume that, in addition to a generalized subliminal excitation of all place representations connected together into one map which ensues whenever any two or more parts of a map are activated, hunger specifically excites those place representations where food has been experienced. These two will sum, bringing the representation of the place containing food in that environment close to activation. Since the place that the animal occupies will also be receiving afferent drive there will be two sets of place representations which are potentiated. The programming system can now search for the appropriate motor programme which activates both of these representations simultaneously; this programme will take the animal from where it is to where the food is. In this sense, then, the theta can be thought of as a retrieval system which, given one place representation, searches for the proper programme to retrieve another. Here we agree with Vanderwolf that at least one function of immobility theta reflects the activation of motor programmes without movement.

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* Of course, this would also imply a larger spread of velocities and a consequent spread of the volley produced during natural stimulation.

4.8.1(d). The CA1 field. Again, we must consider two possibilities. If the map is constructed in CA3, then the primary function of CA1 is to generate mismatch signals when the representations of places do not match the stimuli experienced in the corresponding part of the environment. If, on the other hand, the CA3 field does not contain the map then this function must also be attributed to CA1. If the map is constructed in CA1 it might occur in much the same way just described for CA3. There is an alternative mechanism, however, which could achieve the same end and for which there is somewhat more evidence. It consists of an amalgamation of the imbricated pattern of termination of afferent fibres (as demonstrated by Zimmer and Hjorth-Simonsen for several pathways in the hippocampus, see pp. 120-2) with the shifts in theta phase as an electrode is moved down through the CA1 apical dendrites. Recall that we interpreted the imbricated termination pattern as evidence that a fibre projected to the proximal part of its termination zone on the dendrites of some cells, to the middle part of the zone on others, and to the distal part on still others. Any mechanism which selectively blocked off the same portion of the dendrites of all these cells at the same time would act as a sorter, and might fulfil the same function as the theta mechanism proposed for the CA3 field. Fig. 30 suggests how the continuously phase-shifting theta in CA1 would operate. A constellation of inputs arriving on the rightmost line would make contact on different parts of the dendrites of different lamellae. In the model illustrated the input moves more distally on each successive dendrite as it moves leftward. Which of these target dendrites will be activated by the input depends on their relative states of excitability as determined by the theta mechanism. At any given time some patch of each dendrite will be relatively receptive while the rest will be unreceptive. In Fig. 30 the 'theta excitability window' is portrayed as conferring receptivity to the distal patch of the dendrites at T_1 and moving proximally towards the cell body as the theta cycle progresses. Consequently, an input on the leftmost line will activate the top cell of column A if it arrives at T_1, the next to the top cell in column B if it arrives at T_2, etc. The representation of the inputs experienced in an environment such as seen in Fig. 29(a) will be formally similar to that illustrated in Fig. 29(e).

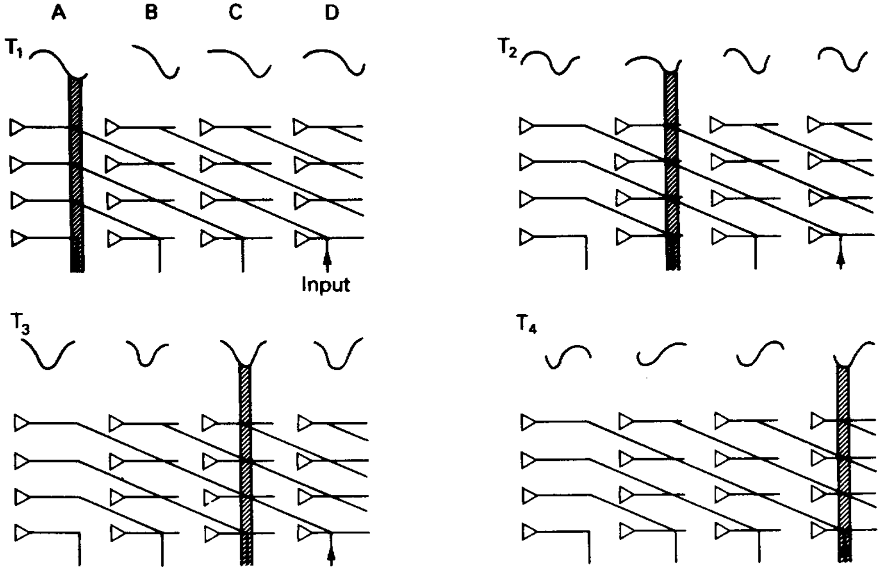

FIG. 30. A model of the CA1 field. As the theta cycle progresses the patch of excitability in the dendrites of the CA1 pyramids changes from the most distal part (T_1) through middle parts (T_2 and T_3) to the most proximal sections (T_4). The same input entering the CA1 field (far right) will excite a different cell depending on the timing of its arrival relative to the theta cycle. At T_1 it excites the topmost cell in A, T_2 the second cell from the top in B, etc.

In addition to place units which receive afferent drive from the stimuli in an environment, there are place, or rather *misplace*, units which appear to have a high 'spontaneous' rate or to receive a diffuse source of afferent drive and are inhibited by stimuli or place representations. The evidence for this (see pp. 202-9) is that these units begin to fire at higher rates and to larger portions of an environment when the environment is drastically altered or when significant numbers of stimuli are removed from the environment. High rates in these units would function to signal the absence of an entire map, or particular place representations, or mismatches between the representation of a place and the stimuli being experienced in that place. This misplace output acts as a signal to the motor circuits in the brain-stem which activates exploration patterns. Large outputs from this system would also activate 'fear' circuits in the brainstem. Which of these two circuits dominates would depend on the genetic constitution and the past experience of the animal. We shall discuss this in greater detail in the chapter on exploration (p. 243).

Exploration has several purposes. First, to check that the 'missing' stimulus really is absent or changed and that the rest of the environment has remained the same. Probably the first question the system asks is: am I in the right place? Next, the change is incorporated into the system during a rapid one-trial learning. Here, the change probably takes the form of strengthening the other inputs to the place units and perhaps also blocking the now-absent input. One thing is certain: the old input is not literally erased, only rendered ineffective. Thus it is possible (although perhaps not in the rat) to retrieve the information that a particular stimulus was present in the past but is now absent. In this way not only can a place unit be used to represent different places in different environments, but also the same environment at different times. As we shall see in the chapter on human memory, this capacity underlies long-term, context-dependent memory.

The final question concerns the physiology of the changes which take place in the mapping system during learning. On the basis of the long-term potentiation experiments (see p. 125), which show lasting increases in synaptic efficiency following a small tetanus of synchronous inputs, we shall postulate that any inputs which occur on the same dendrites simultaneously will be enhanced. Whether or not it is necessary for the cell actually to fire is unclear at present. The learning changes would serve to enable any of several stimuli occurring in a place to activate the map's representation of that place and to tie together place representation of the same environment. Finally, as we just pointed out, it is unlikely that representations are actually erased; rather, new representations are created in addition to existing old ones, so that an animal could know that it had experienced a configuration in the past, but that this was not the most recent representation.

5

Introduction to the lesion review

5.1. Nature and purpose of the review

IN this section of the book we review lesion and stimulation studies of the hippocampus. Throughout, animals with damage in the hippocampus or fornix are referred to as *hippocampal* or *fornical* animals, respectively. We have tried to include in this review every published study of the effects of hippocampal disruption on behaviour and apologize in advance for any omissions.* Given the large number of lesion studies on the hippocampus it is possible to group most of them according to the type of behaviour investigated. For each such group a table is provided in the Appendix which lists the relevant studies and their results. In the text a selection of studies from each group is discussed, including particularly those which document an important aspect of the hippocampal syndrome. We hope that the tables will provide an easy reference source for the reader and further that they redress any imbalance in the text resulting from our selection of studies for discussion. Some consideration of studies involving either electrical or chemical stimulation of the hippocampus is included in a separate chapter where the specific methodological problems raised by the use of these techniques can be considered.

Only those studies whose intent was to limit lesions to the hippocampus (or fornix) are included. No control, however, could be taken over the wide disparity in size and, perhaps more important, locus of lesion within the hippocampus. We assume, in this discussion, that the hippocampus throughout its length is a unitary structure *in the sense that it has an integrated function.* This is not to say that we are assuming complete equipotentiality of function within the hippocampus. One of us has suggested that there are differences in function between the dorsal and ventral parts of the hippocampus in rats (Nadel 1968); this possibility was noted at about the same time by several other investigators (e.g. Jackson 1968, Jarrard 1967). These regional differences could reflect the uneven distribution of CA fields within the hippocampus (see Lorente de No 1934, p. 155) and the different inputs and outputs of these fields. At various points in this review we shall confront the fact that the site of a hippocampal lesion can determine its effect upon behaviour. We have already seen that an

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* Our review of the literature terminated in January 1976.

anatomical basis for differentiation exists within the hippocampus. What can presently be said about the specific functions of segments of the hippocampus?

The first thing that must be said is that much of our discussion is going to be simplified; we shall treat dorsal and ventral, anterior and posterior, lesions as the same, though it is clear that these are not equivalent in all respects. More pertinent, it now seems certain that fornix lesions cannot be treated as equivalent to hippocampal lesions, while neither is the same as entorhinal damage. However, there is enough commonality in all these lesions to allow us the assumption of a unitary system. While this totality undoubtedly comprises several separable subfunctions, we concentrate in this book on the mapping ensemble. The collection of interrelated functions we assign to the mapping system can, and should, be allocated to portions of that system. We choose not to attempt this fine-grain analysis here for two reasons. First, such an analysis would not affect the basic position adopted here; our chosen level of analysis does not require it. Second, there are not sufficient data at present to produce a consistent picture of the effects of lesions limited to one portion of the system or another.

Within each chapter in this section we discuss a particular aspect or type of behaviour and its modification, if any, following hippocampal disruption. Chapter 6 introduces the problem of reactions to novelty, and assesses the proposal that the hippocampus is critical for exploratory behaviour. We then examine the role of the hippocampus in discrimination and maze learning, concentrating upon the way in which animals utilize place hypotheses in these tasks (Chapter 7). Following this we examine the role of the hippocampus in behaviours based on reactions to threat, including fear, aggression, and avoidance (Chapter 8). In Chapter 9 we turn to a number of testing situations in which the role of the cognitive mapping system should be minimal, and this is followed by a consideration of reactions to reward shifts and extinction (Chapter 10). Next, we consider the general effects of hippocampal lesions upon such things as eating, drinking, and sexual behaviour (Chapter 11). This is followed by a brief discussion of stimulation studies (Chapter 12). The section is concluded with a chapter in which the role of the locale system in long-term memory is briefly assessed.

Prior to analysing the effects of hippocampal lesions upon any behaviour we attempt to uncover the factors underlying that behaviour in normal animals. In particular, we shall try to spell out the role of locale and taxon systems and to make predictions concerning the effects of hippocampal lesions. The verification of these 'predictions' does not, of course, constitute proof of the theory, at least in so far as they concern experiments already in the literature. We hope that sufficient, as yet unexamined, predictions will be suggested in the course of this review to provide adequate tests of the theory. Our present aim is to incorporate all that is known about the effects of hippocampal lesions within a minimal set of basic postulates resting upon the cognitive approach to brain and behaviour discussed earlier. Further, these postulates must be consistent with what is known of the anatomical and physiological properties of the hippocampus as described in the previous section of the book.

In several cases data are discussed which cannot be explained without making unwarranted *post hoc* extensions to the theory; in such cases we present the data and discuss possible solutions to the problems they raise without settling on a final answer. In other cases the discussion raises interesting possibilities that go well beyond the scope of this book or the available data; here we have been content simply to point out these possibilities and leave it at that.

It is a basic contention of the present theory that the hippocampus is not involved in fundamental motivational processes; that is, it is not a necessary link in eating, drinking, sexual behaviour, and so on. Evidence supporting this position is presented towards the end of this section. However, there are circumstances in which changes in aspects of these fundamental behaviours appear to follow upon hippocampal disruption and these are discussed at various points in the review. In these cases it can be seen that the absence of the cognitive-mapping system influences even the most simple behaviours in a specifiable way. Throughout the discussion of more complex learning situations, then, it is assumed that the absence of the hippocampus has a number of effects, but that the common thread linking these together is the lack of a cognitive-mapping system; it is this central defect that we shall concentrate upon.

5.2. Methodological considerations

Before turning to the lesion data we should comment on the validity of the lesion-cum-behavioural testing paradigm as a tool in the analysis of brain function. Strong biological and logical objections could be (and have been) raised against a heavy reliance upon this technique. From purely biological considerations one could argue either that *something more* than the removal of a discrete area of neural tissue has been effected by the lesion, or conversely, that *not even that* had been accomplished.

5.2.1. THE LESION AS SOMETHING MORE THAN FOCAL DESTRUCTION

5.2.1(a) Epileptic activity. Electrolytic lesions may produce an irritative focus which bombards distant structures with abnormal patterns of activity. It is now well established that such excitation produces long-lasting changes in these distant structures which may cause them to function abnormally (e.g. Morrell 1961, Goddard, McIntyre, and Leech 1969). More recent work has shown that abnormal epileptiform patterns can result from the de-afferentation that any lesion produces (Anderson *et al.*1971).

The hippocampus is a particularly epileptogenic structure and can be expected to be a likely generator of such abnormal barrages after electrolytic lesions. On the other hand, direct elicitation of epileptiform activity in the hippocampus through implantation of penicillin, or other irritating drugs, leads to a syndrome of behavioural defects demonstrably different from that produced by electrolytic or suction lesions of the hippocampus (e.g. Schmaltz 1971, Schmaltz, Wolf, and Trejo 1973).

5.2.1(b). Denervation hypersensitivity. The loss of afferent activity (rather than any trophic factor) which results from a lesion produces marked changes in nerve (Stavraky 1961, Sharpless 1964). In addition to the epileptiform bursting activity noted above, changes reported include collateral sprouting from neighbouring intact axons (Raisman 1969) and the potentiation of normally silent afferents (Wall and Egger 1971, Merrill and Wall 1972). In all of these examples the resultant reorganization of the target tissue would tend to be maladaptive and thus would exacerbate the damage produced by the primary lesion (see, however, the *not even that* argument below).

5.2.1(c). Hormonal and transmitter imbalance. Lesions in areas involved in hormonal regulation might shift the pattern of hormone secretion with consequent functional changes in the target areas for these hormones. As we note later (pp. 357-62), the hippocampus is involved in certain aspects of the pituitary-adrenocortical system, and lesions of the hippocampus might cause long-lasting changes in resting levels of the hormones produced by this system as well as in acute reactions to particular trigger events. One reported effect of this change in hormone function, for example, is a striking increase in liver glycogen levels in rats with hippocampal lesions (Murphy, Wideman, and Brown 1972). Other, perhaps more important, changes could involve alterations in neural excitability and neurotransmitter levels (cf. de Wied and Wiejnen 1970), which have been associated with changes in pituitary-adrenal function.

Further, lesions in one area might directly alter neurotransmitter levels in other areas to which it normally projects; Donoso (1966) has demonstrated such an effect of hippocampal lesions on noradrenaline levels in the hypothalamus.

5.2.1(d). Effects on sleep. Brain lesions frequently lead to changes in sleep patterns. In some instances paradoxical, or rapid-eye-movement (REM), sleep increases following a lesion; in the case of the hippocampus lesions decrease total sleep time (Jarrard 1968), but there are no data demonstrating marked changes in REM sleep. REM deprivation has global effects on brain function; it leads to increased amplitude of evoked potentials in entorhinal cortex following pre-pyriform stimulation (Satinoff, Drucker- Colin, and Hernandez-Peon 1971), to decreased recovery cycles (Dewson *et al* 1967), and to decreased seizure thresholds (Cohen and Dement 1965). All these point to general changes in excitability of many different brain areas as an indirect result of a brain lesion which affects REM-sleep time.

Some idea of the extent and magnitude of these global changes following central lesions can be obtained from physiological studies on the spinal cord or sensory relay nuclei following chronic lesions. Septal lesions, for example, lead to increased evoked potentials in the trigeminal nucleus upon stimulation of the trigeminal nerve (Rose and Frommer 1971). Perhaps more interesting is the finding of Griffin (1970) that the decreased habituation in spinal reflexes following frontal lesions in the rat persists after spinal-cord transection. This indicates a long-term change consequent upon the lesion in the frontal area.

5.2.2. THE NOT EVEN THAT ARGUMENT

In contrast to the above arguments, adherents of the *not even that* position suggest that structural and functional reorganizations following a lesion might act to ameliorate the behavioural defect. Evidence for this position comes from (1) the recovery of the lost behavioural capacity with experience, (2) the attenuation of the effect when the lesion is produced in several stages separated in time, as opposed to the usual single-stage lesion (e.g. Stein *et al.* 1969), and (3) the reduced or absent effect when the same lesion is produced in neonatal animals. All these examples have implications for what is known as the *recovery of function* phenomenon (see Rosner 1970, 1974, Dawson 1973, LeVere 1975), and several explanations for this `recovery' can be noted. Basically, these explanations fall into two broad categories: those suggesting that new brain structures take over the functions of the lost tissue and that no behavioural capacities are lost, and those suggesting that there is little neural take-over of function but that alternative behavioural capabilities are utilized to solve certain tasks.

The take-over notion would appear unlikely on purely anatomical grounds. Although one bit of cortex might conceivably substitute for another, the unique machinery of a structure such as the hippocampus is not so easily replaced or simulated. To demonstrate that true take-over of function occurs would require that lesioned animals recover all the capabilities of intact animals, including the ability to solve the same problem in precisely the same way. We have already stressed, and will continue to emphasize, the point that most tasks put to experimental animals can be solved through the use of any of several different strategies, some of which will be dependent upon different brain structures. Recovery of function could thus represent a switch to alternative modes of solution dependent upon intact brain tissue, rather than the actual reorganization of brain function. Neonatal and/or serial lesions, on this view, would permit a gradual adjustment to the use of these alternative hypotheses which typically would not have been used in the intact animal. According to this analysis recovery of function should not appear with behavioural tasks dependent solely upon the types of hypotheses subserved by the damaged structure; we present evidence later supporting this contention for animals with hippocampal lesions (pp. 377-9). Further, recovery of function, when it occurs, should be dependent upon a change to a new mode of task solution. We discuss shortly a way in which this possibility could be tested.

Some recovery of function might be due to the amelioration of certain of the *something more* effects noted above, either with the passage of time, or because serial and/or neonatal lesions have less traumatic effects. One cannot, in this sense, ascribe recovery to anything like a take-over of function, and the constraints concerning alternative behavioural modes of solution mentioned above should apply to this form of recovery as well. On the other hand, by potentially avoiding some of the *something more* effects such techniques as the serial lesion might come closest to producing neither more nor less than the intended damage.

5.2.3. LOGICAL OBJECTIONS

At a logical level problems have been raised about the interpretation of changes in behaviour following lesions; one tends to impute to a structure removed by lesion just those functions missing or aberrant in the operated animal (cf. Gregory 1959). Thus, in the case of the hippocampus a consistent attempt has been made to link this structure with some manner of inhibitory function, based primarily on the fact that animals with hippocampal lesions often appear to lack the ability to inhibit certain responses (or behaviours, or hypotheses) (cf. Kimble 1968, Isaacson and Kimble 1972, Altman *et al.* 1973, see Nadel and O'Keefe 1974, Nadel *et al.* 1975 for a discussion of these models). Gregory points out that such techniques lead to a position where a resistor is labelled a hum-suppressor because its removal from a radio leads to a hum. This, and other examples like it, attest to the fact that one must utilize lesion data with extreme care. On the other hand, there are cases where the imputation of function based on `what's missing' following a lesion is a useful method for proceeding. In a trivial sense the absence of vision after section of the optic tracts is one such example, and it seems clear that the nearer one is to the periphery the better this procedure works. The hippocampus, however, is not a peripheral structure; rather it appears to sit somewhere in the middle, concerned directly, we assume, with neither sensory input processes nor motor output mechanisms. The use of the missing-function technique here is more open to question.

A second point about the logic of the lesion technique concerns the question of control groups. To ablate a subcortical structure such as the hippocampus, using a suction method, it is necessary to remove a considerable amount of overlying tissue. Lesion control groups which have suffered damage only to the neocortex are therefore usually included in such studies. It is hard to know what conclusions to draw when the performance of these controls falls between the performance levels of the experimental lesion group and the intact control group, as is often the case. Even when the two control groups perform at the same level there are objections to the conclusion that any deficit in the experimental animals is wholly attributable to the subcortical damage. If there is both neocortical and hippocampal involvement in the generation of behaviour, then this simply is not so. In that case a lesion in either the neocortex or hippocampus will have no effect, while a lesion of both areas will have an effect. Thus, one could argue that the subcortical structure can be involved, but not that its involvement is essential. The double-dissociation technique would seem to offer a more solid base for reasonable inference.*

We shall close this discussion with a more general critique of the lesion method as it is most frequently used. As one reads through the literature one cannot help but be surprised at the small amount of information that is generated by each individual experiment and at the general absence of any important relationship between different experiments, even when they are done in the same laboratory. We think this is due to a fundamental misconception concerning appropriate methodology; that is, to the almost exclusive reliance upon an inappropriate experimental paradigm, the *crucial experiment* (cf. Platt 1964). The crucial experiment is designed to decide between two strong hypotheses where they can be shown to make mutually exclusive predictions. As such, it assumes a considerable prior knowledge of the phenomenon under study and seeks to control or eliminate all the relevant variables except those pertinent to the distinction between the two hypotheses under test. The crucial experiment produces very little information, but what it does produce is held to be extremely important; it is an 'uptight' business, the serious side of a 'mature' science.

At earlier stages in the development of a science different, more information-rich, paradigms are needed. Here it is necessary to make fewer assumptions about existing knowledge, and the emphasis is on the careful observation of natural phenomena and the selective interference with these processes in an attempt to understand them. Emphasis is placed not on the individual isolated experiment but on the accumulation of information about the phenomenon through the accretion of many small experiments. The atmosphere, one might say, is that of play rather than that of work.

Most of the experiments reported in this section follow the crucial-experiment paradigm although they were not designed specifically to place in opposition two strong hypotheses. Typically, animals are studied in artificial environments constructed to eliminate most stimuli and calculated

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* In the double-dissociation technique at least two experimental lesion groups are included and several behavioural tests employed. The hope is to demonstrate a defect in some tasks with one lesion and in other tasks with the second lesion, thereby doubly dissociating the lesioned areas.

to force the animals to learn a particular task in a specific way. Differences between groups are (or are not) found and it is concluded that the group with a certain lesion lacked (or retained) a given function. Information generated in this way is useful only if the conceptualization of behaviour guiding the research is correct. If such things as response inhibition do not exist then experiments testing the effects of lesions on response inhibition cannot be very helpful.

The more ethological approach suggested above would, in our view, be preferable at this stage of our knowledge. Rather than mask the fact that different animals learn the same task in different ways, this could be exploited and studied. Animals would be trained on tasks which utilize their natural repertoires as much as possible, with as much information available as could reasonably be provided. Following (or in some cases during) learning, the experimental situation could be systematically manipulated with the aim of determining how each animal solved the task. This emphasis on intra-animal differences would suggest that each animal be run through a series of studies so that any consistent pattern of learning strategies could be revealed.

Among the few lesion studies using this approach are those by Means and Douglas (1970) and Hamilton (1972). In experimental work using intact animals such techniques are used more often, as in transfer studies in discrimination learning and in transfer-of-control studies in avoidance learning. Of course, such techniques can be criticized; in particular one can object that they yield data contaminated either by new learning or some manner of interference from one situation to another.

We have been developing an experimental paradigm, which we call the *probe technique,* in order to assess whether brain-damaged animals learn in the same manner as do normal animals, without respect to learning rates. Animals are trained on a task to a loose criterion and then given a small number of additional trials on post-criterion days. Interspersed amongst these trials are the `probe' trials in which certain aspects of the situation are manipulated. Normal trials are given both before and after these probe trials to determine whether the probes have had any residual effects upon criterion performance. Using this technique we have been able to show that equivalent learning rates can mask qualitatively different modes of learning; some of these results are described in the course of this review.

In sum, a cognitive approach to brain-behaviour interaction demands the use of experimental techniques which allow for the possibility that a given task can be solved in any of several ways. Though we are aware of the objections one can raise concerning the use of lesion techniques, we feel that these techniques can be used profitably so long as the shortcomings are kept firmly in mind. In this review we rely heavily upon the lesion data, partly because of the great wealth of it and partly because much of it can be accounted for within our theoretical framework. The striking thing about the accumulated lesion data on the hippocampus, in fact, is the consistency of results across different lesion techniques, sizes, and sites when viewed from what we consider to be an appropriate theoretical perspective. In the discussion which follows we attempt to analyse the syndrome resulting from hippocampal damage both in terms of the putative `missing function' of the hippocampus and in terms of the functions of what is left to the animal's brain.

6

Exploration

6.1. Novelty

THE ongoing activity of any animal is a complex mixture of numerous separate activities, including such things as walking, eating, drinking, sniffing, grooming, sleeping, and so on. Each species engages in a mixture of activities that is, to some extent, unique to that species. Similarly, reactions to particular forms of external stimulation may vary from species to species. However, within this broad range of genetic variability there are patterns of behaviour that remain constant across species. Thus, in the face of threat most animals seek to flee to safety, or remain immobile, or engage in some form of attack. Similarly, in the presence of food, water, or a sexual mate, most animals (assuming a state of need) engage in some form of approach and consummatory behaviour. Lastly, when confronted with biologically neutral stimuli most animals tend to ignore these stimuli and attend to something, or somewhere, else.

These patterns, however, all refer to classes of external stimulation about which the animal has knowledge (innate or acquired). Reactions to stimulation, the significance of which is uncertain, follow a different course. Initially, there is usually a cessation of overt responding during which the animal prepares itself for, and engages in, the registration of information; only then is some overt response emitted which could involve approach or avoidance. This pattern of behaviour is, to some extent, dependent upon the occurrence of genuine *novelty,* that is stimulation never before encountered, but portions of this behaviour pattern can reflect the occurrence of any transitory change in the external environment. In this chapter we discuss the nature of novelty and change, the course of an animal's reactions to these, the role played in these reactions by the locale and taxon systems, and the ways in which the hippocampal animal's reactions differ from those of the normal animal.

6.1.1. NOVELTY AND NOTICEABILITY

It is important to distinguish between two different properties items or places can have for an organism. We shall define *novelty* as a property of those items or places which have not been experienced before. *Notice*ability refers to a property of those items or places which attract attention.

Part of the confusion between these two arises from the fact that in the intact organism novelty almost always implies noticeability as well, though the converse is not the case.

Few items or places are completely novel; novelty typically consists in new configurations of familiar elements. New pictures hide old lines, old colours; the novelty of the wife in the best friend's bed lies neither in the wife, nor the friend, nor the bed, but in the unfamiliar conjunction of the three. Novelty 'wears off' rapidly with several exposures; repeated exposures to items or places *with the opportunity to explore* leads to a growing sense of familiarity. On the other hand, temporal parameters *per se* do not seem to be important; an event recurring after a year is no more novel than when it recurs after a day. Novelty, then, would seem to depend on a long-term memory sensitive to contextual (typically spatial) configurations and capable of remembering single occurrences. Within the framework of the present theory *an item or place is novel if it does not have a representation in the locale system* and thus excites the mismatch cells in that system.

The implication of this is that novelty is a matter of 'an item in a place'; the location of an object is an integral part of what constitutes its novelty and the uncertainty its occurrence elicits. This aspect of novelty, though rarely commented upon, is supported by several types of research. Peeke and Veno (1973) studied the aggressive responses of fish as a function of both the qualitative properties of the target fish and its location. Responses to a target located in the same place show a steady decrement; this is the process known as *habituation.* A change in either the target or its location reinstated the aggressive response. Thus, habituation, or the decrement in response with increasing familiarity, depended upon the location of the eliciting stimulus. Similar results were reported by Shalter (1975) for the response of birds to calls. This principle applies to neutral, as well as to biologically meaningful, stimuli. Corman, Meyer, and Meyer (1967) and Corman and Shafer (1968) have shown that exploratory responses can be a function of the spatial location of a stimulus.* A more convincing result has been reported by Wilz and Bolton (1971). Gerbils were allowed to explore, in an open field, either a group of objects in a particular spatial arrangement or a single object located in a specific place. The rearrangement of these objects (or a new location for the single object) was as effective in eliciting exploration as a totally novel environment. They conclude that their animals were 'sensitive to change in the spatial orientation of

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* In the latter study rats were tested in a black open field with one white square in the centre. After this exploration had waned the white square was replaced by a black one; this 'new' stimulation elicited renewed exploration. Dember (1956) has shown similar effects in a spontaneous alternation situation (p. 260). The nice thing about such tests is that, on the crucial-test trial, they distinguish between the qualitative properties of the stimulus and its location; the field is homogeneous, but certain places are novel. Another example of the effectiveness of familiar stimuli in new places is reported in a study by Karmos et al. (1965), as discussed on p. 167).

familiar objects, even when the position of a single object is involved' (p. 118).

Noticeability, on the other hand, *refers to the degree to which a stimulus excites the nervous system* and elicits a variety of reflexes including the orienting reflex or directed attention. To borrow from part of James's (1890) catalogue of things which attract attention, loud bangs, shiny things, blood, and so on are highly noticeable, though not necessarily novel. The important variables controlling reactions to noticeable stimuli appear to be the recency and frequency of the same or similar experiences, the rate of change, and good or bad associations.* On the present view these context independent reactions are a function of the extra-hippocampal taxon systems. Note, however, that in so far as novelty also involves noticeability, novel items and places will elicit reactions appropriate to both noticeable and novel events.

6.1.2. BEHAVIOUR ELICITED BY NOVELTY/NOTICEABILITY

6.1.2(a). Exploration. The hippocampal locale system is assumed to form the substrate for maps of environments an animal has experienced; *these maps are established in the hippocampus during exploration, a species-specific behaviour pattern concerned with the gathering of information.* Animals are assumed to attend only momentarily to those neutral aspects of the environment which are predicted from the map; the appearance of a biologically meaningful event elicits approach (and consummation) or avoidance, and thus allows the taxon system temporarily to control behaviour. However, attention can also be captured, within the locale system, by those aspects of the environment which are unpredicted, or novel. Investigation of this discrepancy ensues, either to fill out the map or to modify it so as to delete features now missing from the environment.** Thus, *exploration is a direct response of the animal to the detection of a mismatch by the locale system; in the absence of the hippocampus all forms of exploratory behaviour should disappear from the animal's repertoire.*

6.1.2(b). Other reactions to novelty/noticeability. Traditional theory (e.g. Montgomery 1955) assumed that novelty elicited two states within an animal, *curiosity* and *fear,* and that ensuing behaviour was the result of competition between these two. The dominant response to curiosity was exploration, while the response to fear was either withdrawal or immobility. This distinction is analogous in certain ways to that made by Sokolov (1963b) between the *orienting* and *defensive reflexes* elicited by unexpected stimulation.

Learning theory based on drive reduction models of behaviour has had inordinate difficulty incorporating the existence of exploration. Recently, in one attempt to deal with this problem, Halliday (1968) suggested that novelty elicits only a state of fear, that exploration is motivated by this fear, and that the information obtained during exploration subsequently serves to reduce fear. We prefer to assume that exploration is a specific response to novelty, that it competes with fear, and that it can serve to reduce fear as an indirect by-product (cf. Russell 1973). We do not feel it necessary to account for exploration in terms of hypothetical underlying drives; it is assumed only that exploration is emitted in response to external (unpredicted) stimulation and that its function is the gathering of information about that stimulation, pursuant to the construction of maps.

Most normal animals, however, do display a certain amount of hesitancy when confronted with novelty, and it can be assumed that this reflects fear elicited by novelty. The choice on the part of the animal between approach behaviours, including exploration, and withdrawal behaviours depends upon a number of factors (cf. Bronson 1968) and shows wide individual variation. It is beyond the scope of the present discussion to elaborate on these, though we do consider the nature of reactions to fear-eliciting events later (pp. 294-6). In the remainder of this chapter we concentrate upon those reactions to noticeability and/or novelty which result when generalized fear is not the dominant state elicited by the environment.

Similarly, we do not discuss at length the nature of an animal's reaction to unpredicted stimulation from biologically meaningful stimuli, be they potentially rewarding or punishing. References are occasionally made to such stimuli in order to show that the basic principles applied to neutral stimuli hold for meaningful ones as well, in particular when we discuss the nature of responses to repetitive stimulation. Of course, a number of behavioural situations that we discuss later, such as extinction, discrimination reversal, and reward shifts, all incorporate features of novelty. The animal's ability to adjust rapidly to these changes depends to some extent on the occurrence of exploration. In so far as such exploration is assumed to be lacking in animals without a locale system the discussion of reaction to novelty in the present chapter is germane to our subsequent discussion of these other situations.

6.2. The form of reactions to novelty/noticeability

An animal's immediate reactions to novelty/noticeability should be viewed as preparatory ones which facilitate the reception of information and the subsequent choice among approach, avoidance, or a lack of interest. Intense stimulation, novel or not, tends to elicit a *startle reaction.* In its most developed form this consists in limb extension followed by generalized

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* Whereas novelty is primarily related to long-term representations, noticeability is basically a short term process, as can be seen in its sensitivity to recency and frequency of stimulation. This separation between long-term and short-term effects is reflected in the kinds of models derived to explain the habituation to novelty and noticeability, as we shall see shortly.

** By delete we do not wish to take the strong position that old maps are destroyed, only that an up-dated map becomes available.

flexion leading to a crouching posture, all within 10-20 ms of stimulation (Fleshler 1965, Horlington 1968). The crucial determinants of the startle reaction, which can be classified as a defensive reflex, are the intensity and suddenness of the stimulating event (Landis and Hunt 1939, Fleshler 1965), where intensity is best conceived in terms of signal-to-noise ratios (Davis 1974). Less intense stimulation, which fails to elicit the defensive pattern of the startle reaction, may nevertheless elicit a similar behaviour which can be termed an arrest reaction. This brief reaction consists in a cessation of ongoing activities, a maintenance of the posture exhibited at the onset of stimulation, and various autonomic changes (e.g. Grastyan et al. 1965). The arrest reaction is rapidly followed by a complex of reactions traditionally labelled the orienting response; it includes changes in muscle tonus, respiration and circulation, neuro-endocrine responses, desynchronization of the cortical EEG, and the orienting of the body and sense organs towards the source of stimulation (Sokolov 1963a,b). All these reactions play a role in preparing the animal to register and analyse the stimulation.

Following the orienting response the normal animal will either engage in overt exploration, moving towards and actively investigating the source of stimulation, or it will engage in some form of defensive behaviour. *Only novel stimulation will elicit exploration;* noticeable familiar stimuli will elicit either approach or avoidance in accordance with their biological meaning, or will be ignored if they are insignificant for the animal.

6.2.1. THE RELATIONSHIP BETWEEN EXPLORATION AND RESPONSES TO NOTICEABILITY

The early responses to noticeability just described (with the exception of the startle response) have often been lumped together with exploration under the general rubric of *orienting behaviour.* This characterization, however, overlooks the facts; these early responses neither necessitate exploration, nor are they uniquely elicited by novelty, as is exploration. Further, this does not appear to have been Sokolov's (1963a) intention:

> 'The orientation reflex in the restricted sense of the word, should be distinguished in the reflex as the non-specific reaction resulting in the tuning of the analyser when exposed to a new stimulus. This elementary reaction is quite distinct from the complex exploratory chain of reflexes, aiming at investigation of the object in detail, and involving a whole series of conditional orientation reflexes' (p. 11).

Thus, there are ample reasons to assume that the early reactions to noticeability form a cluster of responses different from exploration. Particularly important in the present context is the fact that the various components of an organism's reaction to noticeability/novelty develop differently with repeated elicitation. In most, but not all, cases repetitive elicitation causes a decrement in response, termed *habituation.* The habituation of startle, arrest, and orienting, as well as the change in response to repeated presentations of inherently meaningful stimuli, would appear to follow one set of laws which we shall associate with taxon function. The habituation of behavioural exploration would appear to follow different laws which we shall associate with locale function. We can best document these assertions by briefly considering the general problem of habituation and the theoretical models currently proposed to account for it.

6.2.2. MODELS OF HABITUATION

In general, two types of model have been proposed to account for decrements in response with repeated elicitation. The first class draws its data primarily from the study of short-term response decrements to repetitions of biologically significant stimuli such as those eliciting spinal reflexes, startle reactions, and the species-specific behaviours of the ethologist. Groves and Thompson (1970, 1973; see also Groves and Lynch 1972, Thompson *et al.* 1973) present such a model and suggest that it can account for habituation of the orienting reflex as well as the above behaviours. They postulate two interacting effects of repeated stimulation, one involving sensitization (or an increment in response), the other habituation. These properties are in accordance with the following facts noted by Groves and Thompson concerning short-term sensitization and habituation: (1) during the initial repetitions of a stimulus incremental, rather than decremental, effects are often seen; (2) the extent of the incremental effect is dependent upon the intensity of the stimulus; (3) the incremental effect decays spontaneously upon termination of the stimulus; (4) both incremental and decremental effects are related to the frequency of stimulation in a complex fashion depending in some measure upon intensity; (5) both incremental and decremental effects to an initial repeated stimulus will generalize to another stimulus to the extent that the two stimuli share common elements.*

It should be noted that one can often see quite complex mixtures of incremental and decremental factors operating simultaneously in the response to a repeated stimulus. For example, the changes in the mobbing response of chaffinches repeatedly elicited by a stuffed owl can best be viewed as a decremental process for that exact stimulus combined with an incremental process for similar stimuli (Hinde 1970). Kimble and Ray (1965) have shown that repeated elicitation of the frog scratch reflex leads to decrements if the stimulus is applied in exactly the same spot but increments if the stimulus location is varied, while Ewert and Ingle (1971) have reported similar phenomena for toad's and frog's prey-catching responses. Explanations for these phenomena may reside in the different time courses for incremental and decremental processes within the

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* There is a complex, and confusing, relationship between the processes postulated by Groves and Thompson and those postulated by us for the taxon systems. Their processes of sensitization and habituation are both essentially short-term mechanisms and are best seen in the absence of biological rewards or in simplified reflex systems.

hierarchically organized neural systems mediating the reception of the stimulus and the generation of the response (cf. Hinde 1970). Such a proposal is consistent with the local nature of the presumed effects underlying these processes. Thus, this model of habituation, which can be termed the 'self-generated depression' hypothesis (Thompson and Spencer 1966, Horn 1967, Wall 1970, Wickelgren 1967a,b), has been ascribed either to a failure of synapses with use (Thompson and Spencer 1966) or to the parallel activation of an inhibitory pathway (Wickelgren 1967b, Wall 1970). It is important to remember that these models of habituation have been derived from the study of short-term decremental processes and do not necessarily apply to decrements lasting for long periods; Groves and Lynch (1972) explicitly state that their model is meant to account solely for short-term decrements.*

The second model of response decrement with repeated elicitation, associated primarily with Sokolov (1960, 1963a,b), was designed to explain habituation of the orienting reflex to a neutral novel stimulus. According to Sokolov the neo-cortex maintains a model of the stimuli in the environment. Novel stimuli produce a mismatch in this system which signals the reticular formation and thereby produces the orienting reflex. As a model of the stimulus is built up in the neo-cortex the mismatch signal decreases and the orienting reflex habituates. For his part, Sokolov (see Pakula and Sokolov 1973) has indicated that this neuronal model approach is not required when one considers habituation in simplified systems.

Groves and Thompson (1970) argue that habituation of the orienting reflex can be accounted for within their dual-process model, and bearing in mind Sokolov's crucial distinction between orienting and exploring we would agree. It does not appear necessary to postulate the existence of Sokolov-type neuronal models to account for the orienting reflex and its habituation. The self-generated depression mechanism would seem sufficient to handle this. Thus, habituation of orienting reflex components has been reported in unconscious patients whose neo-cortical EEG's were 'very pathological' (Gulbrandsen, Kristiansen, and Ursin 1972).

Exploration of novelty, on the other hand, cannot be analysed in the same manner. There is no obvious relation between frequency and intensity of novelty and exploration. In fact, one could argue that novel environments could not even be described in such terms. Further, habituation of exploration follows a much different time course than does habituation in the other systems; there is no direct analogue to the sensitization often seen in these systems, and the decrement itself usually lasts much longer.

We have suggested that exploration is driven by the occurrence of unpredicted predicted events and that it ceases when the source of novelty is incorporated into the cognitive map. In a sense, then, a comparator model akin to that postulated by Sokolov, directing exploration rather than reflex orienting, is assumed to exist within the hippocampus.

Thus, two general modes of reaction to noticeability/novelty are postulated for the intact animal: a series of reactions characterized by startle, arrest, and orienting associated with taxon function and of an essentially short-term nature; active investigation of novelty, associated with the locale system and having relatively long-term consequences. On the basis of this distinction we would expect hippocampal animals to show relatively normal startle, arrest, and orienting, including normal short-term habituation of these responses, but not to show behavioural exploration. In their dependence upon taxon systems we would further expect the reactions of these animals to be unusually dependent upon the frequency and intensity of stimulation.

6.3. Effects of hippocampal lesions on reactions to novelty/noticeability

Studies of reactions to novelty/noticeability can be subdivided into those involved with (1) reactions to novel items introduced into basically familiar environments (Table A13), (2) reactions to novel environments (Table A14), and (3) spontaneous alternation (Table A15). By virtue of the procedures used in these studies the first category is primarily concerned with the immediate responses to noticeability, while the remaining two categories are more directly concerned with behavioural exploration. We attempt to show in this review that hippocampal animals react in an essentially normal fashion as regards startle, arrest, and orienting but that they lack exploration. A more detailed discussion of the nature and determinants of exploratory behaviour in normal animals is included prior to the discussion of those studies which attempted to measure it directly. Finally, we briefly discuss several studies investigating *latent learning,* which is typically assumed to involve the acquisition of information during exploration.

6.3.1. A NOVEL ITEM IN A FAMILIAR ENVIRONMENT

An important distinction must be drawn between two related paradigms involving the introduction of a novel item into a familiar situation; this concerns the ongoing behaviour of the animal. In one case the animal is engaged in some directed activity during the introduction of the novel item (the *competitive* case), while in the other the animal is not so engaged (the *non-competitive* case). The relevance of this distinction will be made clear in the ensuing discussion.

In both of these situations one can measure either autonomic or behavioural responses to noticeability/novelty. Unfortunately, experiments using either type of measure have rarely taken into account the differentiations

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* Two recent papers by Williams, Hamilton, and Carlton (1974, 1975) document the independence of habituation in the two systems. Anticholinergic drugs influenced the habituation of exploration but not that of the startle response. The latter showed habituation in both 15 and 36 day old rats; exploration habituated only - in 36 day olds. The authors concluded that the habituation of reflexes could not be viewed in the same manner as that of exploration.

between the various components of an animal's response to unpredictability. Thus, psychophysiological studies typically confine their measurements to a single response (usually heart rate or galvanic skin response) whose relation to the other components of the overall response is left unspecified, while behavioural studies often ignore the important distinctions between arrest, orienting, and exploration.

6.3.1(a). Reactions in the absence of directed behaviour. Psychophysiological measures of an animal's response to a novel item have been taken in a number of studies. Bagshaw, Kimble, and Pribram (1965) presented novel tones to monkeys and measured galvanic skin responses. Hippocampal monkeys showed completely normal reactions to the initial presentations of the tone, to its repetition, and to the introduction of a new, dishabituating tone after response to the initial tone had habituated. Crowne and Riddell (1969) and Sanwald *et al.* (1970) measured cardiac responses to the introduction of novel items, either tone and light combined or tone alone. Work with normal animals indicates that the initial presentations of a moderately intense novel tone produce cardiac deceleration, while later presentations of the same tone may elicit cardiac acceleration (Flynn 1960, Stern and Word 1961, Black 1964, Weisbard and Graham 1971). It has been suggested that the initial deceleration is probably associated with arrest and orienting, while the subsequent acceleration is associated with investigation (Lynn 1966). In both the Crowne and Riddell and Sanwald *et al.* studies the initial deceleration was seen in hippocampal rats; in the latter study the development of the acceleratory response with stimulus repetition was also measured and found to be absent in hippocampal rats. Thus, these studies suggest that the autonomic aspects of orienting, but not exploration, are intact in hippocampal animals.

Behavioural reactions to novel items were measured in several other studies. Startle responses to intense stimulation were reported to be normal (Kemble and Ison 1971) or somewhat exaggerated (Ireland and Isaacson 1968, Coover and Levine 1972) in hippocampal animals; the habituation of startle was normal in all these studies. This exaggerated startle response will be considered shortly when we discuss the general problem of hyperreactivity in hippocampal animals.

Hendrickson, Kimble, and Kimble (1969) measured the orienting response (defined as an arrest reaction and/or somato-motor orienting) to the introduction of novel stimuli. Under conditions where their rats were not engaged in any directed activity hippocampals showed normal orienting behaviour. Kim (1972, and personal communication) reported that in the non-competitive situation hippocampal rats showed significantly less *strong* orienting behaviour (which involved non-transitory reactions) but no deficit in *weak* orienting behaviour (which involved transitory reactions). Hippocampal rats habituated all orienting responses at the same rate as did control rats. Thus, these studies suggest that the initial stages of orienting are intact in hippocampal animals, and that these reactions habituate normally as well. Later stages, which might involve active exploration, appear to be absent.

In two studies by Rogozea and his colleagues (Rogozea and Ungher 1968, Ungher *et al*. 1971) changes were seen in hippocampal and fornical cats in orienting to repeated neutral stimuli but not in orienting to biologically significant stimuli such as food or tail pinch. Both normal and lesioned cats oriented to the initial presentations of the neutral stimulus. Repetition of the tone, which was presented for 3 s duration at 10 s intervals, led to rapid habituation of orienting in the normal cats. Lesioned cats, however, developed a pattern of compulsive motor activity which took a considerable time to decrement. While the authors felt that this pattern masked defective habituation, this interpretation is not warranted by the data; the motor activity shown by the lesioned cats during the later stages of the study was unlike the orienting behaviour they showed earlier. Similar results for reactions to meaningful stimuli were seen in a study by Nonneman and Kolb (1974). Hippocampal cats oriented normally to model cats or the smell of urine introduced into a novel environment. Habituation to these stimuli was virtually normal, though there were some changes. This situation, of course, shades into a study of fear responses which we shall consider in greater detail later (pp. 294-315).

Some informal observations of our own could be mentioned here. We placed normal and hippocampal rats in a relatively small open box with low sides and tested their responses to various weak auditory stimuli. Their responses to scratching the side of the box with a pen top every minute or so were especially interesting. Normal rats ceased responding after a few such stimulations, showing dishabituation when the location of the stimulus was shifted or the sound markedly changed. Hippocampals, on the other hand, became quite agitated with each stimulation; regardless of what they were doing at the time they almost invariably began to run in response to the scratching sound. At times they ran in the general direction of the stimulus as long as it was maintained, while at other times they simply ran forward. The running usually ceased with stimulus offset. When the frequency of stimulation was increased to once every 5-10 s the running pattern decremented with a few minutes. Neither the running we observed, nor the compulsive motor activity reported by Ungher *et al.* can be considered orienting behaviour. Rather, these data suggest again that hippocampal animals can be hyper-reactive to certain stimuli. Further, they indicate that *the interval between repetitions of the eliciting stimulus is an important variable determining the occurrence of response decrement in hippocampal animals.*[*]

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[*] In general, these results agree with the Groves and Thompson model concerning the correlation between habituation and repetition rates.

There is some direct evidence that behavioural exploration of a novel item in a familiar environment is deficient in hippocampal animals. Glickman, Higgins, and Isaacson (1970) have shown that hippocampal gerbils react to, and explore, novel objects placed into their home cages significantly less than do normal gerbils. Similarly, Ungher *et al.* (1971) note that while their fornical cats orient normally to meaningful stimuli they are deficient in their investigatory reactions towards these stimuli. Finally, Dalland (1976b) has shown that hippocampal rats do not react to novel objects introduced into their living situation.

Thus, available evidence suggests that hippocampal animals show normal startle, arrest, and orienting, and that their habituation of these reactions upon stimulus repetition proceeds normally as well. However, little behavioural exploration of novel items is seen in these animals. Lastly, there are several indications that hippocampals over-react to some stimuli in a compulsive manner, though these aberrant responses can be made to decrement with appropriate inter-stimulus intervals.

6.3.1(b) Reactions in the presence of directed behaviour. A more powerful test of the attractive force of a novel item is to introduce it while the animal is engaged in some directed activity, such as a motivated task. The normal animal is usually distracted from its task and explores the novel item. If, for instance, a rat is running for food in an alley and white walls are suddenly introduced on a particular trial, the rat will stop and explore the new walls.

Hendrickson, Kimble, and Kimble (1969) measured orienting responses to novel items while rats were either drinking or attending to the source of a massed barrage of tones. There was a strong suggestion in this experiment that the hippocampal rats totally failed to orient while they were engaged in these other tasks. Similarly, Wickelgren and Isaacson (1963) report that hippocampal rats fail completely to orient to a distracting stimulus (white walls) introduced while the rats were running in an alley for reward. There seems little doubt that in these competitive situations hippocampal animals are less distractible than are normal animals (see Table A13); the question is whether this signifies a total lack of response, or merely an absence of exploration. In a recent paper Harley (1972) discusses an interesting aspect of the Hendrickson *et* al. paper that was not pursued in the original report. She states that

> 'In one condition the same hippocampally lesioned animals were tested for orienting to an auditory stimulus first in a competitive and then in a noncompetitive situation. They showed no orienting in the first situation as expected but oriented in the second situation at the same level as control rats which had habituated to the stimuli during the first presentation. This implies that the first experience had affected the hippocampally lesioned animals despite their behavioural unresponsiveness.
>
>
>
> Naive animals both lesioned and normal, oriented at clearly higher levels when tested in the noncompetitive situation without prior exposure to the competitive one' (p. 347)

Thus, the 'failure of orienting' in the competitive situation, as reflected in the behavioural measures used in the Hendrickson *et al.* (1969) study, may have resulted from the lack of exploration, rather than orienting. In the original report the authors attributed what they conceived as a total lack of distraction to an inability to ‘shift attention during the presentation of novel stimuli or in mismatch situations’ (p. 226). The comments of Harley (1972) indicate that this cannot have been the case. The hippocampal animals clearly attended to the novel stimuli (in the sense that they perceived and habituated to them), though they did not investigate them directly.

Kim (1972, and personal communication) has also tested the orienting reactions of hippocampal rats in a competitive situation similar to that used by Hendrickson *et al.* As before, Kim separated orienting responses into those which were weak or strong, with the defining difference between the two being the persistence of the response. Kim found that hippocampal rats in the competitive situation showed significantly less strong orienting, significantly more weak orienting, and significantly shorter durations of orienting when both categories of responses were combined. Thus, Kim's data suggest that the primary defect in orienting behaviour in hippocampal rats in competitive situations is that they orient for a shorter period of time and in a weaker fashion. This is consistent with the view that they do not fail to shift attention, but rather fail to react beyond an initial, transitory, orienting response.

Other workers have similarly stressed the fact that the deficit in distractibility is rarely complete. Thus, Raphelson, Isaacson, and Douglas (1965) and Riddell, Rothblat, and Wilson (1969) report that the latency of hippocampal rats in a runway is affected by the introduction of novel stimuli, though this effect is considerably less than that seen in normal animals. Similarly, Crowne and Ridden (1969) report that an increase in latency to respond on a discrimination problem in hippocampal rats upon introduction of novel stimuli indicates the partial nature of the defect. A study by Kaplan (1968) provides an important clue to the nature of the hippocampal impairment in these situations. Rats were trained to press a lever for food and were then tested for their reactions to the introduction of a flashing light. Kaplan noted that control rats ‘typically froze when the flashing light was initially introduced, then explored the area surrounding the light before starting to bar press again’. As to the hippocampal rats: 'when the light was first introduced, there was a brief startle reaction, followed by orientation toward the light' (p. 277-278). The hippocampal rats returned to their lever-pressing behaviour quite rapidly. These observations suggest that hippocampal animals, even while engaged in some directed activity, show normal arrest and orienting responses to a novel stimulus. However, they fail to explore the stimulus, returning instead to their motivated task. Thus, Cohen (1970) and Cohen and Swenson (1970) report that hippocampal rats explore distracting stimuli significantly less than do normal rats. Their animals were trained a runway response and then subjected to the introduction of a novel side alley. The hippocampal rats almost never entered this new alley.

The most startling example of distractibility in hippocampal animals engaged in directed activity was provided by Douglas (1972). Monkeys were engaged in a sequential response task; on certain trials the first response in the sequence initiated a loud buzzer. Both control and hippocampal monkeys were visibly startled by this buzzer; some literally hit the roof of the apparatus. Normal monkeys ‘failed completely to complete the sequence and instead turned away from the stimulus and investigated the source of the buzzer’ (p. 536). They returned to the task a minute or two later. The hippocampal monkeys ‘though startled, completed the sequential response while on the way up’ (p. 536). After settling down they secured the reward before attending to the source of the buzzer.

We can see from these observations that a strong distracting stimulus will affect hippocampal as well as normal animals, though the former seem to lack the normal tendency to explore the distracter and have a strong tendency to complete any behavioural sequence that they have begun. These findings suggest that the reactions of the hippocampal animal are dependent not upon novelty *per se,* but rather on the relative intensity (or noticeability) of the various stimuli in the environment. In any case the hippocampal animal fails to react to stimuli, novel or otherwise, with exploration, though startle, arrest, and orienting can be intact. This has been directly confirmed in a recent study by Gustafson (1975), who found that distraction durations were lower in hippocampal rats and that this was due to reduced exploration coupled with normal orienting. This dissociation between the effects of any stimulation and the exploration-eliciting effects of specifically novel stimulation is an important one, to which we shall return shortly.

As a final point we should mention an apparent exception to the general finding of decreased distraction in hippocampal animals reported in a study by Douglas and Pribram (1966). Their lesioned monkeys displayed greater than normal distraction under certain circumstances when the distracter was introduced into a discrimination procedure. The most plausible explanation for this finding is that the distracter was so similar to the stimulus controlling the animal's ongoing behaviour as to be confused with it; we shall consider this study in greater detail later (pp. 275-6). However, it is worth noting that this increased distraction was only seen when a new stimulus was used; it was not seen when a familiar stimulus (to which the animals had already stopped responding) was introduced in a new place. This clearly indicates that 'a familiar stimulus in a new place' is not treated as a noticeable event by hippocampal animals.

6.3.2. GENERAL ACTIVITY AND EXPLORATION IN NOVEL ENVIRONMENTS

Responses to novelty have also been tested by placing animals in novel environments and measuring their reactions, usually in terms of rather gross activity scores. Before we consider the behaviour of hippocampal animals in such situations we need to discuss the way in which measures of general activity are related to the occurrence of exploration.

6.3.2(a). General activity and exploration. Behaviours that are active, but without any obvious goal such as food or water, have commonly been referred to as exploratory. However, it was recognized almost 20 years ago that not all of such activity could be classified as exploration (e.g. Montgomery 1953b). As Bindra (1959) pointed out, *general activity* is concerned not with the 'incidence of a given class of response' but with the 'extent to which an animal makes *any* type of response' (p. 31). *Exploration,* on the other hand, according to Bindra, 'refers to the incidence of only certain classes of acts . . . associated with . . . novel environmental stimuli' (p. 33). Novelty, of course, can only be specified in relation to an animal's past experience. Halliday (1968*)* suggested that

> 'an animal explores a stimulus situation if it cannot match it on the basis of its past experience . . . An animal would therefore explore a situation with which it was unfamiliar until its “internal representation” of the situation matched the external environment' (p. 113).

Exploration, in that it is aimed at learning about novelty, decrements with continued exposure to an initially novel environment (e.g. Glanzer 1961). However, an animal no longer exploring one environment will immediately begin to explore another, new environment (Halliday 1966). As we have seen, this re-activation of exploration also follows from a spatial rearrangement of familiar objects in the same 'environment'.

The above indicates that general activity, as a behaviour separable from exploration, can only be appropriately measured under *constant* and *familiar* environmental conditions, with no confounding element of environmental change. Such activity shows a regular circadian rhythm; that is, it varies over time in a cyclical fashion (Richter 1922). It is increased by hunger (or thirst) in a variety of situations, including open fields (Fehrer 1956) and complex mazes (Dashiell 1925). Moreover, *general activity is greater in the presence of increased environmental stimulation, though this need not be novel stimulation* (Hall 1956). By comparison, exploration is either decreased by food deprivation (Montgomery 1953a, Zimbardo and Montgomery 1957) or is marginally increased (Bones and de Lorge 1962). *It is substantially increased only in the presence of specifically novel stimulation,* and this has led Bolles (1967) to conclude that its 'main determinants ... are associative' (p. 290). This differentiation between general activity and exploration was seen in a study by Halliday (1967); no correlation between the amount of activity and the extent of exploration in a Y-maze was found, though they both tended to drop out at about the same time.*

Berlyne (1966) has discussed two types of 'exploratory' behaviour under the terms 'specific' and 'diversive' exploration. Within a somewhat different framework he referred to some of the properties of exploration and general activity that we have just pointed out. Specific exploration, in Berlyne's terminology, is nearly identical to what we have called exploration. It occurs when an 'animal is disturbed by a lack of information' and it supplies the 'precise information that the animal misses' (p. 26). Berlyne's diversive exploration, which resembles (with certain qualifications) what we have called general activity, 'is not preceded by receipt of partial information about the stimulus patterns at which it is aimed, and thus seems to be motivated by factors quite different from curiosity' (p. 27). This distinction between exploration and diversive, or general, activity is being pursued at great length here partly because it has not often been made within the literature concerned with lesion effects on reactions to novelty. More important, exploration, as a response to specifically novel stimulation, is assumed to be a function of the cognitive-mapping system, while general activity, as a set of responses to any form of stimulation, is not.

Berlyne compares exploration, in humans, to epistemic behaviour, stating that it is

> 'aimed not only at obtaining access to information-bearing stimulation, capable of dispelling the uncertainties of the moment, but also at acquiring knowledge, that is, information stored in the form of ideational structures and giving rise to internal symbolic responses that can guide behaviour on future occasions' (p. 31).

This description, without the reference to symbolic responses, is close to what we mean by cognitive maps. With the distinction between exploration and general activity in mind and the prediction that only exploration is a function of the locale system, we can now turn to a discussion of the behaviour of normal and hippocampal animals in novel environments.

6.3.2(b). Responses to novel environments. In a novel environment the normal animal's behaviour reflects a mixture of curiosity and fear, as we have already noted. Typically,* the animal will remain quiet for a while, perhaps sniffing about its perimeter. Slowly, it will move out and explore its surrounding, often withdrawing back into areas already explored (and hence known to be safe). Once explored, an area is less likely to be visited again on a subsequent foray. In time, the animal will thoroughly explore the entire situation and will become relatively quiescent, or eat if it is hungry and there is food available. At this point we can say that the animal has completed its exploration of the novel situation. On subsequent exposures to the situation the animal might make a cursory check to ensure that nothing has changed, but its activity will be much less than it was on the first occasion. The tendency to explore is quite strong in normal animals; for instance, in a maze with several routes to the goal hungry animals will vary their paths, often taking some that involve more work rather than sticking to the same familiar path (Dashiell 1925).

This normal preference for novel situations over familiar ones reflects the predominance of curiosity over fear; this predominance is not inevitable. Thus, if a buzzer is sounded just prior to allowing an animal to choose between novel and familiar places (thus inducing a certain amount of fear) the animal will tend to prefer the familiar place (Montgomery and Monkman 1955), indicating that fear has become predominant. All this provides a picture of a curious, active animal, seeking out and exploring new situations but constantly pulling back from such novelty when afraid.

Compare this with the behaviour of hippocampal rats. Some move off immediately they are placed in a novel situation; others sit quietly for a while before becoming active. Once active, all hippocampals adopt a stereotyped pattern of behaviour; this typically involves moving about more than normals would do, with a noticeable tendency to go back to the same areas over and over again. In a large box this repetitive behaviour takes the form of running (Kimble 1963, Eichelman 1971), usually around the perimeter (Jackson 1967), and there can be surprisingly little decrement in this behaviour with time. Table A 14 lists those studies which have measured activity levels in a variety of situations. Except for a few cases the consensus is that hippocampals are more active than normals.

This difference could be due to one or more of the following causes: (a) increased exploration; (b) decreased habituation of exploration; (c) decreased fear of novelty; (d) increased drive; (e) increased general motor activity; (f) increased reactivity to stimuli. We shall consider each of these in turn.

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* The distinction between exploration and general activity comes out clearly in studies concerned with the effects of early sensory deprivation, which increases general activity, but not exploration (Montgomery 1953b, Nielsen 1971), as well as in studies of the effects of amphetamine treatments (e.g. Kumar 1969, Ibuka 1971).

* The typical rat, like the average man, is a statistical fiction. Our purpose here is to describe the behaviour of a well-handled rat whose personality falls in the middle of a continuum. We have seen introverted, scared rats which remain immobile for tens of minutes in novel situations, as well as exuberant extroverts which race off within seconds of entering a new situation. One cannot generalize to feral animals or other species, of course. Thus, wild rats are a good deal less curious about novelty (Barnett 1958).

(a) Increased exploration. There is no suggestion, either in the literature or in our own observations, that hippocampals engage in what is clearly exploratory behaviour. In line with the distinction between general activity and exploration, hippocampals would appear to be *hyperactive,* but at the same time *hypoexploratory* Thus, they do not selectively investigate novel, as opposed to familiar, objects, even when such objects are placed into their home cages (Dalland 1970, Glickman, Higgins, and Isaacson 1976b). Their responses to neutral objects often do not decrement from test session to test session. On the contrary, there is the possibility that responses to an object increase with the passage of time, at least with certain inter-response intervals; that is, that sensitization effects are seen. We describe an example of this shortly.

(b) Decreased habituation of exploration. Since there is no reason to believe that hippocampal animals explore, it follows that their hyperactivity is not due to a failure to habituate exploration. We would add, moreover, that given any meaningful definition of exploration the notion of exploratory behaviour which does not decrement with repeated exposure to the explored situation is a logical contradiction.

(c) Decreased fear of novelty. The observation that some hippocampals, like normals, do not begin to move for a few minutes when first introduced into a novel situation might be interpreted as fear of that situation. The fact that most do not hesitate in this way strongly suggests that there is a reduction in such fear in hippocampal animals. The failure of all hippocampal animals to respond immediately in novel situations means that activity measures must not be too brief if they are to reveal differences between normals and hippocampals. The consistent failure to find such differences in one laboratory (Bender, Hostetter, and Thomas 1968, Hostetter and Thomas 1967, Spiegel, Hostetter and Thomas 1966) might be due to the fact that they observed their animals for only four minutes in an open field.

Evidence consistent with the hypothesis of decreased fear of novel situations comes from the study of Glickman *et al.* (1970). They found that hippocampal gerbils defecate less in the open field than do normal gerbils; Kimble (1963), however, found no differences between normal and hippocampal rats on this measure. Nonneman, Voigt, and Kolb (1974) also reported no differences on this measure, but they felt that both defecation and urination were poor indicators of emotional reactivity. Perhaps better evidence concerns the finding that hippocampal rats, unlike normal rats, do not attempt to jump out of the open field box (Jackson 1967, Eichelman 1971). Jarrard and Korn (1969) have shown that hippocampal rats have somewhat lower heart rates than do normal rats in the open field, in spite of their increased activity. Finally, Jarrard (1968) has shown that hippocampal rats will begin to eat in an open field much more readily than will normal rats. This could reflect both decreased exploration and decreased fear.*

(d) Increased drive. The repetitive running behaviour of hippocampal rats in the open field and their raised baseline of general activity might suggest an increased drive state. Evidence against this view is the finding that the amount of running in an activity wheel, typically assumed to reflect drive (Strong 1957), is virtually unchanged or actually decreased in hippocampal rats (Douglas and Isaacson 1964, Strong and Jackson 1970, Campbell et al. 1971, but see Peters and Brunner 1976). Further, food or water deprivation usually affect the activity of hippocampal animals in a completely normal way (Gotsick 1969, Sengstake 1968).

(e) Increased general motor activity. It might be argued that the increased activity of hippocampal animals reflects a general motor system excitation which enhances the normal pattern of activity. The main argument against this view is the selective increase in certain behaviours as opposed to others that has been observed in most studies. Time sampling of the behaviour of animals in their home cages shows that hippocampals sleep less and sniff more during the night (Jarrard 1968, Kim et al. 1970, 1971a). In the Jarrard study this result was obtained 45 days after the animals were placed in the observation cages, indicating that the increased sniffing was unrelated to novelty.

As noted above, hippocampal rats spend much of their time in the open field running around the perimeter in a stereotyped fashion. On the other hand, they have been reported to rear significantly less than do normals in this situation (Jackson 1967), as well as in an enclosed box (Clark 1970). Similarly, as we have seen, they rarely attempt to jump out of open fields.

The evidence, then, suggests that the hyperactivity of hippocampal animals does not reflect a general increase in all activities, but rather a shift in the probability of occurrence of certain behaviours. Many of those behaviours which are diminished reflect exploration or the operation of the cognitive-mapping system. These shifts appear related in some way to the specific environment to which the animal is exposed; this brings us to the last postulated cause of hyperactivity in hippocampal animals, one noted already in discussing reactions to intense stimulation, the exaggerated reaction to external stimulation.

(f) Increased reactivity to stimuli. Kaplan (1968) thought that the hyperactivity of hippocampals might be due to a hyper-reactivity to stimuli, since there were indications in the literature that the difference in activity between normals and hippocampals increases with the size and complexity of the environment.

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* We discuss later several studies of hoarding behaviour in rats which suggest that hippocampal animals are not affected by the insecurity inherent in a novel environment (pp. 353-4). These studies, in conjunction with that of Jarrard (1968), indicate that hippocampal animals lack the normal fear of unknown places. See our discussion of fear (pp. 304-6).

He showed that the same group of hippocampals were hyperactive in a large, well-lit box with holes, but not in a small, dark jiggle-box (a box with a sprung floor which can be used to register movement). This suggests that activity in hippocampal animals is related to the extent of external stimulation of any sort, and is thus general activity rather than exploration.

We have come to the same conclusion on the basis of our own observations of fornical and hippocampal rats in various novel environments. Two of our observations are worth mentioning in this context as they shed some light on the mechanisms controlling the behaviour of hippocampal animals. The first, of one of our hippocampals on a novel elevated plus maze, underlines the sensitization to stimuli that can sometimes occur with repeated responses and calls to mind our discussion of the self-generated depression model of habituation and its postulation of incremental, rather than decremental, effects under certain conditions. The plus-maze was in constant use and had a number of fecal boli strewn about it. When first placed on the maze the rat sat for about a minute and then began to move off. As it did so it sniffed at a fresh bolus left by the preceding animal. Subsequently, its activity steadily increased as it ran back and forth from arm to arm, often visiting only two of the four arms for several minutes. During this period it never passed that bolus without pausing to sniff at it. Yet at the same time it ignored all the other boli on the maze. After 10 minutes or so the rat began to sniff at another bolus in the same arm. After that, with each passing minute, more and more boli were sniffed until the animal was spending most of its time running from bolus to bolus, devoting one or two sniffs to each. Thirty minutes after the rat had first been placed on the maze this behaviour continued unabated.

This observation demonstrates the complete absence of anything remotely comparable to exploration in hippocampal animals, their high-activity level consisting almost entirely in repetitively stereotyped behaviours; these are best described as microstereotypies, for the form of the behaviour can be remarkably constant. We have noted that behaviours related exclusively to the taxon systems, such as these stereotypies, should depend exclusively on such variables as frequency and constancy of stimulation for their habituation. High stimulation rates and constant stimuli favour decrements in response, while low rates and variation in the eliciting stimulus could yield incremental effects instead. If the behaviour of the hippocampal animal is due to processes such as these we would expect the incremental effects just described. Further, we would expect a rapid decrease in responsiveness with increased consistency and frequency of stimulation. Our second observation, that of hippocampals in a small glass jar, is pertinent here. One animal in this situation remained motionless for 2 min and then developed a stereotyped sniff-rear pattern which was repeated at the rate of once every 10 s or so. However, this pattern was almost never repeated twice in the same part of the jar. Typically, the animal sniffed-reared-sniffed in one place and then moved a certain minimal distance before repeating the sequence; sometimes the second sniff was followed by a partial rear in the same place. This pattern suggests that each sniff and rear cycle has two effects: it increased the likelihood of further sniffs and rears, but decreased the probability that the particular stimuli immediately available would trigger such behaviour.

These observations, and the mechanisms we have postulated to explain them, help to account for many of the behaviours of the hippocampal animal. In large open boxes more stimuli are available and consequently activity levels will be higher than in small boxes. Further, and most important for the hippocampal animal, the time between successive exposures to the same stimuli increases in large boxes. In smaller boxes the rate of repetition of the same stimuli could 'allow' the hippocampal animal to habituate as rapidly as the normal animal, though these two types of animal appear to be habituating in markedly different ways. This analysis also explains why there should be no difference in activity wheel behaviour between normals and hippocampals; animals in this situation are repeatedly and rapidly exposed to the same stimuli. Thus, the peculiar features of the behaviour of hippocampal animals in novel situations can be ascribed to two main factors: first, the absence of exploration is due to the lack of a cognitive-mapping system; second, the stereotyped behaviour patterns that are seen, and the importance of such variables as frequency and consistency of stimulation, are due to the remaining influence of the taxon systems. We see later, in our discussion of extinction, another example of how important these characteristics of the taxon system are in determining the behaviour of the hippocampal animal.

Before moving on to discuss spontaneous alternation behaviour there remains the problem of explaining the curious fact that studies employing an ultrasonic activity-measuring device (Boitano and Isaacson 1967, Boitano et al. 1968, Jarrard and Korn 1969) have failed to report hyperactivity in hippocampal rats. It is known that the rat's hearing extends at least as high as the frequency used in these devices (40 KHz, Gould and Morgan 1942), and, in fact, is probably maximally sensitive in that very range (Gourevitch and Hack 1966). We therefore decided to see if there were any differences in the responses of normal and hippocampal rats to this sound. Three normals and three hippocampals were tested in an elevated box with the transmitting and receiving heads of the ultrasonic device located about a metre away. None of the normals paid much attention to the onset of the sound beyond a slight ear flick. Two of the three hippocampals immediately ran to the side of the box closest to the source of the sound and spent a considerable portion of their time leaning out over the edge towards the source. The third did not orient towards the sound in such an obvious way but instead became extremely agitated. He, and one of the other hippocampals, developed audiogenic seizures at 8 and 6 min after the sound onset, respectively, and remained for the rest of the observation period huddled in post-ictal depression. It appears, then, that the sound emitted by these devices is both audible and strong for the rat, that the normal rat rapidly habituates to it, but that the hippocampal rat, as is the case with other strong stimuli, suffers incremental effects, leading in some cases to increased running and possibly seizures. Although it is not possible to demonstrate that similar effects were produced in the three studies under discussion we would caution against the use of these devices, particularly in the study of brain-damaged animals.

6.3.3. SPONTANEOUS ALTERNATION

When a sated rat is given two consecutive trials in a two-choice apparatus, such as a T-maze, it is highly likely that its second choice will differ from the first. This phenomenon, termed spontaneous alternation, tells us basically two things about an animal. First, some trace of the first choice and its consequences is available to the animal later, and, second, the animal is motivated to change its behaviour on the second trial, relative to some dimension of the situation. It is clear that normal animals rarely alternate responses (in the sense of left or right turns) *per se,* but rather alternate the place to which they go, or the particular stimuli they approach (e.g. Walker *et al.* 1955). Alternation tends to increase as the animals are confined for longer periods in the initially chosen goal area, and it has been suggested that something akin to stimulus satiation accounts for this effect (Glanzer 1953b). Placing the animal in one of the goal boxes for a period of time causes the animal to choose the arm leading to the other goal box on a subsequent trial (Glanzer 1953a). The strength of this alternation tendency is demonstrated by the fact that after 10 forced trials to one choice arm rewarded by food the hungry animal will alternate to the nonfood side when given a free choice (Sutherland 1957).

Alternation behaviour can be viewed most simply as reflecting a tendency on the part of the animal to acquire information about unknown parts of its environment, and thus as exploratory behaviour; the longer the animal is allowed to explore one part of the environment the less attraction this part holds relative to other, unknown, areas. Strong support for this information-acquisition view of spontaneous alternation was provided by Sutherland (1957). He showed that alternation will occur when the two arms of a T-maze lead to different goal boxes, but will be decreased when the arms are extended to lead to a common goal box. This shows that stimuli which are not available at the choice point can determine alternation behaviour; the rat will vary its choices in so far as such variation brings it into contact with new places or stimuli.

The hippocampal animal approaches the alternation situation on somewhat different terms. According to the present model, it is bereft of cognitive maps and any tendency to explore novelty. It would thus be predicted that these animals would not alternate. Table A 15 lists the results of those studies concerned with the behaviour of hippocampal animals in spontaneous alternation situations. The general picture is that these animals do not alternate.

6.3.3(a). The hippocampus and spontaneous alternation. The first time an animal is confronted with a choice its particular response is assumed to be random. This is yet another statistical fiction. The first choice of an animal reflects a number of factors, motivational, emotional, and other, and can be taken as a reliable indicator of the hierarchy of response alternatives in that animal's repertoire at that particular moment. When confronted with the same situation shortly thereafter, assuming no change whatever (including no effect of the first choice), one must predict that the choice would be the same, reflecting the action of the same factors that led to the initial choice. Of course, as we have just seen, the first choice biases the second in determinable ways. In the normal animal the action of the locale system is assumed to produce a strong tendency to alternate place. In the hippocampal animal, on the other hand, response factors seem to be the main ones that bias second choices, and they bias these animals towards repeating their first choice.

The studies listed in Table A15 provide some support for this response-repetition view of the behaviour of hippocampal animals in spontaneous alternation situations. Nevertheless, we should point out that there is a serious methodological flaw in all but four of these studies (Dalland 1970, 1976, Stevens 1973a, Stevens and Cowey 1973). Alternation tests in the remainder of the studies were applied successively to the same animal, varying from one test per day to five tests per day (that is, six trials in all): Underlying this methodology is the assumption that, in the normal rat, all traces of previous experience in the maze are gone within some short period of time. This, however, is strongly contradicted by the study of Blanchard, Shelton, and Blanchard (1970b), which showed that the memory for a single experience with non-reward objects can last up to nine days. Optimally, animals should be tested for alternation only once, as in the studies noted above.

While many of the studies support the view that hippocampal animals fail to alternate in the spontaneous alternation situation, there are some exceptions. Two factors seem to be important: (1) intertrial interval; (2) confinement in the goal arm chosen on the first trial. According to Roberts, Dember, and Brodwick (1962) and Kirkby *et al.* (1967) hippocampal rats repeat responses after 8-10 min inter-trial interval. However, 50 min inter-trial interval can lead to other behaviour. If this time is spent in the goalbox then alternation is seen on the next trial (Kirkby *et al.* 1967, Stevens 1973, Stevens and Cowey 1973). If the time is spent in a waiting cage outside the apparatus, random choices ensue (Stevens 1973a). These data suggest that satiation to the goal-arm stimuli can lead to alternation in lesioned rats. However, the Stevens and Cowey data confuse the issue somewhat. Rats with dorsal hippocampal lesions respond randomly with 50s inter-trial interval and repeat responses with a 50 min interval spent in a waiting box. On the other hand, rats with ventral hippocampal lesions repeat responses after 50s, but alternate after 50 min in the waiting box. These data stand in opposition to the results of the Dalland (1970) and Stevens 1973a) studies, though there were lesion site differences, of course. We have no explanation for these discrepancies.

In addition to these conflicting data there are two studies using multiple tests which produced alternation (Gross, Black, and Chorover 1968, Ellen and DeLoache 1968). The first study produced alternation with short inter-trial intervals but not long ones; it is possible that very short inter-trial intervals will produce response alternation due to a short lived inhibition in the response system. The second study produced alternation in the lesioned animals to the brightness of the goal arm but not to spatial direction. These exceptions to the general picture of absent alternation in hippocampals suggest that there are taxon mechanisms available for shifting an animal's responses and that they are sensitive to such things as stimulus satiation and repetition rate. This agrees, of course, with what we have already seen in discussing habituation, and we shall see that it is important in understanding the behaviour of hippocampals in a wide variety of situations. We shall consider the mechanisms underlying such shifts in behaviour in hippocampal animals in detail later (pp. 337-48). The bulk of the data under discussion here indicate that spontaneous alternation is driven primarily by the locale system and that it is absent in hippocampal animals, except with very short inter-trial intervals or very long confinement in the goal box.

Dalland's study provides the clearest picture. She showed that her lesioned rats were specifically repeating responses, and not approaches to specific stimuli, by rotating the start arm 180° on the second trial. In a recent report (Dalland 1976a) this response repetition finding was replicated and extended to show that it occurred regardless of the place or stimuli to which the response led. However, in a separate experiment she found that preventing lesioned rats from entering one goal arm (by a nylon mesh door) led to the choice of that arm on a subsequent free-choice trial. A control study showed that this result was not due to the novelty of a door being removed on the second trial; the mere removal of a hurdle from a normal two-choice T-maze had no effect on the responses of hippocampal rats. These data, for which we have no simple explanation, were interpreted by Dalland to suggest that one of the variables controlling the repetition of responses is the voluntary nature of the first choice.

Gaffan (1972) has shown the hippocampal deficit in spontaneous

alternation using a design which rules out response factors. On the first exposure to a T-maze rats were allowed to move about only in the start arm, though they could see both goal arms through clear plastic doors. One of these arms was white, the other black, on this trial. On the second trial the plastic doors were removed and the colour of one of the arms changed so that both arms were the same. This second trial was given under one of two conditions: either the same as the first trial or with the rats made fearful by the sounding of a buzzer just prior to the trial. Normal animals chose the arm which had been changed when tested under nonfear conditions and the arm which had not been changed when tested under fear conditions. Hippocampal animals chose randomly on both conditions. This study clearly shows that the normals, but not the hippocampals, are choosing on the basis of information about stimuli in particular locations and not on the basis of previous responses or the amount of exposure to the stimuli in the situation.

It appears then, that the normal animal tends not to repeat its responses over two choices when such repetition involves the simultaneous repetition of location and/or stimuli. On the other hand, the hippocampal animal tends to repeat its responses whether or not this entails going to the same place, or to go randomly. Only very short inter-trial intervals or confinement to the initially chosen goal area can alter this picture. These data support the view that hippocampal animals lack those mechanisms driving exploratory behaviour in normal animals.*

6.3.4. LATENT LEARNING

In the typical latent-learning situation an animal is put in a novel environment and allowed to explore. The animal is neither hungry nor thirsty, and the location of food or water within this environment is not the source of motivation for the animal's exploration. Nevertheless, if subsequently made hungry or thirsty, the animal will quickly learn to go to the proper location to find its reward. This type of learning, which depends upon exploration, has been termed *incidental,* in that it does not appear to be a direct result of the animal's interests or motivations. Within the framework of the present model latent learning is neither incidental nor unmotivated. It results from the action of the cognitive-mapping system and leads to the development of maps of experienced environments. Thus, we would predict that hippocampal animals would not show latent learning. Two studies of latent learning in hippocampal rats have been reported, neither of which found dramatic effects in their normal subjects. Means (1969b) reported some latent learning in his hippocampal rats but the data are unconvincing.

* The data on spontaneous alternation provide a good example of the problems associated with the use of combined cortical-hippocampal lesions. In a few of these studies the cortically lesioned controls failed to alternate properly (Means, Woodruff, and Isaacson 1972, Stevens 1973a). The interpretation of a deficit in the experimental lesion group is impossible with such control data.

Kimble and Greene (1968) report no latent learning in their hippocampal rats and conclude that these animals may have 'built a poorer "cognitive map"' (p. 100), but these data are also not very convincing. A definite conclusion concerning latent learning in hippocampal animals awaits a proper experiment, one in which normal animals show a clear-cut effect.

6.3.5. CONCLUDING COMMENTS ON EXPLORATION

We have observed that the behaviour of hippocampal animals in response to novelty differs from that of normal animals in several ways. First, hippocampal animals betray no knowledge of the unfamiliarity of a new place, either in terms of curiosity or fear. Second, they explore novel items less than do normals. Third, their hyperactivity in novel, or other, environments results from their lack of fear, their tendency to engage in repetitive, stereotyped behaviours, and their hyper-reactivity to stimuli. Fourth, they fail to alternate choices in most spontaneous alternation situations.

The distinction between exploration and general activity is a particularly important one in the context of hippocampal lesion effects. Activity seen in hippocampal animals would not appear to be exploration. It does not decrement with continued exposure to a novel situation in any way specific to that situation, at least when the situation provides adequate variation. The absence of this important defining characteristic of exploration is seen clearly in a study by Kamback (1967). Rats were given the opportunity to press either of two levers in an operant chamber, one providing light, the other nothing. Both normal and hippocampal rats preferred to press the light-on lever. In the normal animals this preference declined over time, suggesting that it was based to some extent on curiosity and exploration. In the hippocampal animals the preference did not decline over time. As Halliday (1968) pointed out, light-contingent lever pressing can only be considered as exploratory when it declines over time. The absence of such a decline in hippocampals is consistent with the view that in these animals the preference was based on the greater stimulation the response provided, and was not based on an exploratory tendency.

7

Discrimination and maze learning

7.1. Discrimination-Background

IN the literal sense discrimination refers to the capacity of the organism to detect differences. Traditionally, discrimination studies have been concerned with an animal's ability to respond differentially to items or places in the environment. In some studies the interest lies in the limits of the sensory/perceptual systems, as in the determination of thresholds, or acuity. More often the psychologist's emphasis is on the learning based on the ability to form discriminations: how animals come to identify the relevant aspects of an environment, and how they choose adaptive behaviours.

The logic of discrimination learning usually stresses some association between reward and one value of a variable dimension, e.g. the brighter panel, the leftmost door, and so on. Most such studies utilize items rather than places, for reasons to be discussed shortly. None the less, this type of learning occurs within a spatio-temporal context, and the role played by this context has proved of some interest. The importance of temporal contiguity between the relevant discrimination response and the reward has formed a large part of experimental psychology in recent decades, though work with taste-aversion learning (cf. Kalat and Rozin 1973) suggests that such contiguity is not always essential to learning. On the other hand, the importance of spatial contiguity between the discriminandum and the site of response and/or reward, though well documented, has neither been thoroughly explored nor explained. Munn (1950, p. 115 ff.) has described how, early in animal experimental work, apparatus was designed to circumvent the difficulties introduced by spatial discontiguity. Thus, the Lashley jumping stand and the Yerkes discrimination box forced animals to respond directly to the discriminanda. Now, it is standard procedure to 'shape' animals to respond at the appropriate site before discrimination training begins.

These techniques enable the experimenter to focus directly on the discrimination process but prevent any examination of the role of exploratory place learning. An animal introduced for the first time into a discrimination situation will respond as it does in any other new environment, with exploration and the construction of cognitive maps. The elimination of this place learning from the concern of the experiment does not mean that it fails to occur; considerable recent work attests to the importance animals attach to locations. However, this has not been reflected in theories of discrimination learning to date (cf. Mackintosh 1974).

The debilitating effects of cue-response spatial discontiguity and the similar, though lesser, effects of cue-reward discontiguity were documented by Miller and Murphy (1964) and are discussed at length by Cowey (1968). The basic problem introduced by such discontiguities is clear; animals are faced with a conflict in deciding *where* to focus their attention. The site of the cue, the response, and the reward are all important; to which should the animal attend?

In some discriminations strong approach behaviour can be seen to develop once the animal has identified the relevant cues. For example, in a study by Grastyan and Vereczkei (1974) cats were trained to approach food in one part of a straight alley or T-maze. Subsequently, a signal (buzzer) was introduced, correlated with the availability of reward, either in the start area or in the 'incorrect' arm of the T-maze. Animals began to orient to the source of the signal rather than to the reward site, leading ultimately to a complete breakdown in appropriate goal-directed behaviour. Here, the conflict between a cue and a place led to maladaptive behaviour; as the authors point out most learning situations involve the superposition of relevant cues and places, and conflicts such as the above are rarely seen. *Sign tracking* or *auto-shaping* (e.g. Hearst and Jenkins 1974) offers an example of the synergistic action of place and guidance hypotheses. A number of different behavioural paradigms fall into this category, sharing a common feature; animals respond to an item correlated with reward (or avoid one correlated with punishment) even though these responses are not necessary in obtaining reward (or avoiding punishment). Indeed, after a certain amount of training such responding survives punishment, either by direct shock or by the indirect loss of rewards as in Grastyan and Vereczkei's study.

It is clear from these data that discrimination situations elicit strong hypothesis behaviour, and that animals are quite sensitive to conflicts between relevant places and guidances. Any understanding of the effects of hippocampal lesions upon discrimination learning demands a careful analysis of the types of hypotheses used by intact animals.

7.1.1. HYPOTHESIS BEHAVIOUR IN DISCRIMINATION LEARNING

We have already described a variety of hypotheses that animals can use to solve problems. Place hypotheses were postulated to involve the functioning of different neural systems than those involved in taxon hypotheses, and this difference held to underlie the divergent behavioural properties of these classes of hypotheses. The foregoing description of place learning in discrimination situations suggests that place hypotheses might be unusually important, and the particular properties of the locale system thus become critical to an understanding of discrimination learning. Evidence from a variety of studies indicates that place hypotheses are used preferentially if the situation allows. Thus, in a T-maze place learning takes precedence if sufficient information is available to sustain it. This is particularly important at the onset of training; subsequent to this it appears that animals learn to respond on the basis of other information. That is, after reaching a learning criterion and with continued training intact animals seem to make increased use of a taxon hypothesis (typically an orientation hypothesis), even when they had previously been using a place hypothesis (Hicks 1964, Mackintosh 1965).

This tendency to utilize response routines with well-learned habits leads to stereotyped behaviour patterns which are often quite difficult to alter; this is related to the property of persistence already described for the use of taxon hypotheses, especially those involving the repetition of exactly the same responses. A similar effect is produced by insoluble or highly stressful situations (Maier 1949). Here, behaviour becomes quite stereotyped, even in the face of non-reward. It is noteworthy that once such fixations appear they often cannot be broken by the subsequent introduction of rewards contingent upon other responses. This lack of variability in behaviour is not limited to the molar level of the animal's choice behaviour. Maier points out that 'even the manner of jumping and types of escape behaviour become highly specific' (p. 30). This extreme rigidity in behaviour, based on the repeated use of particular responses, is a regular feature of hippocampal behaviour in general and normal behaviour in those situations completely eliminating the participation of the locale system.

The normal animal, in most situations, does not easily fall into fixated response routines. The hippocampal animal, on the other hand, manifests such routines quite often. In the absence of any distinct external item, and lacking a locale system, the hippocampal animal must utilize an orientation hypothesis to solve spatial tasks in particular and other tasks where spatial factors play a disguised role. This tendency manifests itself in a variety of experiments, and could provide the basis for an understanding of 'response perseveration' in these animals; we discuss this possibility later (p. 340).

These observations suggest that one of the primary aspects of discrimination learning concerns an appreciation of the relevant *places* in the situation. The normal animal, we assume, initially learns *where,* and not necessarily to *what,* to respond. This is an important distinction within the framework of our model. Learning about places is a function of the hippocampal cognitive-mapping system, and the animal without a hippocampus would be expected to approach a discrimination problem in a fundamentally abnormal way.

In the absence of a system directing attention to places, the hippocampal animal is forced to attend to items or to associate rewards with particular responses. A brief look at several studies demonstrates this point:

(1) Stevens and Cowey (1972) tested rats on a lever-press alternation task; the rats were provided with a light cue telling them which of two levers to press on any given trial. In one condition there was cue-response contiguity; that is, the light cues were mounted within the manipulanda (panels). Both hippocampal and normal rats solved this task easily. In a second condition the light cues were spatially separate from the levers. Hippocampals solved this task at about the same rate as in the first condition; normals, on the other hand, took significantly longer to learn. This suggests that, for the hippocampal rats, the locations of cue and response site, and their spatial relation, was irrelevant. Presumably the normal animals, for whom the spatial relation between cue and response was important, were faced with a conflict; attention could be directed to the place containing either the cue or the response object. In order to solve this relatively simple problem the normal animal would have to ignore places.

(2) Gambarian et al. (1972) tested hippocampal cats on several simple conditioned reactions. In order to obtain food the cat had to approach and press a lever upon presentation of a non-directional conditioned stimulus. When two levers were available at opposite ends of the testing box, and a different conditioned stimulus used to signal food for each lever, hippocampal cats performed roughly at chance level. That is, though they reacted appropriately to a conditioned stimulus by approaching and pressing a lever, they failed consistently to approach the lever in the correct place.

(3) In an experiment by Karmos and Grastyan (1962), cats were placed in a box containing a food well on a ledge and a speaker mounted beneath it. A series of clicks, delivered through the speaker, signalled the availability of food. The authors describe the behaviour which persisted in their hippocampal cats as follows:

> 'if the conditional stimulus was delivered at a time when the food tray just happened to be in the visual field of the animal, a quick reaction as in a normal animal resulted. When at the time of delivery of the conditional stimulus the animal was sitting with its back to the food tray, long-lasting, searching, sniffing orienting movements ensued. The special conditional act only appeared when the animal caught sight of the feeding device during this searching behaviour elicited by the conditional stimulus' (p. 219).

(4) Finally Molnar (1973) trained cats on a two-phase conditioned reaction which had both place and cue components. The testing box was rectangular with three identical feeders, one on each of three walls. A small cage with a pedal was attached to the fourth wall. Food was always delivered to the feeder opposite the small cage and never to the other feeders. Non-directional auditory cues came from a loudspeaker on the top of the box, directional cues from a loudspeaker over the correct feeder. On each trial, the animals were required to sit quietly in the centre of the box (place) and to run to the small cage and press the pedal when a non-directional stimulus was sounded from the overhead speaker (place?). The pedal press delivered food to the feeder, and initiated one of three conditions: either the sound went off and was not replaced, or it was replaced by a different non-directional sound from the same overhead speaker, or it was replaced by a directional sound from above the correct feeder. Only in this last of the three conditions could the second sound act as a guidance. The first two conditions required place hypotheses for solution. The cats were trained on all phases of the task before hippocampal lesions were made. After the lesion, they failed on all place components but were as good, if not better, on the cue-guided component. Instead of sitting quietly at the beginning of the trial they paced about the box incessantly. The first sound no longer produced a directed response to the cage but instead the cats continued to wander and search in the manner described by Karmos and Grastyan above. If a cat chanced upon the pedal in the cage and initiated the second phase there was an interesting difference in its behaviour depending on which of the three conditions obtained. If the directional sound came on, the animal ran to the correct feeder, in some cases with a shorter latency than before the lesion. In either of the non-directional conditions, however, the cat continued its hyperactive wandering which included visits to the two nonrewarded feeders.

These examples show quite clearly the difficulty that hippocampal animals have in the simple problem of knowing *where* to respond. No difficulty is apparent in responding to an item or cue, however, as shown by their immediate reaction upon sighting the food tray or hearing the directional sound.

The hypothesized absence of this fundamental place learning in hippocampal animals should have profound effects on all their discrimination behaviour. Even the simplest of tasks might be solved in an abnormal way. Of course, discrimination problems favouring the use of place hypotheses should be quite difficult for hippocampal animals. They must adopt an alternative hypothesis in order to solve such tasks, if possible.

7.2. The effect of hippocampal lesions on discrimination

7.2.1 SIMULTANEOUS DISCRIMINATION

The most commonly used discrimination task, and the one to which much of the foregoing discussion applies most directly, involves the simultaneous presentation of two choices, with the animal being rewarded for responding to one but not the other. Mazes of various types, as well as operant chambers, can be used, and discrimination might be required between places, or any perceivable dimension of items.

Such discrimination tasks can be readily split up into two categories, depending upon whether or not they involve a spatial variable; that is, involve the consistent association or reward with a location in either egocentric or non-egocentric space. Table A16 presents the results of those studies concerned with simultaneous spatial discrimination, while Table A17 presents the results of the remainder of the simultaneous discrimination studies. Aside from these two categories one could mention those few spatial studies which strongly biased animals towards the use of place hypotheses; that is, reward was associated most simply with a location in locale, or non-egocentric, space. We shall discuss these studies separately.

It is clear from Tables A16 and A17 that no obvious generalized deficit is seen in hippocampal animals in most types of simultaneous discrimination learning. However, deficits are sometimes reported, and even in the absence of a changed rate of learning there are indications that hippocampal and normal animals solve such problems in different ways. A close analysis of these sometimes subtle differences offers strong evidence for the assertion that hippocampal animals lack the ability to utilize place hypotheses. However, before considering these studies we shall discuss in some detail those few experiments which directly tested locale function.

7.2.1(a). *Spatial discrimination-locale dependent.* There are few studies in the literature which tested the ability of hippocampal rats to learn discriminations requiring place hypotheses. This is, no doubt, due to the difficulty of devising such a test; locations are often defined in terms of the stimuli at or near them, and a guidance hypothesis based on approaching (or avoiding) one of these stimuli is almost always available to the animal.* Seven studies, two of them from our laboratory, did severely bias the situation towards the use of place hypotheses and against the use of taxon hypotheses.

Rabe and Haddad (1969b) placed thirsty rats on one table of a Maier three-table apparatus** and allowed them to drink water for a short time. The animals were then removed from the rewarded table and put on another one; their task was simply to return to the table where they had just received water. The position of reward varied from trial to trial, as did the subsequent placement of the rat, so that proper performance depended upon remembering where the water was on any given trial. Although a particular cue might be associated with reward during the course of the rat's initial exposure to the water, it is much more likely that the normal rat would utilize its cognitive map of the situation and, by making a slight change in the map on each trial, obtain reward. It is not surprising that only 6 of 21 hippocampal rats reached the minimum criterion necessary for showing better than chance performance in this study.

Olton (1972a) tested rats on a task similar in several ways to that used by Rabe and Haddad. Thirsty rats were given a discrimination task in a plus-maze, with considerable extra-maze information available. A single discrimination problem consisted of five trials. On the first trial the rat was forced to go from the start arm (which remained the same on all problems) to one of the other three arms; on the second trial the animal was forced to go to a different arm. One of these forced choices led to water; the other did not. Thus, after these two trials the rat had information about two of the choice arms. On the remaining three trials free choices were allowed. Each new problem provided different information about the location of water or no water. All rats were tested extensively pre-operatively and performed much as one would expect; on the initial free-choice trial they approached the water arm most often, the no-information arm sometimes, and the no-water arm rarely. On the third free-choice trial they almost always chose the water arm. After receiving 48 problems all rats were subjected to sham operations and given an additional 12 problems; these operations did not affect their performance. Following this, all rats received bilateral hippocampal lesions and, after recovery, an additional 48 problems. Hippocampal damage had a marked effect on performance. Basically, the lesioned rats chose randomly on the initial free-choice trials and began to show a preference for the water arm by the final trials. This random choice on the initial trials indicates that the hippocampal rats could not rapidly learn about the location of water as they had been able to do pre-operatively. The learning that was seen over the free-choice trials presumably reflects learning the correct turn on the given problem. It is clear, as in Rabe and Haddad's study, that hippocampal animals cannot utilize information about the location of water in determining subsequent behaviour under free-choice conditions.*

Plunkett, Faulds, and Albino (1973) compared hippocampal and normal rats in two tasks using a plus-maze. In one case the animals had to learn

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* Pohl (1973), for example, devised a 'landmark' discrimination task which he assumed measured allocentric, or extrapersonal, space. The correct food well in this Wisconsin General Testing Apparatus (WGTA) task was marked by an adjacent item. It is clear that this task is not entirely locale dependent and could be solved by responding to the well near the landmark.

** This is basically a Y-maze with platforms at the end of each arm.

* As Olton (1972a) points out, his results are incompatible with the Douglas-Pribram (1966) hypothesis that the hippocampus is solely involved in error evaluation.

to make a particular turning response, regardless of where they started; in the other case they had to go to the same place. Hippocampal rats learned the response task as rapidly as did normal rats, but had a marked deficit on the place task. A total defect was not seen in this study, perhaps because, as the authors point out, 'place learning and response learning are not isolated in the two tasks' (p. 80). This result has been replicated by de Castro (1974).

In the first of our studies (Riches 1972) thirsty rats were required to go to the same arm of an elevated Y-maze when started from either of the other two arms; the starting arm was varied randomly from trial to trial. Normal rats learned this task quite rapidly; the median number of trials needed to reach a 9/10 criterion for seven normal subjects was 14. As expected, the hippocampals were quite poor at this task. Of the nine rats in this group, two failed to reach criterion in 100 trials. The other seven had a median score of 52 trials to criterion. No hippocampal rat learned the task faster than any normal rat. Those lesioned rats which learned the task were given a series of probe trials in an attempt to determine which cues they were using. These probe trials consisted of trials run in the total darkness, or with distal visual cues at the start and/or choice obscured by black curtains. In pilot studies we had found that almost all animals initially learn this discrimination using visual information, and the probe trials were therefore limited to variations in this modality. Figure 31 portrays the probes and the order in which they were given to the rats. The essential findings were that the performance of both groups fell to chance in the total darkness, but that the type of visual information used by the two groups was different. All of the normal animals but one performed perfectly when either the start arm or the choice arms were blocked with curtains. Further, relatively few errors were made when the entire maze was surrounded by curtains, leaving only the differential lighting on the ceiling as possible location information. It seems likely from these data that the normal rats were not dependent on any single source of information, but rather could respond on the basis of virtually any aspect of the visual environment; that is, they had some kind of 'map' of the situation. On the other hand, the lesioned rats virtually all made mistakes under either the start-curtains or choice-curtains condition. Their performance under the all-curtain condition was no better than chance. This pattern suggests that each hippocampal rat had solved the problem by responding to a limited sample of cues, possibly only a single cue, and that they had no idea of the total arrangement of the situation. These conclusions were strengthened by further probe trials, luring which the animals were started from the goal arm itself with curtains around that arm on two trials and no curtains on a third. Under these conditions every normal animal turned around in the start (goal) arm at least twice, suggesting that they 'knew' that this was the place where reward was normally found (water was not available on these trials). Only four turn-arounds were recorded for the entire group of lesioned animals. They clearly had no idea that this was the place where reward was situated.

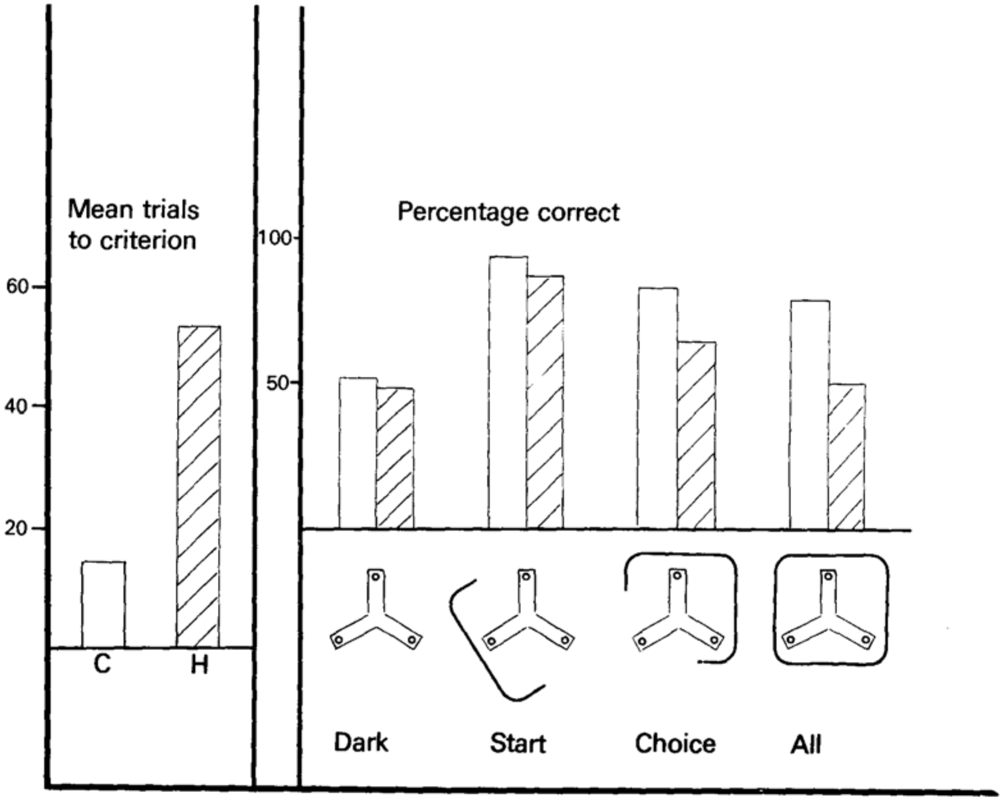

FIG. 31. Performance of controls (C) and hippocampal (H) rats on Y-maze task where the goal arm remained constant and the start varied randomly between the other two arms: left, median performance of the controls and hippocampals which learned the task; right, the performance of the two groups on different probe trials given after criterion was reached.

Our second experiment (O'Keefe *et al.* 1975) was run to preclude the objection that the lesioned rats failed on the Y-maze (as well as in the T-maze used by Plunkett *et al.* and de Castro) because of an inability to inhibit inappropriate body turns. Rats were tested on a circular runwav located in the centre of a room and there were many extra-maze cues available, though none of these were close to the runway. There were eight wells sunk into the runway, at equal spacings, and water could be delivered to any of these through polythene tubing and a hypodermic system from underneath the wells and runway. Two tasks were used, differing in the means by which the rats (with fornix or control lesions) could find the water. In one task water was always to be found in the well located in the same place in the room. In the second task the correct well was 'marked' by a small light. In the place task the light was present but irrelevant; in the light, or cue, task the correct well varied in location from trial to trial. Between trials the maze itself was rotated, the water wells changed, and the starting position varied. This had the effect of preventing the use of intra-maze cues, other than the light, or specific responses. During a preliminary training stage rats were allowed to find water associated with either the place or the cue. After this learning the formal testing stage began. Now, water was available only after the rat responded at the correct well. A response was defined as prolonged sniffing at the well and was registered automatically by an electric circuit incorporating metal plates on the floor of the maze, the rat, and the brass water well. Rats received water on every trial, even if they had responded at incorrect wells first. Criterion for learning was 9/10 trials during which a response was made only to the correct well. The results of this study are shown in Fig. 32; fornix-lesioned rats failed on the place task and were better than normal on the cue task.

As we argued in the paper reporting this study, the defect seen in the lesioned rats could not be attributed to changes in such things as motivation or general learning ability. We also argued that a response-inhibition defect could not account for these data. Rather, this study seems to show a highly selective failure of place learning.*

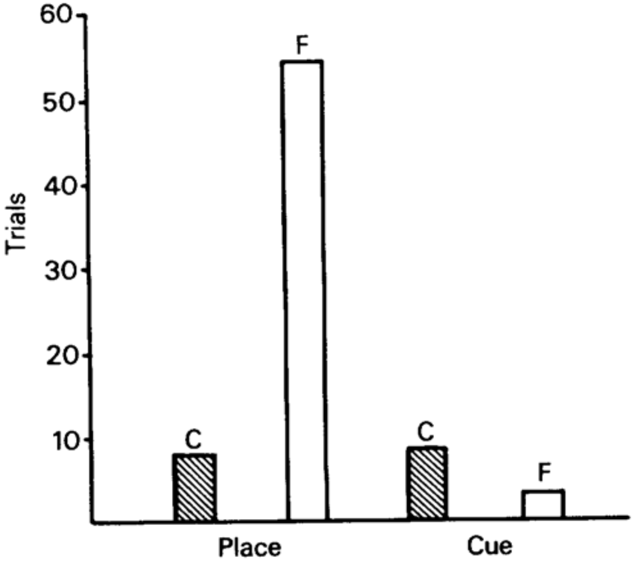

FIG. 32. Circle maze. Median trials to criterion of controls (C) and fornix-lesioned (F) rats on two water-finding tasks: left, results when the well where water could be obtained was always situated in a particular part of the room (place); right, results when the well was marked by a strong light (cue). (After O'Keefe *et al.* 1975.)

Recently Olton, Walker, and Gage (1977) have tested lesioned rats on their eight-arm spatial memory task (see p. 69). In this task the rats are allowed to retrieve food from each of eight arms of a radial maze in any order that they choose. Normals usually choose seven or eight baited arms before re-entering a previously visited arm. The choices appear to be on the basis of the spatial locations of the arms as defined by the extra-maze room cues. The performance of animals previously trained on this maze was reduced to chance levels by lesions in the hippocampus, entorhinal cortex, or fornix. Some of these animals fell into obvious orientation hypotheses such as successive 90° turns or running straight ahead. The choice patterns of others appeared to be random. In a subsequent study (Olton, personal communication) cue groups formally similar to the light cue groups in the O'Keefe *et al.* (1975) study described above were run. For these animals a drawbridge into each of the baited arms was open at the start of the trial and was closed by the animal as it crossed it into an arm. Thus a closed drawbridge provided a non-spatial cue as to which arms had been entered. As in the O'Keefe *et al.* study lesioned animals did not show any deficits in this condition.

These studies show the place-learning deficit in hippocampal animals quite clearly. A final study, which was not intended primarily as a discrimination study by its authors, points up the way in which place hypotheses may operate in tasks which have no obvious spatial component and can thereby influence hippocampal performance relative to normals. Douglas and Pribram (1969) tested monkeys on a task in which different numerals were projected onto panels; the monkeys had to press these in a certain order to obtain reward. First, one numeral (5) was presented in the lower right-hand panel of the 4 x 4 panel array (the DADTA apparatus). When the monkey depressed this panel the second numeral (0) was presented in the upper left-hand panel; a press on this panel was rewarded. Hippocampal monkeys seemed to learn this sequential task more rapidly than normal animals. Following learning, on a few trials within a session, a distractor stimulus (1) was presented in one of four locations simultaneous with the presentation of the second stimulus. This distractor initially affected the latency to respond to the correct stimulus to about the same extent in all the monkeys. However, hippocampal monkeys had a higher probability of actually responding to the distractor. While the normal (and amygdalectomized) monkeys habituated this increased latency effect, speeding up their correct responses on the distractor trials, the hippocampal monkeys continued

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* The published paper includes a thorough discussion of the notion that place learning in this situation was merely a form of distal cue learning, and concludes that this could not have been the case.

to be distracted, as measured by their prolonged latencies to respond to the correct stimulus. However, hippocampals, like the others, virtually ceased pressing the distractor panel. At this point the same distractor (1) was presented at a new location for four trials, and then new distractors (2, *4, 6, 8)* were each presented once at this new location. Both of these conditions increased the latencies of the normal and amygdalectomized monkeys again, while only the new distractors affected the hippocampals, who were virtually unaffected by the change in the location of the old distractor.

We think these results show that the hippocampals had learned the initial task in a way completely different from the other monkeys. These others appear to have learned the location of the relevant panels and could rapidly learn to ignore distracting stimuli appearing in the wrong place. Thus, they habituate both responses to the distractor and the increased latency effect. The same distractor, put in a new location, increases their latencies again. Hippocampal monkeys do not appear to have learned the location of the stimuli to which they must respond. Over the first 32 distractor trials they responded to the distractor almost as often as to the correct stimulus. This suggests that they had learned initially merely to press the second panel, which lit up wherever it was, and had then to learn a discrimination between the correct stimulus and the distractor when the two were presented simultaneously. The fact that they ceased responding to the distractor indicates that they learned this discrimination. However, the fact that their latencies were always longer on the distraction trials means that they *had to* attend to both stimuli in order to know which panel to press. In other words they could not discriminate between these two stimuli on the basis of their location, as could the other monkeys. Once having learned this discrimination, the lesioned monkeys would not be disturbed at finding the same distractor in a new location. A new distractor, however, would affect them, as this would require another discrimination. This lengthy analysis offers a particularly nice example of the different modes of learning in hippocampal and normal animals when measures of the rate of initial learning indicate that little or no difference exists. Experiments such as this, using simple learning situations followed by various probe trials, are the best means for bringing out these crucial differences.

7.2.1(6). Spatial discrimination—locale or taxon. In simultaneous spatial (or *position)* discriminations the animal is rewarded for consistently responding to the alternative in a particular part of egocentric space. Thus, the right arm of a T-maze, or the food-well on the left in an operant chamber, can be associated with reward. We have already noted that several hypotheses could be used to solve such tasks but that the normal animal uses a place hypothesis during initial learning if possible. Means (1969a) and Means and Douglas (1970) tested the usage of particular information by normal and hippocampal rats in this situation. Rats were trained on a T-maze spatial task and were then subjected to a set of cue-utilization probe trials.* In agreement with earlier work, normal rats used place hypotheses during initial learning, switching to others with continued training. Hippocampal rats, on the other hand, did not use place hypotheses at any stage of learning.

The dependence of the lesioned rats on taxon hypotheses could lie at the root of the difficulties they sometimes have with these simple spatial tasks. When a deficit is reported in these animals in simultaneous spatial discriminations it is usually a reflection of prolonged responding to the incorrect choice, that is, an inappropriate persistence on an orientation hypothesis. For example, the deficit reported by Means *et al.* (1972) for hippocampal rats was a direct consequence of the fact that

> 'individual animals would make one or more long sequences of incorrect responses. The effect was all-or-none. Either the animal made a long sequence of errors (10 or more) or it did not. The frequency of response sequences of intermittent length is no different than that found in other groups of animals' (p. 462).

Those hippocampal animals not producing such long sequences learned the problem at a normal rate.

This result suggests that spatial discriminations which bias animals towards incorrect orientation hypotheses could routinely elicit deficits in hippocampal animals. Thus, in a typical spatial task animals have an initial preference for one of the two choices; if lesioned animals are trained against this preference a deficit results (e.g. Samuels 1972). On the other hand, training the animal towards its preference might, in the absence of any tendency to explore the other arm, lead to better than normal performance in the hippocampal animal. When initial preferences are not taken into account a mixture of these could be expected, such that the group scores would be the same for lesioned and normal animals. Of course, there are other factors determining the likelihood of an orientation hypothesis being used, as distinct from a guidance hypothesis. The topography of the situation, for instance, will affect the ease with which orientation sequences are utilized; thus, the presence of barriers or obstructions biases against the chaining of response sequences. Additionally, the use of correction or non-correction techniques is important. The correction technique involves allowing the animal to respond appropriately on every trial; the non-correction technique involves terminating a trial

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* This procedure involved the use of a plus-maze. By starting the animal from different sides and by interchanging the stimulus patterns it was possible to put into opposition the use of place, guidance, and orientation hypotheses. Several such trials enable one to define fairly accurately which of these an animal is using.

upon commission of an error. It is clear that correction militates against response chaining, while non-correction can foster it (cf. Sutherland and Mackintosh 1971, pp. 26-7).

If we consider the effect of correction *versus* non-correction we note that in all but 1 of the studies listed in Table A16 using correction no deficit was reported for hippocampal animals. Thus, the technique which limits the use of persistent orientation hypotheses tends to prevent a deficit in the lesioned animals. On the other hand, not all studies using non-correction techniques report deficits. Within this category the question of initial preferences seems to be crucial. In the two studies where rats were trained by non-correction techniques against their initial preference deficits were reported (Samuels 1972, Means *et al.* 1972); we have already noted that in the latter study the deficit was directly related to the adoption of persistent orientation hypotheses in some of the lesioned rats. In the remaining studies on rats no account was taken of initial preference and learning typically appeared to proceed at a normal rate. However, in two studies, one of them on rats and the other on monkeys, the data suggest that this normality might be an artefact of group statistics. In the Means and Douglas (1970) study one subgroup of hippocampal rats was impaired; the authors point out that by chance there was a preponderance of rats trained against their preference in this subgroup. More tantalizing is the observation in the Mahut and Zola (1973) study that, of the six lesioned monkeys, three were significantly better than normal while three were significantly worse. As a group the lesioned animals did not differ from controls.

The results of spatial discrimination studies suggest the following conclusion: in those situations either allowing for or fostering the use of orientation hypotheses hippocampal animals will show a deficit when trained against their initial preference. This deficit results from the particular persistence of orientation hypotheses, as discussed earlier. The normal animal, in the same situation, uses a place hypothesis and thereby avoids persistent choice of the wrong arm.

The importance of orientation hypotheses, with their attendant rigidity, in hippocampal animals is seen in several situations related to spatial discrimination. We shall discuss a number of these in subsequent sections. Three examples are worth mentioning here.

(1) In a probability learning situation two discriminanda are associated with reward in a certain ratio, say 70:30. The normal animal in this situation tends to maximize his rewards by responding predominantly to the most-rewarded choice. Stevens (1973c) reported that hippocampal rats learn spatial probability tasks at a faster than normal rate; this could reflect the use of a persistent orientation hypothesis. More recently, however, Stevens and Cowey (1973) and Nonneman et al. (1974) have reported deficits in this task in hippocampal

animals. These latter studies investigated probability learning after the animals had been trained on position or alternation tasks in the same maze. Reward in the probability task was maximized to the side that was previously non-rewarded. Thus, these studies actually constituted a complex form of reversal learning, which almost inevitably produces strong deficits in animals with hippocampal lesions (see p. 282). In contrast to this, Stevens (1973c) assessed preferences in a simple choice test and trained his rats against this preference; this does not constitute a reversal problem. If the probability task is non-spatial the tendency to choose an orientation hypothesis could lead to slower maximization in the hippocampal animal (e.g. Douglas and Pribram 1966, study. 1).

(2) Hsiao and Isaacson (1971) tested rats on a Y-maze, with water being available in one goal arm and food in the other. On alternate days the animals were food or water deprived and had to make opposite responses to gain the appropriate reward. Hippocampal rats were retarded in learning this task, primarily because they made the same turning response on both food and water days.

(3) Bauer (1974) trained rats on a Y-maze discrimination of brightness; half of the rats in each group were pre-trained on the maze without the brightness cues. These rats fell into position habits during pre-training as they had no basis for predicting which arm would contain reward. This experience hindered subsequent learning of the brightness task, but most severely in the hippocampal rats. Kimble (1975) confirmed this result, though his control rats were not impaired by the pre-training.

7.2.1(c). Non-spatial discriminations. In non-spatial discriminations the relevant stimuli are typically spatially separate; in simultaneous discriminations they must be, while in non-simultaneous discriminations they need not be. We shall discuss some examples of this latter category later (pp. 331-5). In order to prevent the use of spatial information in the simultaneous task the stimuli are interchanged in some random sequence, such that spatial hypotheses (either place or orientation) would afford 50 per cent correct performance at best. None the less, it is clear that spatial information represents a powerful irrelevant cue in these situations and that the adoption of spatial hypotheses can constitute a source of considerable error in performance.* Our analysis of spatial tasks suggests that when forced to adopt inappropriate orientation hypotheses hippocampal animals will show deficits. On the basis of this one might expect

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* Irrelevant cues might constitute strong sources of error in spatial discriminations, but they are rarely used in such studies. Samuels (1972) has shown that such irrelevant cues are not as powerful in interrupting spatial tasks as in the reverse case, but more data are needed here.

that those non-spatial discriminations fostering the initial use of orientation hypotheses could similarly elicit deficits.

The data presented in Table A17 demonstrate that in a wide variety of situations hippocampal animals learn non-spatial discriminations at a seemingly normal rate. However, there are exceptions; further, in some of those studies reporting normal learning rates there are hints that hippocampal and normal animals are learning in different ways. In a study of brightness discrimination in a Y-maze (Kimble and Kimble 1970) data were presented on the use of hypotheses by the rats during learning; a hypothesis was defined as three or more successive choices to the same feature (bright, dark, left, right); with at least two of these involving an alternation along the non-chosen dimension. Although hippocampal rats, as a group, learned this task at a normal rate, there were differences between the groups in the use of particular hypotheses. The mean length of orientation hypotheses (left or right turns) was significantly greater in the lesioned rats than it was in the control rats.[*] [**]

An experiment by Olton (1972b) provides more direct evidence for the assertion that, in situations fostering the initial use of irrelevant position hypotheses, hippocampal animals might be impaired. Rats were trained on a pattern discrimination in a two-choice box. Both normal and hippocampal rats rapidly began to respond almost entirely to their preferred side, even though reward was correlated with the shifting patterned stimuli. Within four to five days both groups of rats began to show an increase in latency to respond when the negative patterned stimulus was presented on the preferred side, though still ultimately responding to that side. At this point the two groups diverged. Normal rats, within three days, broke their position habit and reached criterion on the visual task. All but 1 of the 10 lesioned rats failed to reach criterion within 13 days, continuing to respond almost entirely to their preferred side even though they took longer to respond to the negative stimulus on that side. These data attest to the difficulty hippocampal rats have in breaking orientation routines, even in the face of knowledge that the response is inappropriate. This is reminiscent of Maier's (1949 p. 41 ; see p. 267) description of normal rats forced into response routines. Upon introduction of punishment associated with the negative visual stimulus his rats began to show a resistance in jumping to that stimulus. Nevertheless, most of his rats failed to break out of their persistent habits, in the same way that Olton's hippocampal rats increased their latencies to the negative stimulus but could not switch their responses.[*]

Thus, the development of persistent response habits by hippocampal rats can, as in normal rats, impair the animals' ability to learn non-spatial tasks. What distinguishes the hippocampal animal in this regard is the relative ease with which such persistent habits are adopted. Of the remaining studies on non-spatial discrimination, those reporting deficits seem amenable to the interpretation that the hippocampal deficit was a function of either prolonged position hypotheses (e.g. Andy, Peeler, and Foshee 1967, Duncan and Duncan 1971) or other stereotyped behaviours interfering with proper discrimination (e.g. Woodruff and Isaacson 1972, Woodruff, Schneiderman, and Isaacson 1972). We return to a fuller discussion of persistent habits in hippocampal animals later (p. 337ff).

7.2.1(d). The reversal of simultaneous discrimination. When an animal has learned to discriminate between two stimuli, or places, so as consistently to choose the positive and avoid the negative, the situation can be altered so that the previously positive choice becomes negative and *vice versa.* This constitutes a discrimination *reversal* problem; it involves giving up a previously correct hypothesis and switching to a new one. Superficially, this switch might seem quite easy to accomplish, in that the same stimuli (or places) remain relevant in the reversal task. Thus, overtraining on the initial task often has the effect of facilitating reversal; this overtraining reversal effect (ORE) can be explained by assuming that with increased training the animal is more likely to choose a new hypothesis based on the same stimuli (cf. Sutherland and Mackintosh 1971, pp. 252 ff.). However, the ORE is rarely seen in spatial tasks; in fact, a number of studies have reported the opposite effect, prolonged reversal after overtraining on a spatial task (e.g. Krechevsky and Honzik 1932). As we have pointed out, most spatial tasks are initially solved by normal animals with place hypotheses but added training causes a shift towards the use of orientation hypotheses. Further, we have suggested that these are unusually prone to persistence. The absence of an ORE in many spatial tasks might be attributable to this shift with overtraining, a possibility also noted by Sutherland and Mackintosh (1971, p. 283). A number of studies demonstrating an ORE in spatial tasks were so constructed as to prevent the building up of smooth response sequences (e.g. Capaldi 1963, Ison and Birch 1961), the condition essential to the development of

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[*] The fact that, as Isaacson and Kimble (1972) stress, these prolonged runs were seen only with position and not brightness hypotheses must not be over-emphasized. An incorrect position hypothesis is partially rewarded, and this is not true for an incorrect brightness hypothesis.

[**] Kimble (1975) has re-analysed these data and pointed out that the hippocampal rats fell into two subgroups: those which learned very rapidly, and those which took considerably longer to learn. Thus, the bi-modality seen with position tasks is evident in non-position tasks as well. It is likely that the slow learners in the hippocampal group were those who adopted an incorrect orientation hypothesis. In a separate experiment published for the first time, Kimble (1975) demonstrated that the tendency to adopt fewer overall hypotheses, more and longer orientation hypotheses, also holds for lesioned rats tested in a Y-maze brightness discrimination where reward was given on every trial, regardless of the animal's choice. This confirms the notion that hippocampals are less flexible in their choice behaviour and more likely to persist in orientation hypotheses whether they are 'right' or 'wrong'.

[*] Olton's study shows that hippocampal rats can obtain information about stimuli which are not controlling their choice behaviour. This is incompatible with any attention-shift dysfunction theory, as is the study by Harley (1972) using compound cue discriminations.

strongly persistent orientation hypotheses. The reader is referred to Sutherland and Mackintosh for further discussion of this point. It is worth noting one further study here, as it will come up in our later analysis of the lesion data; Mackintosh (1965) has shown that the mere inclusion of a barrier which slowed rats down could lead to faster spatial reversals.

These considerations are important in view of the unusually crucial role orientation hypotheses play in hippocampal animals. Our previous discussion of discrimination learning, in conjunction with the above data from normal animals, would suggest that hippocampal animals should be particularly deficient at spatial reversals, in that they typically solve spatial tasks through the use of orientation, rather than place, hypotheses. No particular prediction is offered concerning the reversal of non-spatial tasks; however, those situations fostering the use of inappropriate orientation hypotheses could elicit deficits in reversal, in the same way they do in initial learning.

Spatial reversals. The results of those experiments concerned with the reversal of spatial discriminations are shown in Table A18. As suggested above, hippocampal animals show profound deficits in most of these studies. The few exceptions to this seem to involve procedures which either prevent the use of smooth response sequences or provide the animals with a means of breaking such sequences; that is, an alternative hypothesis. In a series of experiments Cohen and his co-workers have tested rats on a variant of T-maze spatial reversal (Cohen, LaRoche and Beharry 1971, Cohen and LaRoche 1972, 1973). A plus-maze was used, with only three arms open at any time. After learning an initial spatial habit rats were started from the previously unused arm (180° from the initial start arm) and required to go to the same place for reward. Thus, for rats using place hypotheses this new task does not involve a reversal in the standard sense of the term, while for those using an orientation hypothesis a reversal is required. Under this simple condition the hippocampal rats had a deficit in learning the new task. However, the addition of brightness cues at the start of the second task eliminated the deficit (Cohen *et al.* 1971). Presumably the animals could use these cues as an alternative to the orientation hypothesis, in effect treating the second task as an entirely new problem. Similarly, the presence of doors at the choice points also eliminated the deficit (Cohen and LaRoche 1972, 1973).

In several studies Stevens (1971, 1973b) explored the relationship between reversal and inter-trial intervals. An unusual procedure was used in these studies; the animals were minimally trained (either 6 or 10 trials without a learning criterion). Under these conditions hippocampal rats showed a deficit only when reversed to their initially non-preferred side, and then only when the inter-trial interval was 4 min. With a 5 second inter-trial interval reversal proceeded at a normal rate. These data are only suggestive, and further studies of the relation between inter-trial interval and reversal are needed, but they are consistent with the view that orientation hypotheses can be altered when they are repeatedly nonrewarded at a rapid rate. This point has already been raised in our discussion of habituation and will be discussed at length in the chapter on extinction (pp. 344-5).

Essentially, then, spatial reversals provide a measure of the extent to which an animal relies upon orientation hypotheses; the difficulty in reversal seems related to the use of these persistent habits. Hippocampals are deficient in this situation because they typically cannot use alternative hypotheses.

Non-spatial reversals. The results of studies using non-spatial reversals are given in Table A19. A split appears in these data between different species. Deficits are seen in most studies using hippocampal rats and cats, while hippocampal monkeys reverse at a normal or faster than normal rate. All of the studies reporting deficits in hippocampal rats and cats are unanimous in indicating that these animals do *not* have unusual difficulty in giving up their responses to the previously positive non-spatial stimulus;* most of the deficit results from the adoption of, and subsequent persistence in, an inappropriate (50 per cent rewarded) orientation hypothesis. Thus, Isaacson *et al.* (1968) note that

> 'hippocampally damaged animals do not perseverate the old response, they give it up and reach a plateau of 50% correct responses due to a fixation of an approach response to one side or the other' (p. 74).

The only exception to this pattern was seen in a part of the Isaacson *et al.* (1968) study where correction techniques were used. As we have already seen, this technique tends to prevent the use of persistent orientation hypotheses; hippocampal cats tested in this way reversed at a normal rate.

Reversal studies in monkeys extend this analysis. Zola and Mahut (1973) have carefully analysed data from both visual and tactile reversals in monkeys with fornix lesions or hippocampal damage. Lesioned animals gave up their responses to the previously correct object as rapidly as did normal monkeys. In contrast to what has been reported for rats and cats, Zola and Mahut showed that lesioned monkeys did not adopt persistent orientation hypotheses which could retard reversal. This was particularly apparent in the group tested on visual reversals. In this case the lesioned monkeys reversed *more* rapidly than did the intact monkeys, a result we

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* This point has been made in a different way in an experiment by Riddell, Malinchoc and Reimers (1973). Rats were initially trained on a brightness discrimination and then shifted to a position discrimination. Hippocampals accomplished this shift as rapidly as did normals.

discuss below. In the tactile reversals the animals did occasionally use orientation hypotheses, but no difference was seen in the rate of reversal between the various groups.

Zola and Mahut showed that the facilitation in visual object reversals in the lesioned monkeys was related to their achieving criterion more rapidly once they had given up their reponses to the previously correct stimulus; both groups took about the same number of trials to reach this stage. We have no simple explanation for this paradoxical facilitation of reversal; it might be related to the fact that orientation hypotheses are not used by monkeys in the WGTA to any great extent. This is consistent with the observation that normal reversal follows the use of either correction or non-correction techniques in this apparatus. Further, in one study reporting reversal deficits in hippocampal monkeys (Douglas and Pribram 1966) a different apparatus was used (the DADTA). We have already discussed one study using this multi-panel apparatus (pp. 275-6) and have seen that orientation hypotheses might play an important role in it. While no data were given in the Douglas and Pribram (1966) study concerning the use of orientation hypotheses, much of the deficit in the hippocampal monkeys appears related to prolonged responding at the 50 per cent correct level; this is suggestive of the use of such a hypothesis.

Thus, we can conclude that the reversal of non-spatial discriminations proceeds normally in hippocampal animals except when they adopt maladaptive orientation hypotheses. Situations constructed to prevent such strategies allow for normal reversal; situations allowing for such hypotheses typically produce deficits. Normal monkeys might use place hypotheses in the latter case, but these are easily dropped if proved incorrect.

7.2.2. SUCCESSIVE DISCRIMINATION

Another type of discrimination task that has been used with hippocampal animals involves presenting only one stimulus on any given trial, with the proper response depending upon which stimulus is presented. For example, if both arms of a T-maze are white the reward would be on the right, while if both are black it would be on the left. This task, termed successive discrimination, is typically more difficult for the normal animal than is the simultaneous task.* Most authors agree that there are two ways in which the normal animal can solve this task (cf. Lovejoy 1968, Sutherland and Mackintosh 1971, Mackintosh 1974). The animal could learn to make a particular response given a particular stimulus (i.e. if black, turn left; if white, turn right), or it could learn a compound discrimination (i.e. approach white on the right or black on the left, but avoid white on the left or black on the right). The former alternative does not appear to involve place learning and also does not seem to be used by normal animals except in rare cases. One such case, it should be noted, occurs when the stimuli are separate from the place where the response is to be made. Here, of course, the locale system would be at a disadvantage. The latter alternative, involving a compound discrimination solution, could be facilitated by the use of the locale system as the animal can learn to go to a stimulus in one place but not in another. Given this, it is likely that hippocampal animals would have deficits in such tasks. Four studies have tested hippocampal rats on successive discrimination (Kimble 1963, Lash 1964. Seidenstadt and Hagstrom 1970, Isaacson, Schmaltz and Douglas, 1966) and deficits were seen in all cases. The nature of the deficit seems analogous to that seen in simultaneous discrimination.

Lash (1964) tested rats on successive discrimination in three different mazes. These mazes differed in the extent to which the responses made in them were discrete and easily discriminable. Hippocampal rats tend to adopt more pronounced orientation hypotheses in those mazes allowing for the most discriminable responses; their performance on these three mazes was inversely related to response discriminability. The more likely they were to adopt an orientation hypothesis, the poorer the performance. A study of the post-operative retention of a pre-operatively acquired successive discrimination provides data consistent with this view. Isaacson et al. (1967) trained rats on two mazes, a Y-maze and an arrowhead maze. Responses on the latter are more discriminable, and this maze was easier to learn pre-operatively by all the rats. Post-operatively, the hippocampal rats had deficits on the successive task; they showed pronounced orientation hypotheses on the arrowhead maze in particular.

Isaacson and Kimble (1972) and Isaacson (1974) have re-analysed the data from Kimble (1963). They suggest that animals solve this task as though it were two separate problems, using what we described above as 'learning a response to a particular stimulus'. The data they present, however, fail to support their analysis. If normal animals did, in fact, treat this situation as two separate problems, learning first one (if white, go right) and then the other (if black, go left), one would expect criterion performance on one task combined with random performance on the other. This pattern was seen in only one of the six normal animals tested (Isaacson and Kimble 1972, Table 2). Two of the animals showed a pattern involving criterion performance on one task (e.g. if black, go left) combined with total failure on the other. In other words, these animals responded on the basis of a place or orientation hypothesis, always going to the same place or making the same turn. The remaining three normal animals seemed to learn both components of the task concurrently, indicating the use of the compound discrimination discussed above. It is worth noting that these three rats had the fastest learning rate. Hippocampal

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* This successive discrimination task should be differentiated from another task involving separate presentation of stimuli. In this latter case a single stimulus is either presented or not, and the animal is rewarded for responding in its presence and not in its absence. This task, termed successive go-no-go discrimination, will be discussed later.

rats (Isaacson and Kimble 1972, Table 5) were more likely to fall into an orientation hypothesis; three of the five hippocampals showed turning hypotheses and the other two showed a pattern of responding consistent with the use of an hypothesis like that described by Isaacson and Kimble, that is, solution of one problem combined with random responding on the other. None of these animals learned both tasks concurrently.

These data, then, demonstrate that normal animals learn either by the use of a compound discrimination or a two-component orientation hypothesis, and that the former solution is faster. Hippocampal animals, on the other hand, use only the latter solution, or a one-component orientation hypothesis yielding maladaptive behaviour. They seem incapable of utilizing the compound discrimination strategy.

7.3. Maze learning

The use of complex mazes has had a long history in experimental psychology since their introduction by Small (1901). We have already referred in a general way to some of this work (pp. 50-2). Here we shall want to look more carefully at the factors which early experimenters identified as contributing to the rat's ability to 'solve' the maze. Much of the relevant literature is summarized by Woodworth (1938) and Munn (1950 pp. 181-226, 255-65). The reader's attention is especially directed to the classic article by Dashiell (1930). Before Dashiell's work, it had become generally accepted that one factor involved in maze learning was what Hull (1932) called the *goal gradient* factor. Animals tended to eliminate errors at choice points near the goal end of the maze earlier in training than those at the start of the maze. Hull postulated that the effect of reinforcement was strongest at the goal and diminished progressively the further one went backwards through the maze.

It was clear, however, that other factors were also involved. Dashiell's important contribution was to identify a *goal-direction* factor. He had noticed a curious improvement in the performance of a group of rats trained on a set of maze problems which were the forerunner to the Hebb-Williams maze. The maze was a rectangular box into which partitions in various configurations could be set. Different problems were made up from different configurations of barriers but the start box was always located somewhere along one side of the box while the goal was always on the opposite side. The rats showed a steady improvement across mazes suggesting that, in addition to the specific route for each problem, they were acquiring some general knowledge of the direction of the goal from the start and other parts of the box. Further evidence for this goal-direction factor was a marked reduction in their willingness to enter blind alleys which pointed away from the goal and a greater difficulty in avoiding blinds which pointed towards the goal. This latter

observation was confirmed in a multiple-T, six-choice point maze in which the blind alley entered most pointed towards the goal and the three alleys entered least pointed away from the goal. Similarly, in an elegant little maze with one blind pointing forward and the other backward, animals run in a configuration in which the goal was located forward made more entries into the forward-pointing blind while animals with the goal backward entered the backward blind more often. Similar analyses of the relative difficulty in eliminating goalward-pointing blinds were done by Tolman (1932), Spence (1932), and Hull (1952), the latter two in a pseudo-numerical fashion which added little to the original observations.

Dashiell's final demonstration of the goal-direction factor involved the maze which now bears his name. This is a checkerboard pattern of alleys and blocks which offers the rat numerous alternative paths to the goal. In such a maze rats do not repeatedly choose the same path but vary their route from trial to trial. They learn not an S-R routine but the direction of the goal.

In addition to the goal-direction and goal-gradient factors, other less important factors contributing to the rat's maze performance were listed by Woodworth (1938): (a) the entrance or start-box area is important in orienting the animal; often when a rat is placed in the middle of a familiar maze, it will head back to the start and then turn around and make a successful run; (b) anticipatory errors occur when the rat prematurely makes the last turn before the goal; for example, in a linear maze with the correct response sequence RRRRRRRL, the most difficult choice point is the final right turn (Spragg 1933); (c) egocentric predilections such as a tendency to continue forward after a detour, wall hugging (thigmotaxis), or a preference for left or right turns.

According to the theory we are advancing, there are two ways that the cognitive map would be useful to an animal running a maze. First, it locates the animal's position in the maze, and second it points the direction of the goal from that position. The second type of information can be used to generate the hypothesis 'go towards the goal' and is entirely responsible for the goal-direction factor. The first type of information makes available a set of place representations which can be incorporated into place hypotheses about the individual choice points: 'go from place A (first choice point) to place B (second choice point)'. While there is no obvious difference as far as we can see amongst various maze configurations in the usefulness of place hypotheses concerning individual choice points, the same is not true about the goal-direction hypotheses. Mazes with many choice points where one alley runs in the direction of the goal and the other away from it will be more sensitive to goal-direction hypotheses than those in which all or most of the choices have both alleys perpendicular to the goal direction (such as in linear mazes).

Clearly, then, the theory predicts that animals with hippocampal lesions will be impaired on all mazes. Further, in contrast to normals, hippocampals should be insensitive to goal-direction factors such as whether blinds point towards or away from the goal. Finally, since the lesioned animals must rely on routes (concatenations of guidances and/or orientations) to solve the maze, they will be at least as sensitive as normals to these factors. For example, their performance should improve if they are provided with a set of cues at choice points or given a trail to follow. If there is a goal gradient (and the data are by no means unequivocal) then hippocampals should show it in a pure form, learning the choices at the end of a maze more quickly than those at the start.

7.3.1. HIPPOCAMPAL LESIONS AND MAZE LEARNING

Table A20 lists studies concerned with complex maze learning in hippocampal animals. Deficits were reported in 18 of 20 studies. We searched through this literature for studies which provided information on the nature of the deficit in order to test the more specific predictions of the theory. Six gave information about the performance of the animals on individual choice points or on mazes of different configurations: Kimble (1963), Kveim, Setekleiv, and Kaada (1964), Jackson and Strong (1969), Winocur and Breckinridge (1973) Myhrer 1975a, and Myhrer and Kaada 1975. Kimble, Kveim et al., Myhrer 1975a, and Myhrer and Kaada 1975 used the Hebb-Williams maze.* Kimble chose two problems, one which he considered easy (no. 1) and the other difficult (no. 6). The easy problem requires the animal to run directly from the start to the goal, while the more difficult one is solved by a twisting route which at one point requires the rat to turn away from the direction of the goal. The first is clearly aided by a goal-direction factor while solution of the second is hindered by it. The unoperated controls in Kimble's study made an average of three times as many errors on the difficult maze as on the simple one, in spite of the fact that the method of scoring errors afforded almost twice as many opportunities for making errors in the simple maze. The hippocampals were significantly impaired on both mazes, but made twice as many errors on the simple maze. In view of the differences in possible errors in the two mazes (see above) this probably represents equally poor performance on both mazes. Thus, unlike the normals, their behaviour seemed not to reflect a sense of the goal direction; Kimble describes the hippocampals as running back and forth in the side alleys of both mazes and failing to enter the centre of the maze as the control animals did (see also Hughes 1965). Myhrer 1975a found essentially the same results but also had a large deficit in his neocortical controls.

We tried to extend this type of analysis to the Kveim *et al.* study which used all 24 problems of the Hebb-Williams maze and presented the data for each problem separately. If all the problems could be rated as reliably in terms of their sensitivity to the goal-direction factor as nos. 1 and 6, we would predict that the hippocampals, although impaired on all problems, would be relatively less impaired on problems where the goal-direction factor hindered the normals (as in no. 6) and relatively more impaired on problems where it helped them (as in no. 1). Kveim *et al.* did find that some problems were relatively easier for the hippocampals than others, but we have not been able to convince ourselves that these are different from the rest in terms of the goal direction factor. It should be noted that the Hebb-Williams maze does not lend itself to this type of *a priori* analysis in the way that the multiple-T non-tracing mazes used by Dashiell, Tolman, and other earlier workers do. Furthermore, the use of a dome over the maze by Kveim *et al.* would tend to militate against the goal-direction factor. Finally, the rank-order correlation of 0.78 between the scores of the normals and the hippocampals suggests that the hippocampals found all mazes equally more difficult and that there was no interaction between problems and the lesion.

Jackson and Strong (1969) used a six-alley, twelve-cul Lashley-III maze. In this maze the rat runs along each alley roughly at right angles to the direction of the goal until it comes to an opening in the wall in the direction of the goal. Correct performance requires the animal to turn through the wall in the direction of the goal and then to make another 90° turn so as to head along the new alley in the direction opposite to which it had just been coming. Jackson and Strong point out that two different types of error are possible in this maze. The first (called a door error) occurs when the rat fails to turn into the wall opening and continues into the cul at the end of the alley. This error is sensitive to goal direction and should be selectively biased against the lesioned animals. The second type of error (an alternation error) is committed when the animal goes through the opening but turns in the wrong direction in the new alley. Since the new alley is perpendicular to the goal direction, alternation errors are insensitive to this factor and should not distinguish between the two groups. This is exactly what Jackson and Strong found. Confirmation that the Lashley-III maze is sensitive to the goal-direction factor comes from a study by Zack (1968). He showed that normals, but not hippocampals, were disturbed by rotation of the maze in the room after they had learned.

Winocur and Breckinridge (1973) used a six-choice linear maze in which each choice point consists of a pair of doors both roughly parallel to the direction of the goal, rendering the task relatively insensitive to the goal-direction

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* The Hebb-Williams maze (see Rabinovitch and Rosvold (1951) for the configurations) is a successor to the Dashiell maze described above. There are actually only 12 configurations; the mirror images of these make up the additional 12 problems. The difficulties in scoring errors and the inequality of errors at different points in this type of maze which prevented our using the Kveim et al. data were amongst the reasons for the original abandonment of the predecessors to the Hebb-Williams maze.

factor. The order of the correct doors was LRLRRRL. They found that the hippocampals were impaired on all choice points but more so on choices 3 and 5. We can offer no theoretically motivated explanation for this selective impairment. Perhaps the fifth door proved difficult because the rats were using an LR alternation hypothesis. In an important series of attempts to improve the performance of the lesioned animals, Winocur and Breckinridge added guidance cues to the doors at each choice point in one experiment or to just the fifth door in another. Adding cues to all the doors improved the performance of both groups by about 50 per cent but the significant difference between them remained. When the cues were subsequently removed, the performance of the hippocampals but not the normals deteriorated. Provision of a guidance at the fifth door alone was also beneficial for both groups and furthermore erased the selective difficulties of the lesioned animals at doors 3 and 5.

A study by Leaton (1969) also demonstrates the reliance of lesioned rats on guidances and/or orientations. He forced rats, over a series of trials, to run the correct path in a six-unit multiple U-maze by blocking off the incorrect alleys. After this initial training the animals were tested with the blind alleys opened. Leaton found that when the blind alleys were quite similar to the correct alleys the hippocampal rats made considerable cul entries. When the blinds were obviously dissimilar, the hippocampals avoided them as well as the normals.

The performance of hippocampal rats has been tested on the Dashiell maze in two experiments (Niki 1966, Ellen and Bate 1970). As predicted, Niki found not only a deficit but also less flexibility in the number of routes used by the lesioned animals. In the Ellen and Bate study the pathways through the centre of the maze were of a different brightness from those on the outside, providing the animals with a guidance trail to follow. Both normals and lesioned animals showed little variation in routes and there was no difference between them.

In summary, several points can be made. First, there is a universal deficit in maze learning in animals with hippocampal lesions. We attribute this to the absence of place information which would support decisions at choice points where no guidance cues are available. In the absence of either guidance or locale hypotheses these animals must rely on orientation hypotheses such as LR turn alternations or left turns at all doors. Provision of guidances helps hippocampals as much as normals. Second, although strong evidence on the insensitivity of lesioned animals to goal-direction factors is lacking, what there is, is consonant with the prediction. We suggest that future studies of maze learning in hippocampals be designed to test different hypotheses about why these animals fail. As a test of the goal-direction hypothesis derived from the cognitive-map theory, we recommend variants of the simple two blind maze used by Dashiell (1930, see above).

8

Aversively motivated behaviour

IN this chapter we consider the role of the locale system in an animal's response to unpleasant, or threatening, situations, where a *threatening situation is* taken as one which predicts the possibility of pain. We first consider the various forms of threat and the ways in which animals identify and subsequently cope with these threats. Particular emphasis will be given to the distinction between threatening places and cues. Following this we discuss the effects of hippocampal lesions upon behaviour in threatening situations. It will be shown that animals with such lesions retain an appreciation of threat when it relates to cues and respond appropriately to such threats, but fail either to apprehend or adequately to respond to threatening places.

8.1. Behaviour under threat

The most powerful current theory of how an animal learns to cope with threat is traditional two-factor theory (Mowrer 1939, 1947, Konorski 1948, Rescorla and Solomon 1967, and others). This theory postulates two basic processes: (1) the Pavlovian or classical conditioning of *fear* to certain stimuli; (2) the instrumental learning of an adequate response. Traditional theory assumed that the appropriate response was maintained by the reinforcement involved in the reduction of fear. We return to a fuller discussion of this and other, more recent, theories later. Here, we wish to reformulate the two-process model in order to bring it into line with the cognitive approach of this book. We shall postulate that two processes are involved when an animal is confronted with a threatening situation: (1) the identification of the threat; (2) the choice of a particular hypothesis to cope with this threat.

8.1.1. THE IDENTIFICATION OF THREAT

8.1.1(a). Types of threat. There are several ways in which different threats could be classified, and the scheme one chooses obviously reflects the bias of the investigator. Thus, one could separate threats on the basis of the role of learning in their establishment, the possibility of escape, the availability of prediction and/or control, and so on. Within the present framework we consider a separation based on places and cues as most profitable. We hope to show in this chapter that such a distinction provides a powerful approach to the study of aversive learning and that it is essential to an understanding of the behaviour of hippocampal animals in threatening situations. We can further separate threats according to the animal's previous experience with them. Thus, *innate threats* have inherent properties, apparent upon an animal's first exposure to them (e.g. predators); *learned threats* are aversive through prior association with pain, or perhaps another threat (e.g. CS paired with shock, or a house of horrors); *potential threats* are objectively unknown quantities, novel places and cues, which are treated by the animal with caution, a mixture of fear and curiosity.[*]

The fact that learned threats require prior association with pain, or some other threat, raises the possibility that there are fundamental differences in the way animals react to these as compared with innate threats. There are two reasons for assuming that this is not the case. First, there is evidence that the brain mediates reactions to both learned and innate threats in the same way.[**] Second, basic behavioural reactions, barring the reflexive response to pain itself, are much the same for the two classes, as we see shortly. We would suggest that it is more important to separate threats on the basis of whether they relate to a place or a cue than on the basis of whether they are innate or learned. Similarly, we consider such factors as the escapability or predictability of threat as secondary to the place-cue distinction. Such factors can alter the kinds of behaviour animals display in threatening situations, but seem to be less important in determining the overall form of behaviour itself than are places and cues. Evidence relating to these assertions will be presented in the ensuing discussion, but we shall concentrate primarily upon the role of places and cues in animals' responses to threat.

8.1.1(b). Discovering the threat. We would suggest that animals rapidly learn *where* threat comes from. The receipt of pain, or the presence of a threatening item, in a particular place, causes that place to be identified as threatening. This type of learning would seem directly analogous to that discussed for appetitive situations in the previous chapter. That is, animals learn where rewards are to be found and subsequently associate these places with specific cues when possible. In threatening situations animals initially respond on the basis of the dangerous place, directing their responses towards or away from that place.

In some situations it is not possible to identify a specific item as threatening, and behaviour continues to be directed towards, or away from, places. In other situations a threatening item can be identified, and behaviour becomes directed towards, or away from, it.[*] In so far as threatening places and cues elicit different behaviours, the ability of the animal to discriminate the threatening cue will be a major determinant of behaviour. We shall see shortly that the distinctiveness, or *salience,* of the threatening item strongly influences the choice of a coping hypothesis.[**]

According to the above analysis, the responses of animals in threatening situations should depend upon whether the threat comes from the place or some specifiable item. There is some evidence from the work of the Blanchards and their colleagues concerning the way in which animals initially identify and respond to threatening places and items. Blanchard, Dielman, and Blanchard (1968a, b) have shown that shock will cause rats to crouch or freeze, and that this behaviour is linked to the place in which shock had been received. Blanchard and Blanchard (1969a) extended this analysis by demonstrating that rats shocked in one place will not crouch if immediately moved to another, neutral, place. When subsequently returned to the original, dangerous, place the rats crouched. Thus, postshock crouching was not a response to pain *per se,* but rather to the place in which pain had been received.[***] Much the same thing is seen when

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[*] Some insight into the mechanisms operating when an animal is confronted with a novel item (food) has been provided by Kalat and Rozin (1973). They showed that such foods are initially regarded as potentially dangerous and that only some time after they have been ingested without *adverse consequences* does the animal treat them as safe. A novel environment could be treated in the same way. While Blanchard, Kelley and Blanchard (1974) have concluded that novel environments elicit only fear, Cowan and Barnett (1975) suggest that they elicit primarily curiosity.

[**] Goddard (1964), Blanchard, and Blanchard (1972a) and others have provided strong evidence that the amygdala mediates reactions to both innate and learned threats. Similar evidence implicates the amygdala in the 'tagging' of positive rewards. Thus, there are good reasons to suppose that the amygdala is concerned with the *valence* of environmental features.

[*] Analogously to the autoshaping seen in appetitive situations (p. 263), the locations of stimuli associated with threat become aversive. That is, the site of a light which signals the imminence of shock is avoided; lever pressing to avoid shock is more easily learned if the light is distant from, rather than near, the lever (Biederman, D'Amato, and Keller 1964).

[**] A salient CS in a fear-conditioning situation tends to reduce the magnitude of place fear, as animals learn that threat is specifically linked, either spatially or temporally, solely with the CS (cf. McAllister *et al.* 1974).

[***] The question of the time-course of changes in crouching after shock is an interesting and involved one. In most of their studies the Blanchards measured the change in crouching over time, during which the animal was left in the threatening situation. Thus, they were actually measuring the extinction of crouching. Blanchard and Blanchard (1969a) did compare crouching either immediately or one hour after shock, with the latter group kept outside the shock situation for the shock-retest interval. Crouching was approximately equivalent for the two groups for the first hour they spent in the shock situation, independent of the shock-retest interval. This result suggests that crouching does not change, at least over one hour. However, there is a large body of data concerned with the fact that certain aspects of behaviour do change as a function of time after shock, in particular that freezing can increase–the so-called *incubation effect* (e.g. McMichael 1966). In this case the animal spends the shock-retest interval in a different place and the typical finding is that the ability of the threatening place or item to elicit freezing increases monotonically with time. In addition to this there is the *Kamin effect* (Kamin 1957). Here, a U-shaped relearning function is displayed after initial training in avoidance. Some recent work by Pinel and Mucha (1973a,b) has suggested that the incubation effect represents a change in *activity* in response to threat, while the Kamin U-shaped function is based on changes in reactivity to shock over time. As with the Blanchards' results it appears that the incubation effect is linked to the particular threat; there is no suggestion that an animal's activity decreases independently of the presence of threat on retest. The extent to which the Kamin effect is dependent upon re-establishing the original shock situation is unclear. Brush (1971) reviewed much of the incubation and Kamin-effect literature; he also quoted an unpublished

a threatening item is put into an otherwise neutral place. A rat confronted with a cat for the first time will spend a proportion of its time crouching (Blanchard and Blanchard 1972b). Over time an increase in crouching develops, even though no contact with the cat has been made. Some of this freezing persists after removal of the cat, indicating that the rat has identified the place as threatening.* Blanchard and Blanchard (1969b) have shown the same thing with an electrifiable prod which had previously been used to administer shock in another place. Thus, evidence from several studies attests to the relative ease with which places are identified as threatening, either through the receipt of pain or through association with threatening items.**

A somewhat different picture emerges when one considers reactions to threatening items, separately from the places within which they occur. Blanchard and Blanchard (1970a,b) have shown that highly discriminable threatening items elicit some increase in crouching, but a much more specific avoidance of the particular item itself. Thus, as a result of discriminating and identifying a specific threatening item the animal's behaviour becomes relatively tied to that item. This ability to separate out specific threatening items lies at the root of hypotheses based on approach or avoidance of items rather than places.***

The above discussion indicates that animals rapidly learn to identify threatening places and seek to discriminate those items within the place which are specifically dangerous. The seeming predominance of place learning suggests that, as in appetitive learning and primarily at the onset of learning, place hypotheses might be most effective in coping with threat.

8.1.2. COPING WITH THREAT

The forms of coping behaviour elicited by threat fall into a few simple categories. In fact, it has been suggested that the limited behavioural repertoires evidenced in the face of threat are of some special theoretical significance (e.g. Bolles 1970, 1971); we shall discuss this position shortly. When confronted with a dangerous place animals attempt to flee; this appears to be the highest-priority hypothesis. When flight is not possible animals freeze. In studying the rat's reaction to threatening places this importance of flight has been repeatedly emphasized. Thus, Bolles (1970) asked:

> 'Why is a change in geographical location such a particularly effective stimulus change?' (p. 42).

The foregoing would suggest that one highly likely hypothesis in response to threat is: 'get out of this place'. Further, it seems likely that animals would preferentially escape to places previously identified as non-threatening.

Behaviour in response to a threatening item is somewhat more complex. Again, flight would appear to be a likely possibility. Blanchard and Blanchard (1971) have shown that, in response to a cat, rats will flee if possible. They conclude that

> 'the availability of escape is a principle determinant of the relative dominance of these reactions' (p. 361)

referring to flight and freezing. The failure of a flight hypothesis does not, in this case, automatically lead to freezing. The animal can attempt to manipulate the situation so as to make the threat 'disappear'. Thus, animals can learn to press levers to turn off a threatening buzzer. Another possibility is to move to some part of the environment which is not associated with threat, as in the case of the avoidance of an electrifiable prod, or shocked water dish, or cat. Finally, depending upon the topology of the situation and the character of the threatening item, animals will sometimes engage in attack. The crucial determinants of attack would seem to be the distance between the animal and the threatening item. In practice this shows up in the fact that animals attack more frequently in small enclosures and when confronted with threatening items which move, thereby making it difficult to maintain some critical safe distance (see Myer (1971) for a review of some of these studies).

The above suggests that various kinds of hypotheses are possible when an animal is confronted with threat. A threatening place can be coped with by 'going to a safe place' or simply 'leaving the dangerous place'. A threatening item can be coped with by 'fleeing the item', or 'approaching items distinguishable from danger', or 'making a response which temporally or spatially decreases the impact of the item', or, lastly, 'attacking the item'. In some situations more than one of these hypotheses would be appropriate and we would suggest that, to the extent that conflict exists, place hypotheses would have precedence, if only because animals seem to learn more rapidly about places than about items. This overview

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experiment by Brush and Sakellaris which suggests that the Kamin effect is *not* based on changes in reactivity to shock. Thus, the nature of the time-dependent changes in retention of aversive contingencies remains a confused issue. We do not feel that these time-dependent changes basically affect the argument we are making here. Recently, Klein, Murphy, and Freeman (1975) have shown that the Kamin effect is intact in rats with hippocampal lesions.

A different set of issues arises when one considers the effect of multiple inescapable shocks upon subsequent behaviour (e.g. Anisman and Waller 1973). Here, too, there is considerable confusion, and controversy, concerning arguments that are peripheral to our focus.

* Blanchard, Mast, and Blanchard (1973) have studied the factors controlling the rats reaction to the cat. Neither the smell, nor the sight, nor the vocalizations of the cat are in themselves enough to elicit crouching. Movement of the cat is critical. In fact, movement of non-predator items also elicits some crouching.

** A threatening place, in our terms, is obviously related to what has been called s*ituational* fear. It differs in so far as we attribute quite different properties to it, relative to those associated with items, or *discriminated* threats.

*** There is an interesting but as yet untested possibility that putting a neutral item into a dangerous place results in that item being identified subsequently as threatening.

of behaviour in threatening situations can best be evaluated by comparing it with other, more traditional, views in its ability to account for some common varieties of aversive learning.

8.1.3. AN ANALYSIS OF BEHAVIOUR IN RESPONSE TO THREAT

There are a great many behavioural paradigms based on the use of aversion. These can be classified in ways which are parallel to those used in the classification of threats. Thus, there are tasks related to places or items, to innate or learned threats, to escapable or inescapable threats, to random or stimulus-contingent or response-contingent threats, and so on. The appropriate combination of these factors gives rise to the paradigms known as *escape, conditioned fear, punishment, avoidance,* and others. It is not our intention here to discuss the entire range of aversively motivated behaviours. There exist numerous reviews in this field for the interested reader (e.g. Mackintosh 1974). Rather, we shall concentrate upon avoidance, as much of the work, both with normal and lesioned animals, has utilized variants of this paradigm. Where necessary we shall extend the analysis to other paradigms.

Some writers have attempted to classify avoidance tasks in terms of the nature of the response required, in particular separating out so-called *active* and *passive* avoidance tasks. This tendency has been particularly strong within physiological psychology. In so-called active avoidance animals can avoid pain only through some active behaviour, while in passive avoidance the animal can easily avoid pain by remaining immobile. This distinction is unfortunate for two reasons. First, it carries with it the implicit assumption that animals are actually learning responses, and we shall see that this is only one way in which active avoidance can be mastered. Second, it implies that animals solve passive avoidance tasks by remaining immobile. Again, there are reasons to doubt this conclusion. In our discussion of the lesion literature we shall partially maintain the use of this active-passive distinction, merely as a means of classification, without any assumptions about the way in which animals solve these tasks. Our main discussion centres around active avoidance in general, and the variety known as shuttle-box avoidance in particular.

8.1.3(a). Active avoidance in the normal animal. The standard active avoidance paradigm is deceptively simple. A rat is placed in one side of a two-compartment box (A). After some time a neutral stimulus (the conditioned stimulus CS) such as a tone is presented and several seconds later an electric shock (the unconditioned stimulus, UCS) is delivered through the grid floor of side A. After its immediate reflexive reactions to the shock the rat will eventually escape to the other side of the box (B) where there is no shock; the CS and UCS are typically terminated by this response. After a fixed interval (the confinement period or inter-trial interval ITI) the rat is removed from B and replaced at the start (A) for another trial or, alternatively, given a further ITI in a neutral place before starting another trial. Within a relatively small number of such trials the rat learns to move from A to B when the CS is presented, thus avoiding rather than escaping the shock. This behaviour is then maintained in the absence of further shocks.

Two problems are presented to the theorist by this task, termed *one-tray active avoidance*. First, there is the question of the types of hypotheses animals use to solve it. Second, there is the issue of the factors which maintain avoidance behaviour in the absence of any obvious reinforcement. For various historical reasons most of the work in this field has concentrated on the second issue, and conclusions about the first must be inferred from theories built primarily to account for the paradoxical maintenance of avoidance. We can begin our analysis of avoidance learning by looking at the traditional approach, two-factor theory, its recent modifications, and an alternative scheme proposed by Bolles (1970).

Two factor theory. The essence of traditional two-factor theory, as noted already, is that avoidance behaviour involves two main processes: the Pavlovian conditioning of fear to the CS, and the instrumental learning of an adequate avoidance response. Experimental work in support of this model attempted to show that something called fear could be conditioned to neutral stimuli and that responses leading to the reduction of fear were learned. These mechanisms could account for both the acquisition and maintenance of avoidance behaviour.

However, it is now clear that the ability of the CS to elicit fear is not necessarily maintained during the course of stable avoidance behaviour (Kamin, Brimer, and Black 1963, Linden 1969); these findings, and others concerned with the abnormal persistence of avoidance under extinction conditions (cf. Seligman and Johnston 1973, for a discussion of the two-factor model in this context), pose a serious problem for two-factor theory. The traditional model has recently been revised, typically so as to expand the role of Pavlovian conditioning during avoidance to include the development of a response-produced inhibitory feedback which both reduces fear and reinforces behaviour (e.g. Weisman and Litner 1969a,b, 1971, 1972).

The same problem has been addressed by Denny and his colleagues in a somewhat different way. In a series of studies (e.g. Denny and Weisman 1964, Weisman, Denny, and Zerbolio 1967, Denny, Zerbolio and Weisman 1969, Denny 1971) they have provided evidence in support of a relaxation model of avoidance behaviour. Stimuli which signal punishment elicit fear, and their removal is followed by relief and relaxation which act as reinforcers for the responses leading to these states. It is clear that relaxation is, in most essential respects, similar to the conditioned inhibition of fear postulated by revised two-factor theory. Both depend upon events subsequent to the avoidance response. As we shall see shortly, a number of experiments done within the relaxation framework have provided evidence relating to the question of the kinds of hypotheses animals use in solving avoidance tasks. In particular, these data point to the importance of place hypotheses.

Species-specific defence reactions (SSDR's). Bolles (1970) has pointed out that an animal's choice of responses in aversive situations is hardly random. He suggests that

> 'What keeps animals alive in the world is that they have very effective *innate* defensive reactions which occur when they encounter any kind of new or sudden stimulus. These defensive reactions vary somewhat from species to species, but they generally take one of three forms: Animals generally run or fly away, freeze, or adopt some type of threat, that is, pseudo-aggressive behaviour' (p. 33).

The strongest evidence in support of the view that SSDR's play an important role in avoidance learning is the fact that those tasks requiring as responses either SSDR's or slight modifications of these are learned quite rapidly, while those which require responses incompatible with SSDR's are learned more slowly or with great difficulty (Bolles 1970). Thus, one-way active avoidance, as described above, is learned rapidly as it can involve flight. On the other hand, learning to press a lever to avoid shock can take an agonizingly long time.

Bolles has also offered an explanation for *two-way active avoidance,* which is a variant of the one-way task with a crucial procedural difference. Upon completion of a trial (be it escape or avoidance) the animal is *not* removed and returned to the start. Rather, after an inter-trial interval the CS is presented again and the animal must 'shuttle' in the other direction (i.e. from B to A) to avoid shock. This task is considerably more difficult for the normal animal than is the one-way task, and most investigators attribute this to the fact that the animal must re-enter areas where punishment had previously been received. Bolles suggests that the conflict inherent in this situation promotes the SSDR of freezing, which is incompatible with the shuttling (or flight) responses necessary for appropriate avoidance. This invocation of conflict between flight and freezing is a constant feature of SSDR explanations in avoidance, both in the active case, as seen here, and in the passive case, as discussed already for the work of the Blanchards (p. 293). Bolles supposes that specific stimuli, such as the CS or a feedback safety signal, become increasingly important when such conflicts exist. In a series of studies Bolles and Grossen (1969) showed that such stimuli were important in the learning of two-way, but not one-way, active avoidance. Thus, SSDR's such as flight and freezing suffice by themselves in one-way avoidance and most varieties of passive avoidance, while two-way avoidance and such tasks as lever-press avoidance require the mediation of specific stimuli supporting the learning of non-SSDR responses.

'Cognitive' theory. Dissatisfaction with two-factor theory has coincided with the general rebirth of interest in cognitive approaches to behaviour. Recently, Seligman and Johnston (1973) have elaborated such an explanation for avoidance learning. Their model rests on the 'cognitive' notion that animals have preferences and expectancies, and that the combination of these can, in any situation, influence behaviour. Classically conditioned fear remains in the theory as a source of motivation, but its reduction is no longer seen as essential in either the learning or maintenance of avoidance. Basically, then, this model exchanges instrumental acts for preferences and expectancies, and eliminates the reinforcing role of fear or safety. It remains a fundamentally associationistic, two-factor theory. The confirmation of expectancies acts to reinforce those behaviours predicated on these expectancies.

Unfortunately, this model fails to go any further. It is easy to see that animals would prefer no shock to shock, or safety to danger. But, how are these latter identified? As a description of what goes on *after* an animal has figured out its environment the model succeeds. As an explanation of how animals learn about the safe and dangerous aspects of their environment it is reduced, it seems, to the trial-and-error strengthening of expectancies, biased perhaps by something akin to SSDR's.

A synthesis. None of these models, in our view, provide a completely satisfactory account of avoidance behaviour.* One is left wondering about the circumstances under which the conditioned inhibition of fear, or relaxation, occur. The nature of flight, and the factors determining its occurrence, are not adequately spelled out (though Blanchard *et al.* 1975 have begun the process of spelling out some of the stimulus factors controlling freezing). It remains possible that if these factors were elucidated they might point to something like 'relaxation in a safe area', leaving us with the essential problem of determining just why an animal feels safe when he performs an avoidance response. In other words we have not gone very far beyond a description of what the animal actually does. Similarly, the cognitive model of Seligman and Johnston fails to go beyond a description of behaviour to an analysis of its antecedents.

A common thread working through all these difficulties has been the refusal of traditional theorists to distinguish between threatening places

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* There are other approaches to aversive learning not discussed here. One could, for instance, mention Herrnstein's (1969) attempt to account for such learning simply in terms of shock density. Mackintosh (1971) effectively refutes this argument, while Seligman and Johnston (1973) subsume it.

and cues. Though the literature is rife with hints that the 'discriminability' of the source of threat is an important variable and that quite different results obtain when 'situational' rather than 'discriminated' threat is involved, theorists have so far refused to examine the implications of this for their theories.* To suggest that place learning is at the root of all avoidance behaviour is not our aim; however, it is clear that such learning can be important, perhaps crucial, in many avoidance situations, and that a consideration of such learning could provide a means of extending current views of avoidance behaviour.**

We have already specified what we mean by a threatening place, and how it is to be distinguished from a threatening item. Further, we have seen that this distinction enables one to demonstrate that animals react differently to places and items. This framework can be used to examine the ways in which the locale system might influence avoidance behaviour. First, we can consider the question of flight. Neither Bolles (1970) nor Blanchard and Blanchard (1969a) specify the variables controlling flight. While it is clear that these authors cannot mean that flight exists in an environmental vacuum, they have not managed to elucidate those aspects of the environment towards which, or away from which, flight is directed, except in the limited case of flight from a well-specified item. The Blanchard and Blanchard (1971) study suggests that

> 'such factors as availability of escape and discriminability of the eliciting stimulus largely determine whether freezing or avoidance will be the principal response to threat' (p. 361).

However, what is meant by the availability of escape? We have assumed that this depends on the animal's ability to find a place which it considers safe. Given this, one can suggest that flight is a function of several factors: (1) a tendency to move away from threatening places or items; (2) a tendency to move towards safe places. The importance of a change in geographical location lies in the fact that such a change usually involves going to a different place, and that in itself can reduce the threat that confronts the animal. We do not deny the potential validity of the relaxation concept; a new place, given that it is known to be safe, will provide a situation in which the animal can relax. What is crucial, however, is not the relaxation *per se,* but the attainment of safety.

Thus, when confronted with a threatening place or item, animals, as a first hypothesis, seek out a safe place. The predominance of flight reactions in threatening situations is a function of the rapid learning about places and the subsequent rapid availability of place hypotheses. If a safe place can be found avoidance will be rapidly acquired. One-way active avoidance provides a situation in which a relatively safe place exists. We have already noted that neither CS termination nor a feedback safety signal play much of a role in either the learning or maintenance of this type of avoidance in the normal animal (Bolles and Grossen 1969). If, however, we consider the role of place learning, we can see that animals are sensitive to those features of the task situation which affect the use of place hypotheses. Thus, any manipulation which increases the discriminability between the safe and dangerous areas in the avoidance box, or increases the safety or danger of one or the other, can lead to more rapid learning (e.g. Knapp 1965, Moyer and Korn 1964, Reymerse and Rizley 1970).† The inter-trial interval also seems to be important in determining the rate of learning. With a short interval the animal could fail to learn that the 'safe' side of the box was indeed safe; responses to that side are rapidly followed by placement in danger again. This importance of safe time in the shuttle-box situation can be contrasted with the effects of increasing inter-trial interval in a situation where safe-place learning is clearly unimportant. Thus, several studies have shown that lever-press avoidance deteriorates, rather than improves, with increasing intervals (Pearl and Fitzgerald 1966, Cole and Fantino 1966, Morris 1973). In this situation avoidance behaviour does not involve going to a safe place; rather, it rests on a response hypothesis.

Two-way active avoidance provides a situation in which there is no safe place; punishment is received in both parts of the box and there are only temporarily safe time periods. The fact that normal animals learn this task with difficulty could reflect their tendency to adopt initially a maladaptive place hypothesis, as seen in a study by Theios and Dunaway (1964). In contrast to the one-way task any manipulation which increases the discriminability of the two sides of the box retards two-way avoidance learning, as does increasing the shock level beyond a certain point (e.g. Denny *et al.* 1969, McAllister, McAllister, and Douglass 1971). These manipulations presumably increase the likelihood that place hypotheses will be used and sharpen the conflict between flight and freezing. Lastly, normal animals initially learn the two-way task in an imbalanced way. That is, they learn to go in one direction rapidly (e.g. A to B), but in the other slowly (B to A). This initial imbalanced learning is quite sensitive to variations in inter-trial intervals spent in the two parts of the box (Weisman *et al.* 1967), but does not seem related to the CS in the situation (Morris 1973). Thus, it would seem to reflect safe-place tendencies which must be overcome before adequate two-way avoidance can be manifested.

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*Testa (1974) has discussed the importance of locations in avoidance learning, but in a way we find both incomprehensible and empty of theoretical significance.

** We have attempted a limited redefinition of the avoidance and punishment literature, particularly as it applies to studies of animals with hippocampal damage, in a recent paper (Black et al. 1977). Our discussion there focuses on the same data in a somewhat different way than in the present chapter.

† Differentiating the safe and dangerous areas in a one-way escape task also facilitates learning (Franchina el al. 1973).

In sum we are suggesting that safe-place hypotheses are predominant in normal animals and that the ease with which they learn tasks favouring the use of SSDR's such as flight or freezing reflects this predominance. Learning in situations where such hypotheses are adaptive is sensitive to those environmental variables which enhance the ability of the animal to differentiate the safe place from dangerous items or places. On the other hand, learning in situations where safe-place hypotheses are not adaptive requires the mediation of other hypotheses and the concomitant influence of other variables, such as the salience of the CS, is seen.

Both place and taxon hypotheses can involve either flight or freezing. The use of a place hypothesis in an avoidance situation cannot be determined by the form of the behaviour chosen, but rather by its flexibility and the nature of the variables to which it is sensitive. In order to determine precisely how an animal solves a given avoidance task one must go beyond the rate of learning to a consideration of the variables in the learning situation which affect performance.

This analysis of avoidance, based almost entirely on an examination of one-way and two-way shuttle avoidance, provides us with a view of the various strategies open to animals in threatening situations. We can now turn to a discussion of the effects of hippocampal lesions upon reactions to various threats and subsequent coping behaviours. We shall extend our analysis of normal performance to other aversive learning situations, including passive avoidance, in the context of this discussion.

8.2. The effect of hippocampal lesions on responses to threat

8.2.1. IMMEDIATE REACTIONS TO PAIN AND THREAT

Hippocampal rats react to shock in much the same fashion as do normal rats. The immediate (unconditioned) reaction includes a rapid flinching response (the flexion reflex), followed by somato-motor responses varying in nature and extent with the intensity of the stimulus (cf. Myer 1971). At low intensities shock elicits primarily flinching behaviour (Muenzinger and Mize 1933), as intensity increases jumping becomes prevalent (Kimble 1955, Trabasso and Thompson 1962), while at even higher intensities (beyond 1.0 mA) locomotor activity, particularly running, becomes probable (Campbell and Teghtsoonian 1958, Goodman et al. 1966). While the occurrence of these reactions is influenced by features of shock other than its intensity, such as its consistency, we limit our discussion here to intensity primarily because this is the only parameter of shock that has been studied in hippocampal animals.

Thresholds to the initial reaction to shock have been reported to be unchanged in hippocampal rats (Schlosberg, Johnson, and Lash 1969), as have changes in heart rate consequent upon moderate shock (Caul *et al.* 1969).* However, hippocampal rats over-react to intense shock, both behaviourally (Schlosberg *et al.* 1969) and in terms of heart-rate changes (Jarrard and Korn 1969). The latter effect likely depends on the former (e.g. Obrist, Sutterer, and Howard 1972), and one could characterize the changes in hippocampal animals as involving abnormally strong somato-motor reactions, particularly running, in reaction to intense aversive items.** Of course, we have already noted that much the same exaggerated responsiveness is seen in these animals with ostensibly neutral stimuli (pp. 257-9), and these phenomena could represent an underlying hyper-responsiveness of all sensory systems.

The reactions of hippocampal animals in the period after shock and/or in the face of threatening items, have been studied in several experiments. In Blanchard, Blanchard, and Fial (1970) rats were shocked with a prod, and their reactions to its subsequent re-introduction (without shock) were assessed. Both control and hippocampal rats decreased their activity after being shocked when confronted with the prod; the decrease in the control rats was greater than that seen in the lesioned rats. However, the latter actively avoided this threatening item significantly *more* than did the former. Thus, reactions to threatening items are not reduced in any simple way in hippocampal animals. In a subsequent study in the same paper the authors tested the reactions of rats to shock in an environment devoid of any discriminable item and from which escape was not possible. In this situation hippocampal rats froze less than normal rats. In so far as freezing reflects a response to a threatening place in this task, the deficit in hippocampal rats implies that fear of dangerous places is diminished or absent in these animals. Though comparisons between studies are to be made with caution, one can take, from a study by Blanchard *et al.* (1968a), a non-shocked control group tested in virtually the same fashion as the hippocampal group in the Blanchard, Blanchard, and Fial (1970, experiment 2) study. This control group demonstrated freezing scores quite similar to those seen in the hippocampal rats, and this suggests that the lesioned animals were simply not responding to the dangerous place.

Both the Kim *et al.* (1971b) and the Blanchard and Blanchard (1972b)

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* Both Blanchard and Fial (1968) and Eichelman (1971) purport to show that hippocampal rats are more sensitive to low-level shock than are normal rats. However, neither study clearly supports its claim. In the first study the hippocampal lesion group *did* have a lower jump threshold than its control group. However, this control group had a higher threshold than all the other groups in the study, including two other control groups with cortical damage only. The hippocampal lesion group differed only from its own, seemingly aberrant, control. In the second study the lesion group had a jump flinch threshold that was significantly lower than that for normal animals but was not different from that of cortically damaged control animals.

** These observations are of some importance because shock is often used in behavioural studies, and an exaggerated reaction to intense shock could form the basis for 'deficits' in lesioned animals. Thus, hippocampal rats can fail on certain problems when shock avoidance is used as the motivation, while succeeding on the same problems when food reward is used. Similarly, hippocampal rats have been shown to fail on a pattern-discrimination task at high shock levels while succeeding at lower shock levels (Musty 1970). The exaggerated reaction to shock in these studies typically manifests itself in fixated response strategies which interfere with discrimination.

studies were concerned with the reaction of hippocampal rats to cats. In the first study hippocampal rats were found to move about more in the presence of an inescapable cat than did normal rats; they also ate and drank significantly more than did the normals.[*] Blanchard and Blanchard included a condition in their study in which the rats could escape the cat. They replicated the Kim *et al.* result of increased movement in the face of the inescapable cat, but also showed that the hippocampal rats would escape *more* rapidly than would the normal rats if given the chance to do so. Thus, the greater activity of hippocampal rats in the presence of an inescapable cat cannot reflect decreased fear *per se,* as Kim *et al.* suggested.

In both studies the hippocampal rats were less active in the presence of the cat than they were in its absence. As we have already noted, the immediate immobility elicited in normal rats by a cat tends to increase over time, and some immobility persists after removal of the cat. In hippocampal rats, on the other hand, immobility scores do not change during the time the cat is present (Blanchard and Blanchard 1972b). There does not appear to be a development of the notion of a dangerous place through association with the cat.[**]

It is noteworthy that flight from the cat in this study, and from the shock prod in Blanchard, Blanchard, and Fial (1970), was greater in hippocampal than it was in normal rats. Blanchard and Blanchard (1971) suggested that the conflict between flight and freezing diminishes the ability of normal animals to flee. The absence of that part of immobility related to the threatening place could account for the superior flight performance of the hippocampals.

Additional evidence bearing on the problem of reactions to threatening items comes from studies of conditioned fear (cf. McAllister and McAllister 1971, for a review of the normal data). Estes and Skinner (1941) reported an aversive conditioning procedure which involved superimposing a CS and an unavoidable shock upon a stable appetitive behaviour, in their case lever pressing for water. The normal animal in this situation rapidly inhibits its appetitive behaviour during the CS preceding the shock and typically freezes instead; this immobility in response to an inescapable threatening item has been termed *conditioned suppression* or the *conditioned emotional response (CER).* A number of investigators quote Brady's (1958) assertion that hippocampal damage causes deficits in the CER and assume that there is a basic defect in fear conditioning (e.g. Olton and Isaacson 1969). However, in that report Brady specifically

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[*] Nonneman and Kolb (1974) have reported reduced fear reactions in hippocampal cats in the presence of another, threatening, cat. The lesioned cats mostly ignored the other cat, going directly to food. If chased, however, the lesioned cats showed flight.

[**] A study by Glickman and Morrison (1969) showed this absence of place fear in an elegant way. Control and hippocampal mice were put into single cages with the doors open, such that they had access to an area containing a predator (owl). The lesioned mice were significantly more likely to be eaten than were the controls, as well as being more active.

mentions that his result should be interpreted with caution, as histology was not yet available for his lesioned rats. No further mention of these data has appeared, and in view of subsequent demonstrations that other limbic lesions can lead to deficits in the CER (e.g. the amygdala, Goddard 1964) we feel that these data are insufficient. Several later studies of the conditioned suppression of drinking, a paradigm related to the lever-press CER, demonstrated adequate learning in hippocampal rats (Kaplan 1968, Nadel 1968, McGowan, Hankins and Garcia 1972).[*]

Another method of testing conditioned fear involves measuring flight from a CS previously associated with punishment in a different situation. McAllister and McAllister (1971) point out that this technique provides an

> 'uncontaminated index of fear and allows a clear distinction to be made between the conditioning and the measurement of fear' (p. 108-109).

That is, punishment is not received in the testing situation, and any flight from the CS must be a result of the fear-conditioning procedure. Goldstein (1965) tested hippocampal rats in such a situation and found that they learned the test response of jumping a hurdle to escape the CS almost as well as did control rats. Finally, in an escape-from-fear paradigm, where fear was established in the same test chamber as escape was assessed, rats with hippocampal damage escaped quite well when confronted with the CS previously paired with shock (Haggbloom, Bruner, and Bayer 1974).[**]

These studies confirm the impression that the hippocampal animal treats threatening items in a relatively normal manner–fleeing or freezing in accordance with the utility of these behaviours in the particular situation. Contrary to what has been suggested by numerous writers, there is not a unitary decrease in freezing; that portion elicited by threatening items seems intact in hippocampal animals. However, freezing is not seen in threatening places. A 'freezing deficit' model makes sense only when viewed in conjunction with the separation between places and items; both elicit freezing in the normal animal while only the latter does in hippocampals.

As we have noted, animals sometimes react to painful stimulation with aggression; there is evidence that hippocampal lesions affect these behaviours. There is a clear-cut reduction in shock-induced fighting after hippocampal lesions in rats (Blanchard and Blanchard 1968b, Blanchard, Blanchard, and Fial 1970, Eichelman 1971, Kim *et al.* 1971b, Kolb and

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[*] McGowan et al. found deficits with ventral, but not dorsal, lesions. Nadel found normal learning with either lesion. See Freeman, Mikulka, and D'Auteuil (1974) and Molino (1975) for partially conflicting reports.

[**] Escape in the damaged rats was better than normal, which might reflect the fact that normal rats, during fear conditioning, tend to freeze, and this conflicts with subsequent escape. Similarly, pre-shock can retard acquisition of one-way active avoidance in normal, but not hippocampal, rats (Haggbloom et al. 1974).

Nonneman 1974, De Castro and Marrone 1974)[*] and this seems related to the fact that these animals less often assume the 'boxing stance' typical of fighting between rats (Blanchard, Blanchard, and Fial 1970).[**] However, this latter study also showed that aggression aimed at a moving electrified prod was *increased* in hippocampal rats. Blanchard, Blanchard, and Fial suggest that their results

> 'offer little support for any hypothesis that hippocampal lesions either enhance or attenuate a unitary aggression mechanism' (p. 101).

Rather, they feel that changes in aggression after the lesion relate to a basic effect of the lesion upon the ability of the animal to remain immobile. While hippocampal rats fight less, hippocampal gerbils fight *more* than do normal gerbils, even though they have difficulty adopting the side-by-side inactive posture which often precedes fighting in this species (Glickman *et al.* 1970). Taken together, these findings indicate that hippocampal lesions do not directly influence aggressive behaviour but can affect various behavioural components often involved in aggression. Depending upon the topology of the particular aggressive behaviour under study these changes could result in either more or less fighting.

The results of all those studies concerned with reactions to threatening places and items suggest that fundamental mechanisms underlying what has been called fear are not disrupted by hippocampal lesions. Rats with such damage flee threatening items if possible, and crouch in their presence when escape is not possible. Thus, the *reaction to threatening items, whether it involves an inactive or active behaviour, remains intact in hippocampal rats.* On the other hand, *there is evidence that these animals do not react to threatening places.* This distinction between threatening places and items is also seen in avoidance learning situations, to which we shall now turn our attention.

8.2.2. LEARNING BASED ON AVERSION

8.2.2(a). Active avoidance. We shall discuss three types of experiments under this heading: *(a)* one-way active avoidance; *(b)* two-way active avoidance; (c) active avoidance involving some manipulation within the situation.

One-way active avoidance. The results of experiments concerned with one-way active avoidance are given in Table A21; in about one-third of these studies deficits were seen in the lesioned animals. The observed pattern of results might reflect procedural differences between the studies involving manipulations of parameters to which the lesioned and normal animals were differentially sensitive. In particular, various manipulations of the CS seem to effect these animals differently.

With a few exceptions the studies listed in Table A21 conform to a pattern; those studies using a CS failed to produce a deficit, while those not using a CS produced a deficit. This implies that the hippocampal animals were uniquely dependent upon specific cues in this situation; this would be expected if one assumed that they could not use place hypotheses. Two studies by Olton and Isaacson (1968a, 1969) provide evidence for these assertions. In the first study the effects of pre-habituation to the CS were measured; in the second the effects of prior CS-UCS (inescapable) pairings were measured. In both cases the performance of the hippocampal, but not the normal, animals was markedly affected by these treatments. Pre-habituation to the CS retarded learning while prior CS-UCS pairings profoundly improved it. Plunkett (personal communication) has shown that hippocampal rats tested in this situation develop a sharp generalization gradient to the CS; that is, they are under strong stimulus control.

These observations suggest that one-way active avoidance can be learned with the use of either place or taxon hypotheses, and that normal and hippocampal animals could solve the task in different ways. We have completed a study which directly tested this possibility (Oetliker 1972). Rats were trained on a one-way active avoidance task in a box with transparent plastic side walls and wooden end walls which could accommodate cardboard (black or white) inserts. There was no specific CS and the UCS, electric shock through the grid floor, was delivered through an automatic programme set to give a brief shock every 5 s. Rats were trained to a relatively weak criterion of learning (8 correct avoidances out of 10 trials) in the hope that this would maximize their chances of using a place hypothesis. Both control and lesioned rats reached this criterion within 20 trials, and there was no reliable difference between the groups on this measure. At this point a series of probe trials was instituted. On each day rats received six trials; the first two and last two were 'normal' trials, identical to those given during initial learning, while the middle two constituted the probes. On each probe trial some change was made in the situation. Either the rat was started in the previously safe area, or the black and white cardboard inserts were interchanged, or the entire box was rotated, and so on. In some probe conditions several changes were made simultaneously. The results of these trials were clear; normal animals behaved almost entirely on the basis of where they were put in the box. When put on the safe side they stayed put; when started on the dangerous side they ran. Thus, they seemed to be using a place hypothesis which

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[*] De Castro and Marrone (1974) showed that shock-induced fighting was diminished in fornix-lesioned rats, but only if they had not had pre-operative experience in the aggression situation. Unfortunately, they did not comment on the behaviour of pre-operatively trained rats.

[**] Peters and Brunner (1976) have shown that rats with hippocampal granule cell loss, induced by X-irradiation, 'box' more than controls when intact intruder rats were placed into their home cages. They did not, however, fight more often. This study differs from the others cited above in that fighting was not shock induced.

could be fulfilled with either an active or a passive response.* In fact the only probe condition which altered this pattern of performance involved completely surrounding the apparatus with a black curtain, effectively removing almost all of the extra-apparatus cues. Under this condition the normal animals froze on both sides of the box. This result suggests that they were using extra-box cues to support their place hypothesis, and that in the absence of this information they were unable to determine the safe place and froze *in situ.*

Lesioned rats, on the other hand, did not respond in a way which could be characterized as dependent upon place hypotheses. They often ran when put on the safe side and equally often remained still when put on the dangerous side. It was not possible in this study to determine exactly which cues the lesioned rats were using, though it seems likely that they were not using extra-box information. When the entire box was surrounded by curtains and the apparatus rotated by 180°, these rats ran from 'safety' and remained in 'danger' as defined by the position of these areas in the room. This behaviour, however, was completely appropriate in terms of the intra-box cues available to the animals, which had been reversed during the rotation.

The results of this study, in conjunction with earlier work, indicate that hippocampal animals can solve one-way active avoidance tasks, but that they cannot use place hypotheses to do so. Deficits should appear when normal animals are favoured through their use of a safe-place hypothesis.

Two-way active avoidance. As Table A22 shows, hippocampal animals are usually superior to normal animals at two-way active avoidance. At first glance this facilitation following hippocampal damage can be associated with the absence of place hypotheses. Such hypotheses, as we have noted, are inappropriate in the two-way task, and the animal must disregard them in order to find the correct solution. Lacking such hypotheses, the hippocampal animal would have an immediate advantage in this situation. This is consistent with the fact that CS manipulations, which strongly affect normal performance in two-way active avoidance, have little effect on hippocampal performance (Olton and Isaacson 1968a, Ackil *et al*. 1969). Further, Plunkett (personal communication) has shown that hippocampal rats have a flat generalization gradient to the CS in this task, in contrast to what is the case in one-way active avoidance. While normal rats use the CS to help overcome their reliance on place hypotheses, the hippocampal rats can ignore it. Finally, it has been shown that normal animals learn to go in the two directions at an unequal rate, while hippocampal animals learn to go in both directions at about the same rate (Papsdorf and Woodruff 1970, in rabbits, Olton and Isaacson 1968a, in rats). This was shown by computing correlations between avoidances in the two directions: normal animals had low correlations; hippocampal animals had quite high correlations.

There is one particular aspect of hippocampal performance in this situation which does require discussion. A number of studies have shown that lesioned animals are considerably more active between trials than are normal animals. This increased rate of inter-trial responding could be responsible for the facilitation seen after lesions. Thus, Schlosberg *et al.* (1969) have shown that hippocampal rats have an abnormally strong tendency to run in response to unsignalled shocks, while Lovely *et al.* (1971) have shown that hippocampal rats run more to stimuli which predict shock, even in the absence of an avoidance contingency. The latter authors conclude that the

> 'facilitated CAR performance is simply a consequence of this altered responsivity to electric shock' (p. 349).

The idea that facilitation in two-way avoidance after hippocampal lesions is due to an increased responsiveness to the CS, or shock, would make it unnecessary to postulate the absence of place hypotheses. While it is clear that increases in inter-trial responding often correlate with superior performance in hippocampal animals there are reasons for assuming that this activity change is not fundamentally responsible for the superior performance, but might instead result from the action of the same underlying change.

The basic question concerns why hippocampal animals run more in response to shock or to stimuli which have been paired with shock. This is the case not only in the two-way avoidance studies, but also in the various studies by the Blanchards and their colleagues discussed above. This increase in running appears related to a shift in the normal flight/freezing continuum, a shift we have identified with the loss of that part of freezing related to threatening places. While it seems certain that normal animals freeze more in most two-way avoidance studies than do hippocampal animals, it is not the case that this indicates a higher level of fear on the part of the normal animals. Antelman and Brown (1972) reported that hippocampal animals were, if anything, more afraid of the CS than were normal animals. If the CS from their two-way situation was introduced into a new situation, where the animals were engaged in an appetitive task, the hippocampal rats suppressed their ongoing behaviour to a greater-than-normal extent.* In fact, Antelman and Brown suggest that

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* The distinction between active and passive avoidance breaks down in the face of results such as these. The normal animals seek out a safe place and will use either active or passive behaviours to fulfill this quest. This becomes important when we consider so-called passive avoidance tasks (pp. 311-15).

* This result suggests that hippocampal animals learn something about the CS, in opposition to Plunkett's data on generalization (p. 308). Such data demonstrate the need for multiple measures in assessing the importance of procedural factors.

> 'animals with lesions of the hippocampus were more fearful than control animals' (p. 15).[*]

Thus, the decreases seen in freezing, and the concomitant increases in inter-trial responding, cannot be due simply to a decreased fear of the CS. We would suggest that it is possible that hippocampals move more in this situation because of the absence of place fear. Thus the increase in inter-trial responding rests directly on the absence of place hypotheses. It shows up because the animals have no reason to inhibit it. On the other hand, it cannot be the cause of the facilitation in two-way avoidance learning; while this facilitation is usually associated with increased responding this association is not inevitable. Ackil *et al.* (1969) have shown that pre-habituation to the CS decreases the number of spontaneous running responses in the hippocampals to a relatively normal level but does not remove their superior learning. Similarly, Ross, Grossman, and Grossman (1975) have dissociated the two in fornical rats. We conclude from this that the hippocampal superiority in the two-way avoidance situation is due to several factors, but that the basic underlying cause is the absence of place hypotheses. Given the absence of place fear hippocampal animals are more likely to be active and consequently more likely to 'find' the correct hypothesis.[**]

Active avoidance requiring non-flight manipulations. The final category of active avoidance tasks to be considered concerns those in which flight is not a useful hypothesis. Avoidance of shock demands the acquisition of some other response, such as lever pressing. As we have noted, such tasks are acquired only with considerable difficulty in normal animals.

Perhaps the most common variety of such avoidance tasks is the so-called *Sidman avoidance.* Here, the animal is placed in an operant chamber containing a lever or other manipulandum. Each response to the manipulandum delays the onset of shock for a fixed period of time. This task can be set up in a number of ways: the presence or absence of a CS signalling the oncoming shock is one important variable; the prior experience of the animals in similar situations seems to be another.

Acquisition of operant avoidance can be normal in hippocampal rats (Ellen and Wilson 1963, Micco and Schwartz 1971) or under some circumstances better than normal (Duncan and Duncan 1971, Schmaltz and Giulian 1972). In these latter studies there was the suggestion that the superiority of the hippocampal rats involved their being relatively more likely to respond to the CS with a lever press. Again, we find the hippocampal animals to be unusually tied to the threatening cue.

The only report of a deficit in hippocampal animals in an operant avoidance situation concerns the case where the animals had prior training on a task which required a response that competed with the avoidance response. Thus, Ellen and Wilson (1963) showed that pre-training on a lever-holding shock-escape task interfered with subsequent learning of the lever-pressing shock-avoidance task. This interference from prior operant training is a common feature of hippocampal behaviour in these situations, as we shall see in the next chapter (pp. 320-1). It does not appear to be related specifically to the avoidance contingencies in the Ellen and Wilson study.

8.2.2(b). Passive avoidance. Passive avoidance is a term applied to a class of tasks in which animals must refrain from making certain experimenter-defined responses; failure to refrain results in punishment. The use of the term *passive,* as we have already noted, carries with it the implicit assumption that the best solution in such situations lies in immobility or simple non-responding. Thus, Blanchard and Blanchard (1969b) state that

> 'a "one-trial conditioning" hypothesis for crouching can predict one-trial passive avoidance without any assumption that the S has learned to inhibit a specific response' (p. 374).

While it is clear that rats can learn to remain immobile in order to avoid punishment (e.g. Brener and Goesling 1970), it is not at all clear that immobility *per se* is the preferred mode of solution in most commonly used passive avoidance paradigms. Several investigators (Liss 1968, Olton 1973) have pointed out that animals do not necessarily solve such tasks in a particularly passive way.

We feel that the distinction between active and passive avoidance in the literature is an insignificant one in comparison with the differences between paradigms requiring the use of specific behavioural hypotheses. As our study of one-way active avoidance suggests, animals which have learned actively to avoid places or items will passively avoid them as well (pp. 307-8). What appears to be important is the identification of the threat, rather than the particular response requirement. Thus, prior training on passive avoidance can facilitate subsequent active avoidance (Bresnahan and Riccio 1970), a result that would be impossible if the learning of particular responses were essential in either task. Only when the situation is so constructed as to eliminate the use of either place or guidance hypotheses will an animal resort to the simple motor-response inhibition

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[*] We have already discussed certain aspects of this abnormally strong fear of items related to shock in hippocampals (pp. 303-6). It is worth noting further that in most of these situations the effect of the threatening item was assessed in a previously safe place. Normal animals might initially regard such an item with less trepidation than if it were presented in its original context. Hippocampals, on the other hand, do not seem to be affected by the context within which threat occurs. This failure of context effects in the lesioned animals suggests that the transfer of incentive effects from one situation to another might consistently appear stronger in lesioned rats.

[**] A report by Lover (1975) concerning two-way active avoidance would not have been predicted by this interpretation. He showed that hvpophysectomy removed the facilitation elicited by hippocampal damage (see our discussion of the pituitary-adrenal system (pp. 357-62)).

learning which the passive avoidance terminology implies.

These 'passive' avoidance paradigms require that the behaviour to be punished be one with a reasonable probability of occurrence. This can either be the result of some prior training or of some inherent tendency on the part of the animal. A number of paradigms based on the use of these kinds of behaviours have been devised, and it is surely the case that they do not all reflect the action of the same underlying mechanisms. In particular, tasks vary in the extent to which they depend upon the avoidance of threatening places or items. Further, within a given paradigm performance can usually be assessed in any of several ways, each of which might reflect the action of different mediating factors. Most passive avoidance tasks can be viewed as situations in which place hypotheses are not essential. One would expect, therefore, that hippocampal animals would have no specific difficulty with such tasks. Before turning to the evidence from lesion studies we shall briefly describe some of the more commonly used paradigms.

Procedures for passive avoidance. One major class of passive avoidance paradigms can be labelled *approach-avoidance.* In such tasks the animal is initially trained on an approach task, such as running to obtain food or water, and is subsequently punished for making that response. In most cases this involves the use of a runway or similar apparatus, leading to a goal box with a food or water dish. Punishment in this situation typically involves giving the animal a grid shock while it is consuming the reward; alternatively, shock can be administered through the dish containing the reward. Thus, punishment is usually associated both with the place and the item. Passive avoidance can then be measured in several ways: latency to contact the item; latency to enter the goal area; latency to leave the start area. These measures might reflect the action of different systems. Another variant in this situation concerns the number of punishments. In most cases shock is applied only once, and learning of passive avoidance is assessed under what are essentially extinction conditions. Once the animal returns to the source of punishment (and is not shocked) the situation has changed and one is no longer measuring passive avoidance. In other cases shock is applied on several trials, and the number of shocks taken is a more valid measure of avoidance behaviour.

A related class of passive avoidance tasks involves the use of essentially untrained response tendencies which are highly likely for the species being tested. These typically rely on placing the animal in a situation which is inherently fearful and then punishing its attempts to withdraw. Thus, most animals placed on a raised platform in the middle of an exposed field will step down from the platform in an attempt to move towards the perimeter. This step-down response can be punished and passive avoidance measured as a function of subsequent step-down latencies. Similarly, animals put out on a perch with a considerable drop to the floor, or in a large, open box, will rapidly move into a smaller box if possible. This tendency can be punished and subsequent step-through latencies measured as a reflection of passive avoidance. These tasks involve something like an approach-avoidance conflict, where one component is learned and the other unlearned.

Another commonly used paradigm involves training animals on one-way or two-way active avoidance and then punishing these active responses. In other words, when placed in the start area the animal must now stay put to avoid punishment; running to the other side will lead to shock. This task has been called *shock-shock* passive avoidance. There are other variants that one could mention here; the reader is referred to Black *et al.* (1977) for further discussion of these and for a more detailed analysis of the performance of hippocampal animals in these situations.

Hippocampal lesions and passive avoidance. The results of those studies involving passive avoidance learning in hippocampal animals are given in Table A23. The most striking aspect of these data is that deficits in these tasks, often assumed to be invariably associated with hippocampal damage, have actually been demonstrated in less than half the published studies. Those paradigms which can be described as independent of place hypotheses almost invariably produce no defect in hippocampals. This includes such paradigms as step-down and step-through passive avoidance, as well as the punishment of some consummatory response. Mixed results have been reported for many of the other paradigms. In large measure this confusion can be attributed to differences in the way passive avoidance was measured in the various studies. The data are consistent with the view that hippocampal animals do not passively avoid places devoid of distinctive items, while they do avoid items.

Nonneman and Isaacson (1973) tested cats on an approach-avoidance task. Their animals were trained to approach food and were then shocked for touching the food dish. On subsequent trials their hippocampal cats showed a passive avoidance deficit in terms of their latency to start running towards the area containing the food dish. However, these cats did not, in fact, touch the dish itself more rapidly than did the control animals. This split between immobility in the face of a threatening place and avoidance of a previously punished item was seen in nearly all of the studies performed in this manner. When the measure of avoidance was the latency to move into a dangerous place the hippocampal animals appeared to be retarded; when the measure was contact with the threatening item no defect appeared. Such results were reported by Kimble, Kirkby, and Stein (1966), Andy *et al*. (1967), Brunner and Rossi (1969), and Fried (1971).

A more direct test of the place-item distinction stressed here is available in the study by Blanchard, Blanchard, and Fial (1970). In their Experiment

2, rats were tested for the passive avoidance of what was described as either a highly discriminable stimulus (an electrifiable metal box) or a poorly discriminable stimulus (a portion of the grid). This latter condition was biased towards the use of a place hypothesis. Hippocampal rats avoided the metal box as well as did normal rats but tended to receive more shocks from the grid, though this difference was not statistically reliable. In Experiment 3 of this paper the same rats were tested on another passive avoidance task involving a threatening place and in this case the hippocampals were significantly impaired.

The authors of this report took the position that the hippocampal defect in passive avoidance, and indeed in all avoidance situations, was a function of altered defensive crouching behaviour. At a descriptive level this notion contains a good measure of truth. After raising the question

> 'What function of the hippocampus, disrupted by hippocampal damage, is normally involved in the elicitation or maintenance of defensive immobility' (Blanchard, Blanchard, and Fial 1970, p. 101),

they proceeded to rule out the possibility that lesioned rats have higher pain thresholds, as well as the possibility that the hippocampus is involved in the association between neutral and painful stimuli. They then pointed out that avoidance of a specific aversive item might be controlled by brain processes different from those controlling immobility, and that these two reactions (flight and freezing) could be elicited by differentiable conditions.

We have previously discussed the question of flight and freezing (pp. 294-302) and feel that the analysis given by Blanchard, Blanchard, and Fial is correct up to a point; however, it is important to extend their account to distinguish those different conditions responsible for flight and freezing. Freezing is most often a response to an inescapable place, though it can occur in the presence of an inescapable threatening item. Most studies focus on the former, so that there has been a conflating of freezing and place-dependent threats that is misleading. The fact that immobility reactions seem to be defective in hippocampal animals in most of the studies reported to date could mean either that they cannot react appropriately to threatening places or that they cannot emit immobility responses. We believe the former interpretation is more likely and predict that lesioned animals would freeze appropriately when such behaviour was necessary for avoidance of an object.

Analysis of the passive avoidance data in terms of the place–item distinction imposes regularity on what otherwise appears to be a rather messy set of data. It is difficult to extend this analysis any further, as it is often impossible to know from the published reports the extent to which different behavioural strategies are tapped by different paradigms. Only in those situations requiring place hypotheses, or with the use of measures

reflecting place-learning mechanisms, will a deficit appear in hippocampal animals. The passive avoidance of threatening items is intact in these animals.

One further paradigm, conditioned taste aversion, supports this analysis. This task involves poisoning a novel food and measuring the animal's subsequent avoidance of that food. We noted earlier (p. 292) that the learned aversion and learned safety seen in this situation represented clear examples of the way in which items are tagged with some motivational valence through experience. The effects of hippocampal lesions on this type of learning have now been assessed in eight studies (Table A23). Miller, Elkins, and Peacock (1971) reported that rats with extensive hippocampal ablations failed to show conditioned aversion, but the effect was quite small in their control animals as well. Best and Orr (1973) reported deficits only after dorsal hippocampal lesions, while McGowan *et al.* (1972) reported no deficits with either dorsal or ventral lesions. Five subsequent studies all reported no deficits in hippocampal animals, in terms of the acquisition, magnitude, and extinction of conditioned aversion. Taken together, these studies indicate that the hippocampus is not integral to this type of passive avoidance learning.

9

Operants: the limited role of the locale system

In the previous chapters of this section of the book evidence was marshalled in support of the view that hippocampal animals lack exploratory mechanisms and the ability to construct locale space; consequently they cannot utilize place hypotheses. In the absence of the locale system hippocampal animals solve problems with taxon hypotheses, and their behaviour reflects the properties of the taxon systems. Certain experimental situations will primarily reflect the operations of the taxon systems; some examples of this are classical conditioning and most operant training procedures. In these and other taxon-biased situations we would expect the hippocampal animal to perform at least as well as the intact animal, though perhaps in a different manner. In this chapter we consider a variety of experimental procedures which extend this analysis.

9.1. Classical conditioning and incentive effects

What little evidence is available suggests that hippocampal lesions do not affect the learning of classically conditioned responses. Thus, Allen (1940) showed that hippocampal dogs acquired an olfactory conditioned forelimb response normally, while Isaacson and Douglas (cited by Douglas 1972) showed that hippocampal dogs acquire a forelimb flexion response quite rapidly. Schmaltz and Theios (1972) have shown that hippocampal rabbits acquire a nictitating membrane response more rapidly than do normal rabbits. Finally, Pigareva (1974) reported normal acquisition of a conditioned alimentary reflex with either 100 per cent or 50 per cent reward.* **

A variation of classical conditioning techniques involves what has been termed *incentive motivational* conditioning. When an initially neutral stimulus is paired with reward (or punishment) this stimulus acquires general motivational properties and can then function as an incentive (cf. Bindra 1968). Such incentive stimuli, when introduced into a new situation, often have marked effects upon an animal's behaviour. We have already discussed the way in which stimuli associated with pain exert effects upon an animal's behaviour in other situations; this is one example of an incentive effect. The fact that incentives transfer from one environment to another indicates that the locale system is not essential to their effects; this suggests that hippocampal animals should show incentive effects.

Available data support the view that stronger than normal incentive effects are seen in hippocampal animals. Morey and Brown (1972) measured the effect of positive incentive conditioning on general activity and goal-directed behaviour. During the conditioning process hippocampal rats were significantly more locomotor in response to the incentive stimulus than were intact rats; they did not, however, show more goal-directed behaviour. Antelman and Brown (1972), in the course of a study on two-way active avoidance described above (p. 309), tested the effect of the avoidance CS on licking behaviour in another situation. They found that this negative incentive stimulus suppressed licking behaviour to a greater extent in the hippocampals than it did in the controls. These studies indicate that there is no basic defect in the response of hippocampal animals to incentive stimuli, either positive or negative.

On the other hand, Micco and Schwartz (1971) presented evidence which suggested to them that reactions to conditioned incentives could be defective in hippocampals when inhibition was required. Rats were trained on a lever-press Sidman avoidance task. Following this, in a separate apparatus, positive and negative incentive stimuli (tones) were established. The effects of the introduction of these tones upon avoidance performance were then assessed. In both intact and hippocampal rats the negative incentive stimulus (previously paired with shock) led to an increase in the rate of lever pressing; the positive incentive stimulus decreased lever pressing in the intact, but not the hippocampal, rats. The results summarized in the previous paragraph plus studies involving CER conditioning (see pp. 304-5) indicate that the explanation offered by Micco and Schwartz is highly unlikely; in other experiments hippocampal animals could inhibit responding quite well when confronted with negative incentive stimuli. One possible source of the discrepancy lies in the nature of the behaviour involved. Micco and Schwartz used an operantly trained response, while Antelman and Brown (1972) and Nadel (1968) used a consummatory response (licking at a spout).

9.2. Operant tasks

In the typical operant task the animal is placed in a relatively restricted environment containing one or more manipulanda; responses to these manipulanda are rewarded in some fashion. A typical operant chamber for rats, the Skinner box, is simply an enclosed box containing a lever and a recessed food well in most cases. Presses on the lever are rewarded by

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* Deficits were seen when reward was only given on 33 or 25 per cent of the trials. We shall discuss the partial reinforcement effect in the next chapter.

** Hippocampal lesions can affect the retention of classically conditioned responses (Gralewicz and Strumillo 1971, Gralewicz and Zdankiewicz 1973). The problem of retention is discussed later (pp. 374-7).

delivery of a food pellet through a food magazine. Similar apparatus can be devised for other species. It is important to note that the responses required of the animal in most of these situations are of a very low initial probability of occurrence and the animal must be 'shaped', or specially pre-trained, to perform them.

By their nature most operant tasks minimize the role of the hippocampus in the control of the animal's behaviour, though they do not eliminate it. The impoverished sensory environment of the Skinner box is designed to elicit only a limited amount of exploration. Initially this might help the animal locate the significant features of the box, such as the lever and food well, but very quickly the reward contingencies should strengthen the taxon hypothesis 'press the lever' and locale involvement should be drastically reduced.

It is worth digressing here to describe how the relative insensitivity of operant tasks to hippocampal function is a direct and ineluctable result of Skinner's scientific methodology. We have it on his own word (Skinner 1959) that he is guided in his work solely by the search for order or lawfulness in behaviour:

> 'the notes, data and publications which I have examined do not show that I ever behaved in the manner of man thinking as described by John Stuart Mill or John Dewey or as in reconstructions of scientific behaviour by other philosophers of science. I never faced a Problem which was more than the external problem of finding order. I never attacked a problem by constructing a Hypothesis. I never deduced Theorems or submitted them to Experimental Check . . . Of course, I was working on a basic Assumption–that there was order in behaviour if I could only discover it– but such an assumption is not to be confused with the hypothesis of deductive theory. It is also true that I exercised a certain Selection of Facts, not because of relevance to theory but because one fact was more orderly than another. If I engaged in Experimental Design at all, it was simply to complete or extend some evidence of order already observed' (p. 364).

Since Skinner seeks order not in a theoretical model but rather in surface behaviour, he constantly redesigns his experiments not with a view to understanding behaviour but rather to eliminating the reasons for its variability. This stems from one of his unstated assumptions: that all intra-individual variation in behaviour is due to environmental noise, a chance response to a stimulus. Similarly, inter-individual variation is due to biological and cultural noise. One of the main tenets of our model is that this thesis, underlying so much of present-day psychology, is completely wrong. Instead, we submit that one of the major brain systems controlling behaviour, the hippocampus, is specifically designed to produce variability in behaviour. On this model behaviour cannot be wholly explained on its own level but must be shown to depend upon law-abiding neural systems.

Skinner recognizes this alternative:

> 'It is the function of learning theory to create an imaginary world of law and order and thus to console us for the disorder we observe in behavior itself. Scores on a T-maze or jumping stand hop about from trial to trial almost capriciously. Therefore, we argue that if learning is, as we hope, a continuous and orderly process, it must be occurring in some other system of dimensions—perhaps in the nervous system, or in the mind, or in a conceptual model of behavior' (p. 375).

but rejects it.

> 'When we have achieved a practical control over the organism, theories of behavior lose their point. In representing and managing relevant variables, a conceptual model is useless; we come to grips with behavior itself. When behavior shows order and consistency, we are much less likely to be concerned with physiological or mentalistic causes. A datum emerges which takes the place of theoretical phantasy. In the experimental analysis of behaviour we address ourselves to a subject matter which is not only manifestly the behavior of an individual and hence accessible without the usual statistical aids but also 'objective' and 'actual' without recourse to deductive theorizing' (p. 375).

It is little wonder, then, that Skinner moved very quickly from his original studies on exploratory behaviour to reflexes and ultimately to operant tasks. Although one of his stated principles of research is

> 'when you run onto something interesting, drop everything else and study it' (p. 363),

without a theory or preconceived notion of how animals function he had no means of deciding which things were interesting. One instance is particularly illuminating within the context of our theory. During an alley study Skinner tried to save himself some leg work by getting the animal to return by itself to the start of the alley after each trial. The modified apparatus was essentially a rectangular maze with food always given in one corner (C) after a complete circuit. It worked in principle, but

> 'there was one annoying detail, however. The rat would often wait an inordinately long time at C before starting down the back alley on the next run. There seemed to be no explanation for this' (p. 365).

Having approached so close to the precipice, the quest for explanation, he safely jumps back:

> 'When I timed the delays with a stop watch, however, and plotted them, they seemed to show orderly changes. This was, of course, the kind of thing I was looking for' (p. 365).

The alleys eventually became Skinner boxes, the delays smoothed out, and the important question—why did they delay?—finessed.*

The Skinner box, and most operant procedures employed therein, consequently put few demands upon the locale system. We can now look at the results of studies of operant behaviour in hippocampal animals. In this chapter we shall be concerned primarily with the performance of animals on various operant schedules as such; in the next chapter we shall take up the question of the effects of changes in reward conditions upon behaviour, and the problem of shifts in operant schedules will be considered within that context.

Hippocampal animals have been tested on a wide variety of operant schedules; the results of these studies are given in Table A24. Before considering these in more detail it would be worthwhile to consider the grosser aspects of the behaviour of animals in the operant situation. As we noted above, lever pressing is a low-probability response, at least in rats, and special 'shaping' techniques must be used to generate appropriate performance. Typically, these involve first training the animal to eat or drink, in conjunction with the noise from the reward magazine. Following this, animals are shaped, often by a method of successive approximations, to press the lever for reward. Only when this has been accomplished can the effect of various reward schedules on performance be assessed.

Those few reports which comment upon this early stage all suggest that hippocampal animals react abnormally to the shaping procedures. In the rat, for instance, Schmaltz and Isaacson (1966a) note that

> 'the initial reinforcements have highly differential effects on the animals. The hippocampally lesioned rats take up a position close to the food cup after several reinforcements. The normal and decorticate rats, on the other hand, continue to explore the operant chamber and require many more reinforcements until they are magazine trained' (p. 180),

while Warburton (1972) notes that

> 'during shaping the hippocampal animals showed a marked tendency to remain close to the water nozzle after the first few reinforcements' (p. 351).

Warburton found that this tendency made it difficult to shape lever pressing, while Schmaltz and Isaacson state that

> 'in the majority of cases, the hippocampectomized rats, once magazine trained, were easily shaped to the bar press response. This was due chiefly to the repetitive nature of much of their behavior. Once reinforced for a response, they tended to repeat this response over and over again until another reinforcement was given. In a few instances, however, the repetitive behavior of hippocampectomized animals interfered with the shaping process. Responses became so rigid that it was very difficult to use the successive approximations technique' (p. 180).

Similar findings were reported for the monkey by Jackson and Gergen (1970). They noted that:

> 'during hand training, attempts to use successive approximations became difficult because responses that were initially reinforced were hard to extinguish. These responses quickly became stereotyped, and the animals would frequently perform certain of these responses repetitively in a ritualistic sequence' (p. 544).

Thus, reward appears to have a strong effect upon the hippocampal animal in this situation, leading to repetitive responses to the lever and food or water dish. The intact animal, on the other hand, is more variable in its behaviour, rarely repeating responses. The importance of this difference will become clear in our discussion of the various operant schedules.

9.2.1. CONTINUOUS REINFORCEMENT—CRF

The foregoing discussion of shaping effects applies to an operant schedule where the animal is rewarded for each response to the lever; this is termed a continuous reinforcement (CRF) schedule. In line with the above discussion it is not surprising to note that hippocampal rats can acquire CRF responding more rapidly than can control rats (Schmaltz and Isaacson 1966a, Means, Walker, and Isaacson 1970) and tend to respond more frequently, though this difference is rarely a significant one (cf. Rabe and Haddad 1968, Haddad and Rabe 1969). An important difference between hippocampal and normal rats in the CRF situation was brought out in a recent study by Van Hartesveldt (1973). Her rats were trained with either a large or small reward (0.08 ml or 0.01 ml water); hippocampal rats acquired the response at an equal rate under both reward conditions, while control rats acquired the response as rapidly with the large reward, but were reliably slower with the small reward.*

9.2.2. FIXED RATIO—FR

On this schedule the animal is rewarded for making a fixed number of responses: thus, reward rate and response rate are directly related. Four studies of performance on FR schedules in hippocampal animals have been

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* Why, indeed, did Skinner's rats delay their running? This task appears to be an appetitive counterpart to the two-way active avoidance task (p. 298). There, the situation allows for a conflict between two hypotheses: (1) avoid a dangerous place, and (2) run to avoid shock. In Skinner's situation the conflict lies between similar place and response hypotheses. In a similar study Brown (1946) failed to find any effect of a common start-finish locus on maze performance. However, in this study there was little indication that the rats were utilizing place hypotheses, as both maze rotation and a substantial lateral shift of the maze had little effect upon performance. We would predict that in a properly constructed situation, allowing for conflict between hypotheses, hippocampal animals would perform 'better' than normal animals, failing to show the delay.

* This difference in acquisition rates does *not* imply that the hippocampal rats are insensitive to differences in reward magnitude. In this study, as well as in several others (e.g. Franchina and Brown 1971, Gaffan 1972, Freeman *et al.* 1974) their response rates vary with reward magnitude in a normal way. There is no evidence to suggest that the hippocampus is fundamentally involved in evaluating reward magnitude. We suspect that under the low-reward condition control rats were more likely to explore and thus slower to learn.

reported. Van Hartesveldt (1973) concluded that the data from her study and two earlier ones (Rabe and Haddad 1968, Carey 1969) indicate normal performance by the lesioned rats on FR so long as there is a gradual increase in the FR requirement. Thus, Rabe and Haddad started their rats on CRF and then shifted them to FR-20; they found increased responding in their hippocampal rats, beyond that seen in the control rats. However, Carey (1969) and Van Hartesveldt (1973) shifted their rats from CRF to FR-5, then FR-10, and only gradually increased the response requirements; in these cases the hippocampal rats responded at normal rates. Consistent with this, Rabe and Haddad found that the response rates of hippocampals increased normally when the rats were shifted from FR-20 to FR-30. Finally, a more recent study (Schmaltz, Wolf, and Trejo 1973) also found that FR performance was normal when the ratio was gradually increased. Thus, shifting from CRF to FR-5, FR-10, FR-20, and FR-40 yielded normal response rates. However, shifting to FR-80 and then FR-160 produced abnormal increases in response rates in the hippocampal rats, though they eventually adjusted their response rate to a normal level on the final FR-160 sessions. Thus, FR performance can be normal in hippocampal animals, except when they have been shifted from a schedule with markedly smaller demands. The meaning of this over-reaction will be discussed shortly.

9.2.3. FIXED AND VARIABLE INTERVAL - FI AND VI

On these schedules the animal is rewarded once during a given interval (either fixed or varying about a mean), regardless of the number of responses made during that interval. The FI schedule generates a particular pattern of responding, often referred to as the FI scallop (because of its appearance in cumulative recorder records). Typically, the animal shows a post-reward pause in responding, then starts responding more rapidly as the end of the interval approaches. This pattern does not appear with the VI schedule, which has consequently been of less experimental interest. In the only study utilizing VI with hippocampal animals Jarrard (1965) found increased response rates in his pre-operatively trained lesioned rats.

Four studies of FI performance in hippocampal animals have been reported. Ellen and Powell (1962) reported relatively normal FI performance in their hippocampal animals, with a tendency towards decreased responding. Their lesioned rats developed an FI scallop but responded less frequently during the period just before reward than did controls. On the other hand, it took longer for the hippocampals to develop a marked post-reward pause. Both Beatty and Schwartzbaum (1968) and Haddad and Rabe (1969) report mostly increased response rates for hippocampal rats on FI. Haddad and Rabe included two lesion groups in their study: one with small anterior lesions similar to those used by Ellen and Powell, the other with larger anterior-posterior lesions. The group with small lesions performed normally, in accordance with Ellen and Powell, the group with large lesions had increased response rates.

Analysis of the temporal patterning of responses reveals several differences between hippocampal and control rats. Haddad and Rabe found that their lesioned rats developed a more pronounced FI scallop than did control rats; that is, they responded at an increased rate just prior to the reward. However, they apparently showed no abnormality in the development of a post-reward pause.* Beatty and Schwarzbaum (1968), on the other hand, found a significant retardation in the development of the post-reward pause in their lesioned rats, combined with a less marked increase in terminal rates. In a separate group of lesioned rats these authors investigated FI performance under conditions of non-deprivation. The pattern of increased post-reward responding and normal terminal responding was also evident in these rats, performing for sucrose.

These discrepancies are not easily accounted for. Such factors as the reward employed (Beatty and Schwartzbaum used sucrose, the others water) and the extent of prior CRF training might have been involved in their production. In any case, all of the studies show a disruption of FI performance related in some temporal fashion to the reward, either its prior or its anticipated occurrence.

9.2.4. DIFFERENTIAL REINFORCEMENT OF LOW RATES—DRL

The seeming abnormalities of hippocampal animals on FI schedules, involving aberrant temporal patterning of responses, have been explored more thoroughly using another schedule presumed to require precise patterning of responses. In the DRL schedule the animal must withhold its responses for a fixed period of time before reward becomes available. Responses during this delay interval reset the delay; thus, high rates of responding, or an inability to pattern responses, lead to low rates of reward. A large number of DRL studies have been reported for hippocampal animals, with often confusing results. Before considering the performance of lesioned animals in detail it is worth discussing the nature of normal performance. The DRL schedule is a difficult one for the normal rat. Typically, no more than 50 per cent of the available rewards are obtained, and reward/response ratios rarely go beyond 40 per cent. In the intact rat responses are patterned as follows: there is an immediate post-reward burst, followed by a decline in responding, then by a build-up towards the end of the delay interval. With continued training there is a decrease in short-latency responses which partly accounts for observed improvements. It is clear that it is quite difficult for normal rats to perform well in the standard DRL situation. This difficulty is related to the multiple hypotheses that can be used in this situation.

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* Jackson and Gergen (1970) report the same pattern for the hippocampal monkey.

Responses to the lever are both rewarded and punished; thus, the tendency will exist both to approach and avoid the lever. The intact animal resolves this conflict by using a place or orientation hypothesis to mediate lever pressing and a different hypothesis to avoid the lever during the delay interval. We have already seen that cats in this situation move to the opposite side of the box and lie down during the delay (Bennett 1975, see p. 185). In discussing the 'collateral' behaviour which develops to bridge the DRL delay, Kramer and Rilling (1970) indicate that pigeons, as well, utilize the strategy of moving away from the lever at the inappropriate time. Laties, Weiss, and Weiss (1969) have studied the collateral behaviour of rats, showing that any behaviour which takes the animal away from the lever and keeps it busy will improve DRL performance. The introduction of a cue signalling the termination of the delay period makes such collateral behaviour unnecessary. If we can judge by the change in hippocampal EEG patterns, such a cue alters the cat's delay behaviour (Bennett 1975). In both cat and rat the cue vastly improves DRL performance, yielding up to 70 per cent rewarded responses. Hippocampal animals should be able to generate some form of adequate collateral behaviour, though they could not use a place hypothesis taking them away from the lever. Further, they should benefit from the introduction of the cue.

However, most DRL experiments are preceded by extensive training on CRF. This would have the effect of increasing the strength of the orientation hypothesis mediating lever pressing, perhaps more so in hippocampal than in control animals, as we have seen. As a consequence, such pre-training would make it much harder for the hippocampal animal to find adaptive collateral behaviours strong enough to compete with the hypothesis established during CRF training. In the absence of any possibility of using a place hypothesis, this condition should maximize the hippocampal animal's difficulty with the DRL task. There are two ways in which the lesioned animal could be helped to overcome this difficulty: (1) through the introduction of a cue; (2) through the opportunity to develop strong taxon-based collateral behaviour.

While hippocampal animals are often deficient at DRL there are conditions under which they perform normally. Ellen *et al.* (1973) showed that both extensive lesions *and* extensive pre-training on CRF are necessary for persistent impairments in this task. Earlier studies reporting normal DRL performance in hippocampals utilized either small lesions (e.g. Ellen, Wilson, and Powell 1964) or minimal CRF pretraining (e.g. Schmaltz and Isaacson 1966a). There is a consistent, though apparently not inevitable, relationship between response rate and performance in the DRL situation, and the fact that CRF schedules can elicit increased response rates in hippocampal animals provides a basis for understanding the effect of CRF pre-training on subsequent DRL performance. There are conditions, however, when response rates and performance (in terms of the number of rewards obtained) do not co-vary. Thus, Schmaltz and Isaacson (1968) demonstrated that blinding hippocampal rats reduced response rates but did not noticeably increase reward rate. Schmaltz *et al.* (1973) showed that hippocampal rats trained initially on FR and then switched to DRL did not respond significantly more than did intact rats, but nevertheless received fewer rewards. On the other hand, Nonneman and Isaacson (1973) showed that a deficit in DRL in neonatally lesioned rats only occurred in combination with high CRF rates. Lastly, the addition of a cue signalling the end of the delay interval both decreases response rate and increases reward rate, such that hippocampals perform as well as do normals (Pellegrino and Clapp 1971, Rickert *et al.* 1973) whether or not they have received extensive CRF pre-training.

9.2.5. A SYNTHESIS

The overall picture provided by these operant studies suggests several conclusions: (1) the lever pressing response is one that hippocampal animals acquire rapidly and execute repetitively; (2) this repetitive responding can interfere with performance on certain schedules, particularly those requiring precise temporal patterning of responses; (3) hippocampal animals seem abnormally sensitive to abrupt shifts in reward contingencies; (4) to some extent the deleterious effects of repetitive responding can be ameliorated by the addition of an external cue guiding the animal's responses.

These conclusions suggest to us the following analysis of hippocampal performance in operant situations. Lever pressing itself is easily supported by an orientation hypothesis. This, coupled with the absence of exploration, leads the hippocampal to acquire the basic lever-press routine faster than will the normal and can produce over-responding without loss of rewards in both CRF and FR schedules. Increased responding would not result in fewer rewards in the FI schedule but could result in abnormal temporal patterning of responses. Here, as in the DRL schedule, alternate hypotheses can take the animal away from the lever during those times when responses are either ineffective or actually punished (through an increase in the delay period). That is, we feel that rats cannot 'count time', but rather bridge temporal intervals by engaging in any of a variety of behaviours which fill the required interval. The normal rat can call on behaviours based on place (go to the other side of the box), guidance (do not press unless light is on), or orientation (engage in a sequence of collateral displacement behaviours) hypotheses. The hippocampal rat is restricted to guidance or orientation hypotheses.

We have seen that the provision of a guidance in the DRL schedule enables the hippocampal animal to bridge the delay as well as can the normal animal. Promoting the use of collateral behaviours based on orientation sequences should similarly improve the performance of hippocampal animals.

Preliminary evidence (Van Hartesveldt, personal communication) supports this prediction. There is some evidence available on the use of orientation sequences by lesioned animals in situations other than the DRL. While several early studies suggested that hippocampal animals were deficient at learning such sequences (Kimble and Pribram 1963, Gross, Chorover, and Cohen 1965), later work has demonstrated that these animals learn sequences more rapidly, and better, than do normal animals. For example, Jackson and Strong (1969) have shown that hippocampal rats are superior to intact rats at learning to press up to three levers in various sequences to obtain reward.

We can now turn to a discussion of a mixed group of studies which combine certain features of operant and discriminative behaviour. In some of these tasks locale mechanisms play an important role, while in others they are of miminal importance.

9.3. Delayed response

In this task the animal is typically confronted with several identical food wells, but temporarily prevented from approaching them. One of these wells is then baited with reward and, after some delay, the animal is allowed to respond. Delayed response tasks are seemingly dependent upon locale mechanisms; that is, a food well is baited in sight of the animal, and proper performance requires that the animal remembers the location of that well which was baited and then, when given the opportunity, responds to it. In other words, the animal must use information about *where* to direct its response, rather than to what kind of object it must respond.

There is little doubt that hippocampal damage causes deficits in delayed response; Table A25 gives the results of those studies which have used this task. The suggestion of a deficit was present in early work with monkeys (Mishkin 1954, Orbach, Milner, and Rasmussen 1960). Later work with cats and rats showed profound impairment of delayed response (Karmos and Grastyan 1962, Ungher and Sirian 1970, Niki 1962). However, more recent work, again in monkeys, showed no deficit in delayed response (Mahut 1971). There is little doubt that hippocampal animals remember that *some* food well has been baited for they respond rapidly after the delay interval. Deficits which are reported presumably reflect the animal's inability to distinguish between several identical objects solely in terms of their spatial location. Karmos and Grastyan (1962) tested cats in a situation with food wells on each of three walls of a box, the animal being restrained in a waiting box (with glass wall) attached to the fourth side of the box. A tone presented through the speaker mounted over a food well signalled the availability of food in that well. Hippocampal cats were deficient at delayed response primarily because they responded to the first well that caught their sight upon entering the test area after the delay.

Similar results were reported by Ungher and Sirian (1970). Their fornical cats made errors primarily by responding to the wells which were closest to the restraining box.

In the absence of an ability to remember where the correct well is located, the hippocampal animal could perform adequately by assuming a posture during the delay which 'told' it the location of the correct well. Ungher and Sirian report that fornical cats which used this strategy did manage occasionally to perform adequately. They state that:

> 'after being presented the meat bowl, some cats with fornicotomy and sometimes the controls maintained, over the delay interval, while still in the cage, the bowl-watching posture. This facilitated the delayed response performance in the damaged cats; they seemed to have memorized the cues. If during the delay interval the damaged cat was made to shift from this posture the erroneous responses multiplied ... Diverting maneuvers in controls rarely resulted in erroneous responses' (pp. 177-179).

Mahut (1971) has also suggested that her hippocampal monkeys solved the delayed response task by using a strategy which could override their spatial defect. It seems likely, then, that the spatial component in the delayed response situation is responsible for such defects as are observed in hippocampals.

9.4. Alternation and go–no-go

There are a variety of tasks which involve alternating or interchanging responses in some fashion. We have just discussed one variant, that involving the performance of a sequence of responses, with a complete sequence leading to reward. Available evidence indicates that hippocampal animals are quite capable of learning these sequences. Another, somewhat different, class of tasks typically limits response alternatives to two, and requires that the animal alternate between these in some way. Thus, on successive trials reward could follow a right turn in a T-maze, then a left turn, then a right, and so on. This is simply a rewarded counterpart of the spontaneous alternation task. Similarly, in a two-lever Skinner box successful performance could involve pressing the left lever for reward, then the right lever for another reward, then the left, and so on. In one version of this task cues are not provided, and the animal must alternate on the basis of its memory of the preceding response. In a second version cues are provided as to which lever is to be pressed, and alternation can be achieved simply by responding to the cues. These tasks have typically been labelled *spatial delayed alternation,* as they involve changing responses over time in terms of space.

A third class of tasks, often called *non-spatial alternation,* involves emitting or withholding one particular response. Thus, on one trial reward is obtained by responding, whereas on the next trial there is no reward and proper performance involves withholding the response. This alternation pattern can be tested in either the Skinner box or the alley. It is *not* a true non-spatial analogue of the spatial alternation task noted above; such an analogue would involve the presentation of two items, with reward alternating between the two from trial to trial while the items shifted location in some random fashion. Such an item alternation task has not, to our knowledge, been used with lesioned subjects.

The non-spatial alternation task is a special case of a broader category of tasks labelled *go–no-go:* here, responses are sometimes rewarded, sometimes not. The availability of reward is typically signalled by a guidance; in the special case of alternation the guidance is replaced by the memory of the previous trial.

9.4.1. SPATIAL ALTERNATION

Most experiments involving spatial alternation in hippocampal animals have employed operant chambers; a few studies, in rats, have used two-choice mazes. The results of these studies are given in Table A25. In the studies using a T-maze or Y-maze deficits have generally been noted, even when inter-trial intervals are quite brief (e.g. Racine and Kimble 1965).[*] As in tests of spontaneous alternation (see p. 260) hippocampal animals find it quite difficult not to repeat responses. The deficit seems related in some way to the fact that the type of maze typically used restricts choices to two markedly different turns. Hirsch (1970) tested rats in a modified Thompson box[**] with four doors. Reward followed any choice which was not a repetition of the previous choice. In this situation rats with hippocampal lesions obtained a normal number of rewards; however, they tended to alternate between two choices, while the control animals spread their responses more evenly over the four choices.

Though deficits are often reported in alternation tested in an operant chamber there are exceptions, in monkey (Waxler and Rosvold 1970), cat (Brown, Kaufman, and Marco 1969), and rat (Stevens and Cowey 1972, 1973). In the Waxler and Rosvold study data were presented for individual monkeys. Some hippocampal monkeys were normal while others were deficient, and these differences could not be attributed to any difference in the size of the lesion, as had been suggested by an earlier study (Rosvold, Mishkin, and Szwarcbart 1964). The authors suggested that there are several ways to learn this task, and that some of these might require the hippocampus, while others would not. For example, they noted that some monkeys tended to respond to one manipulandum and then immediately switch to the other one to await the next trial; this strategy correctly 'tells' the animal where to respond and, if used by a hippocampal monkey, could lead to normal performance. Other monkeys tended to respond to one manipulandum, await the next trial, and then switch to the other side. It is likely that a hippocampal animal using this strategy would be impaired in that it would respond immediately to the nearest manipulandurn upon starting the next trial. This being the case, one is surprised that most other studies have reported such clear-cut deficits in spatial alternation learning in hippocampal monkeys. Mahut (1971) also presents individual data and again there was a split in the hippocampal group: two failed completely while two learned, though at a retarded rate. The total failure of both monkeys with amygdala lesions in Mahut's study may partially explain the total deficits reported in the early studies; the lesions in these latter studies often included some damage to the amygdala.

Thus, while the story is not clear as yet, it does seem likely that the monkey without a hippocampus can sometimes find a way to solve this task, perhaps along the lines suggested by Waxler and Rosvold. In the only cat study reported, that by Brown *et al.* (1969), the hippocampal animals were slightly, though not reliably, worse than were the intact animals. The authors did not comment upon the way in which the animals solved the task. We already have noted in our discussion of delayed response that lesioned cats can learn to take up postures which overcome their spatial defect, and this might have happened in the Brown *et al.* study.

Four operant spatial alternation studies have been reported for the rat; two of these found deficits in hippocampal rats (Niki 1966, Riddell, Malinchoc, and Reimers 1973), while two found either normal or better than normal performance in the lesioned rats (Stevens and Cowey 1972, 1973). There were some important procedural differences between these studies which might account for the different results they obtained. Stevens and Cowey (1972) report four studies, all using the same basic paradigm. Rats were placed in a Skinner box with two levers (or panels) on either side of a recessed well in which a food cup was located. In some of the studies cues were presented, either on the panel or above and to the side of the lever, while in others there were no cues to the alternation pattern. Among the results reported in this series of studies were the following.

(1) Both control and hippocampal groups performed reliably better when a relevant cue was provided, whether on the panel or to the side of the lever. This improvement was limited, in Stevens and Cowey (1973), to rats with dorsal but not ventral hippocampal lesions.

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[*] Jarrard (1975) has shown that pre-operatively trained rats perform a Y-maze spatial alternation following hippocampal lesions, even with inter-trial intervals as long as 10 mins. This atypical result could be a function of any of several procedural differences between the various studies: (1) the pre-operative training; (2) the use of correction or non-correction procedures; (3) the use of a heterogeneous maze providing cues to alternation.

[**] This apparatus typically is rectangular, with partitions dividing it into a start area, a choice area containing doors, and a goal area.

(2) The availability of an irrelevant cue (unrelated to the position of the 'rewarded' manipulandum) also improved performance, though to a lesser extent than did the relevant cue.

(3) Hippocampal rats performed equally well when the cue was spatially contiguous with the response site (lit panel) or when it was separate (light to the side of lever), while the control rats were as good as the hippocampals with the spatially contiguous cue, but significantly poorer with the separate cue.

(4) Contrary to the claims of the authors, there is little evidence that the rats learned the alternation task in the absence of the cue.

Performance of all the non-cued groups peaked at between 60 and 70 per cent. Random initial choices, combined with a 'lose-shift' strategy would generate 'learning' scores of roughly 65 per cent. The 'learning' seen in these animals, then, seems to involve the adoption of such a strategy.

Much the same thing was seen by Niki (1966), whose normal rats achieved only 70 per cent correct responses after 10 days of training without cues, and by Riddell, Malinchoc, and Reimers (1973), who set their criterion at 50 per cent correct responses in the absence of cues. The deficit in Niki's study was related to the fact that

> 'bursts of responding on the same bar were frequently observed in the hippocampal animals' (p. 4).

Thus, a major difference in these studies might be the extent to which they fostered the adoption of a 'lose-shift' strategy; this is consistent with procedural differences between the studies. Niki gave his animals 50 rewards per day, regardless of how long this took, while Riddell, Malinchoc, and Reimers gave their animals 20 correction trials each day. Stevens and Cowey, on the other hand, tested their animals for a fixed period of time and the number of rewards that could be obtained was inversely related to the amount of time spent responding to the incorrect lever. In this sense the rats in the Stevens and Cowey studies were under pressure to avoid perseverative errors, while the same was not true in the other studies. This analysis is consistent with data concerning the rate at which the animals decreased perseverative errors in the various experiments. In both the Niki and Riddell, Malinchoc, and Reimers studies the decrease was relatively slow, normal rats achieving 50 per cent only after five to six days, while in the Stevens and Cowey studies 50 per cent performance was achieved by the fourth day. The deficits reported by Niki and Riddell, Malinchoc, and Reimers, then, could relate to the low cost of perseverative responding in their situations, in conjunction with the established tendency of hippocampal animals to engage in bursts of incorrect responses.[*]

[*] The massive defect in Riddell, Malinchoc, and Reimers is certainly related to the fact that the rats had just previously been performing a position task in the same apparatus. This prior experience could only exacerbate the difficulties of the hippocampal rat.

The most interesting aspect of these data is the abject failure of all the animals to acquire the alternation pattern in a remotely successful way in the absence of cues in the operant chamber, as compared with the relative ease with which alternation is acquired in the T-maze. In the latter case there are two distinct locations containing food and the normal animal can adopt a place hypothesis involving alternation between the two. In the operant chamber the food is often in the same place regardless of which lever is to be pressed, while the proximity of the two levers and the restricted overall environment minimize the usefulness of place hypotheses. As in the DRL task, the provision of a guidance improves normal as well as hippocampal performance.[*]

9.4.2. GO–NO-GO AND NON-SPATIAL ALTERNATION

The essential feature of the go-no-go situation is that sometimes a particular response is rewarded while at other times the identical response goes unrewarded. The cue to the availability of reward can be either external (e.g. a tone signalling the fact that a response will be rewarded, the S^+), or internal, as in temporal discrimination, or through the use of a patterned sequence of discrete reward and non-reward trials.

The results from studies of go–no-go are presented in Table A25, separated into those which provided cues and those which utilized temporal or patterned-trial procedures. In the standard go–no-go procedure a confused mixture of results has been reported. In two early studies, which were not adequate tests of go–no-go (Niki 1965, Swanson and Isaacson 1967), hippocampal rats were extensively pre-trained on a CRF lever-pressing task and then a discriminative stimulus (S^-) was introduced signalling the non-availability of reward. In both studies the hippocampal rats failed to decrease their responding during the S^-. The fact that there was extensive CRF pre-training, however, limits the generality of these results.

Schmaltz *et al.* (1973) have reported that rats with hippocampal lesions are deficient in go–no-go, and that this deficiency involves over-responding to the S^- and, during the later stages of the study, the S^+ as well.[**] Similarly, Wild and Blampied (1972) and Woodruff, Means, and Isaacson (1973) report that hippocampal rats are impaired both on retention and acquisition of go–no-go. On the other hand, Schwartzbaum, Thompson, and Kellicutt (1964) showed that hippocampal rats could retain a preoperatively acquired go–no-go discrimination, the only abnormality in

[*] We have already discussed the fact that when the cue is at a remove from the lever normal animals perform less well than do hippocampals (p. 268).

[**] In this study the hippocampal rats did not make more S^- responses than did a control group which received bilateral hippocampal injections of sodium sulphadiazine, a drug assumed not to elicit seizures. Another group of rats given bilateral injections of penicillin, an epileptogenic drug, were significantly *better* than either of the above two groups. This set of results is extremely difficult to interpret (see Chapter 12 for a discussion of chemical injection studies).

their behaviour being an increase in responding during the S^+ beyond that seen in normal animals. This pattern of adequate go–no-go performance coupled with high S^+ response rates has also been reported by Gaffan (1973) in the absence of pre-operative training. Freeman, Kramarcy, and Lee (1973) trained rats on a go–no-go tone discrimination using either the presence or absence of tone as the S^+. Hippocampal rats performed normally when the presence of tone was the S^+ (and its absence the S^-) but were deficient when these conditions were reversed.[*] Further, Freeman and Kramarcy (1974) report normal S^+/S^- learning when either tone or light serve as the S^+.

In two other studies, using hippocampal cats and dogs respectively, Buerger (1970) reported deficient go–no-go performance while Mering and Mukhin (1973) reported normal go–no-go in their hippocampal animals.[**]

In the midst of this confusion we can only suggest the following conclusions: (1) there is no general deficit in go–no-go discrimination in hippocampal animals; under certain circumstances they can perform adequately on this task; (2) while not necessarily performing in a deficient fashion, hippocampals tend to over-respond to the cues; (3) this indicates that in this situation the lesioned animals are, as in other situations we have already noted, unusually dependent upon guidances.

As regards the use of external cues, in both Schwartzbaum *et al.* (1964) and Gaffan (1973) separate tones were used as S^+ and S^- and normal performance was seen in both cases. In all the other studies only one cue was used, with the exception of Mering and Mukhin (1973) who also reported normal performance. Thus, it appears that hippocampal animals have difficulty in so far as they cannot rely upon a cue telling them when responses will be rewarded. It is possible to suggest that their difficulty resides in their use, in the absence of places or guidances provided by the experimental situation, of an orientation hypothesis that carries with it over-responding and stereotyped behaviour.

These studies do not support the conclusion that the hippocampal animals failed to appreciate the significance of an S^-. In five of the studies (Schwartzbaum *et al.* 1964, Wild and Blampied 1972, Gaffan 1973, Freeman *et al.* 1973; Freeman and Kramarcy 1974) go–no-go training was followed by tests of the *generalization gradients* established to the discriminative stimuli; that is, following training the animals were tested with a range of stimuli varying in similarity to the original S^+ and S^-. In all these cases, with the exception of Wild and Blampied (1972), the lesioned animals showed good gradients, both of excitation and inhibition. Thus, tones which were similar to the S^+ elicited responding, while those similar to the S^- elicited considerably less responding.

The results of go–no-go alternation studies are presented in Table A25. With the exception of Franchina and Brown (1970) and Brunner, Haggbloom and Gazzara (1974), all studies have used an operant chamber. In the former runway study reward was available on alternate trials and good performance consisted in a particular pattern of latencies: long after reward and short after non-reward. A deficit was seen in the hippocampal rats in this situation; they started running rapidly on almost all trials, while control rats showed longer start latencies on the trials following reward. On the other hand, Brunner *et al.* reported normal performance on this task in rats with X-ray-induced hippocampal dysfunction.

A series of studies by Walker, Means, and their colleagues has investigated the effects of hippocampal lesions on an operant analogue of this task; rats were given discrete trials in an operant chamber with reward and non-reward trials alternating in a fixed sequence. The results of these studies seem to suggest that lesioned rats can be either better or worse than are normals at this task, depending upon the inter-trial interval. Our previous discussion of performance in operant situations would certainly suggest that the lesioned rats should be deficient at this task; in the absence of specific cues they should over-respond during non-rewarded trials, at least during the early stages of training. Thus, the facilitation reported with brief inter-trial intervals is unexpected. In order to account for this facilitation we must consider the nature of the task more closely. Good performance in this situation consists in either or both of two things: a long latency to respond on the non-reward trials and fewer responses on those trials. Anything in the experimental situation which contributes to these would improve performance. For instance, the location of the levers relative to the food cup might influence latency to respond, as might the amount of food obtained on the previous reward trial.

In the first two experiments in this series the food cup was located across the box, opposite the levers. After a reward trial, upon retraction of the lever, the rat would have to move across the box to eat its pellets. Upon resumption of the trials the lever is re-inserted but no other cues given. Latency to respond on this (non-reward) trial would clearly depend upon several things, including the probability that the animal would be waiting for the lever to reappear or aware of its location in the box. We have already described, in discussing Karmos and Grastyan (1962), how the hippocampal animal fails to respond in similar circumstances unless it

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[*] This deficit might relate to the difference seen in intact animals between feature-positive and feature-negative tasks. When the S^+ is a discrete stimulus learning is faster than when this stimulus is the S^-. This relates to the autoshaping and sign-tracking phenomena we discussed earlier (p. 266), which represent the utilization of guidances. The added deficit of hippocampals on the feature-negative task is consistent with their presumed dependence upon guidances.

[**] Buerger used an unusual procedure. Cats were rewarded for responding during S^+ and for not responding during S^-, but were punished for incorrect responses or non-responses. They had extensive damage to piriform cortex and some damage in amygdala as well. Mering and Mukhin used more confined lesions and got normal go–no-go behaviour except in the case where the S^+ was simply the time since the previous trial. Again, the importance of a salient guidance is noticeable.

actually detects the triggering object (see p. 268). It is possible that in this operant alternation situation the hippocampal animal is at an advantage because it does not know where the lever is located. This is consistent with the fact that changes in the location of the lever can have powerful effects upon the picture just outlined; both Warburton (1969) and White (1974) have found slight deficits at brief inter-trial intervals when the lever was located adjacent to the reward site.

Walker, Messer, and Means (1970) reported that hippocampal rats were normal with 10 s intervals but deficient with longer intervals. Means (personal communication) notes that in this study the food cup was adjacent to the lever, as it was in subsequent reports. Walker *et al.* (1972), varying inter-trial interval, found facilitation at 10 s, normal performance at 20 s, and deficits at 40 and 80 s. Walker and Means (1973) report normal performance at 10 s inter-trial interval, and Means (personal communication) now suggests that there is a slight facilitation for the hippocampal rats at this interval.

In addition to changing the location of the food cup relative to the lever, the later studies simultaneously altered one other feature of the task, the reward schedule used during the go trials. In the two early studies, with the food cup and lever on opposite sides of the box, presses were rewarded on an FI-2 schedule to eliminate multiple presses. Thus, given a 10 s trial, rats could obtain at best five pellets per trial. In the later studies, with food cup and lever adjacent, responses during go trials were rewarded on a CRF schedule, vastly increasing the number of pellets the animals could obtain. The number of pellets obtained on go trials could affect latencies on subsequent no-go trials. Where data are given (Means *et al.* 1970) a clear-cut relationship emerges between the number of rewards obtained and the increase in no-go trial latencies both for normal and for hippocampal rats. This relationship did not hold as clearly for no-go trial responses, particularly for the normal rats. The latency measure, however, appears to be the more sensitive one for dissociating normals from hippocampals, and one could suggest that 'learning' in this situation depends indirectly upon the rate at which rewards are obtained. Facilitation observed in the lesioned rats could simply indicate that they obtain more rewards at the start of training. This would be expected on the basis of their performance in any operant situation. Means (personal communication) occasionally observed his rats in this situation and noted that the hippocampals spend more time near the lever and food cup, and less time grooming and exploring. This pattern is consistent with what we have seen for CRF performance and with the idea that these rats obtain more rewards.

The results of the latest study in this series confuse matters somewhat further. Walker and Means (1973), using adjacent lever and food cup and a CRF schedule on go trials, investigated the effects of interpolating a

different lever during the inter-trial interval. Rats were first trained with a 10 s interval and then switched to a 30 s interval. As noted above there was no facilitation in the hippocampal group at the 10 s interval in this study. At the 30 s interval the hippocampals were impaired, but primarily in relation to intact controls. Cortically lesioned controls also had some difficulty with this task. At this point a new lever was introduced (on the other side of the food cup) during the middle 10 s of the inter-trial interval and responses to it were rewarded on a CRF schedule. This hindered the performance of both hippocampals and cortical controls, though only temporarily. The hippocampal rats responded considerably more frequently to this lever than either of the control groups.

In sum, hippocampals appear to be basically deficient at the go–no-go task, as one might expect, though at short inter-trial intervals certain features of the apparatus, combined with the properties of the reward schedule, can act to overcome this deficit. Without further data concerning the actual behaviour of the rats in this situation it is not worth speculating any further. It is worth pointing out an inaccuracy in a report by Woodruff et al. (1973). They stated that go–no-go brightness discrimination is impaired while go–no-go alternation is facilitated. They further state that when visual cues are available to the hippocampal rat in the alternation situation they are not used, and this would argue against our notion that hippocampals pay particular attention to such guidances. However, this statement is misleading. Visual cues were used in only one study of go–no-go alternation (Walker, Means and Isaacson 1970) and then they were added after rats had reached criterion. We would predict that such cues, if available during learning, should ameliorate any defects observed in hippocampal rats at long inter-trial intervals.

9.5. Summary

Summarizing this section is difficult, for there are a number of situations in which the effects of hippocampal lesions remain unclear. Situations requiring, or favouring, place hypotheses, such as delayed response and delayed spatial alternation, usually produce deficits. Situations clearly requiring taxon hypotheses, such as classical conditioning, appear to produce mostly normal behaviour. Problems of interpretation arise particularly with those situations requiring behavioural flexibility. Abrupt shifts in reward characteristics or response requirements usually elicit deficits, though these can often be overcome by the presence of appropriate guidances. Finally, processes such as generalization seem intact in hippocampal animals.

Part of the problem in providing a clear picture for most of these situations lies in the interaction between locale and taxon mechanisms directing behaviour. For many of the tasks discussed in this chapter the locale system plays a minor role. However, there are exceptions; the lay-out of an environment can often be crucial in determining behaviour, as seen in the ostensibly non-spatial lever-press go–no-go studies. Further, exploration is a basic aspect of the intact animal's response to any change, including that inherent in shifting operant schedules. The failure of hippocampal rats to adjust to such shifts is most marked when the shift is abrupt. In the following chapter we consider this problem in detail, in the context of an examination of the reactions of hippocampal animals to the removal, or change, of the reward features of a task. Our discussion will directly confront the central problem of *perseveration,* or *persistence,* in hippocampal animals, and the conclusions reached in that discussion will help to make sense of the somewhat confused picture presented in this chapter.

10

Reactions to reward change

In establishing that the locale system is integrally involved in reactions to environmental change we purposely omitted any detailed treatment of reactions to changes in biologically meaningful items, though this is central to much of the behaviour we have considered. It is clear from much of the previous discussion that shifts in reward contingencies can often lead to aberrant behaviour in hippocampal animals. In the present chapter we shall provide the basis for understanding these effects.

A wide variety of changes fall under the rubric of reward shifts: changes in the amount of reward, its quality, its locations, the objects with which it is associated, and so on. Though we have already discussed many studies incorporating such changes we have not focused on reward change *per se.* Certain studies are directly concerned with this problem; the most obvious of these involve the complete removal of reward. This leads to what is called the *extinction* of the behaviour previously based on that reward.

According to the present theory, both the locale and taxon systems contain mechanisms which are sensitive to changes in reward conditions and which could lead to the cessation of inappropriate behaviours. As a way of approaching the question of the specific effects of hippocampal lesions upon reactions to reward changes we shall (1) examine the concept of extinction and the general problem of persistence, (2) consider these in terms of the locale and taxon systems, (3) discuss the lesion data concerned with extinction, and (4) turn to the general case of any change in reward contingencies.

10.1. Extinction and persistence

After reviewing the extant theories of extinction, Mackintosh (1974) pointed out that

> 'traditional S-R theory is … left with no account of the learning process underlying extinction' (p. 418).

Partly, this results from the tendency to consider extinction a unitary process and to attempt to explain all extinction phenomena within a single theoretical framework. Such an approach is likely to be no more successful than its counterpart in learning theory. More specifically, this failure follows from the emphasis of S-R theory upon the inhibition of old learning without any detailed consideration of the new learning which can occur during extinction.

The inhibition conceived by traditional theory flows from any of several sources: (1) *reactive inhibition* (Hull 1943) follows the occurrence of any response, exhibiting both a transient and a more permanent phase; (2) *generalization decrement* occurs because the extinction situation is distinguishable from training, and the trained behaviour is not conditioned to this new situation; (3) *conditioned inhibition* results from the association of non-reward with the trained behaviour; (4) in a less direct way *interference* results from the exploration elicited by the new situation and from competition with any new behaviours. Only the last of these hints at the new learning that can occur during extinction, and it is a rather indirect mechanism. Though all of these forms of inhibition no doubt occur they cannot, singly or in combination, account for all the facts of extinction.

Effects upon extinction are usually gauged in terms of *resistance to extinction,* or *persistence,* which refer to the extent to which behaviour remains unchanged in the face of the removal of reward. As we have seen, abnormal persistence is one of the dominant features of the syndrome produced by hippocampal dysfunction, and an understanding of the processes underlying extinction would shed considerable light on hippocampal function. For this reason we shall consider the problem of persistence in some detail.

In describing the properties of the various behavioural hypotheses we raised the possibility that different hypotheses might be differentially persistent; that is, they would be used with varying degrees of rigidity. Place hypotheses were assumed to be minimally persistent because they are based upon a type of information structure which can be rapidly and easily amended when the environment changes. The unexpected absence of reward should elicit exploration, the map changed, and the previous place hypothesis rendered inoperable. This is a type of change which falls outside the inhibitory mechanisms postulated by S-R theory. Maps and place hypotheses are not inhibited; they are changed as a function of new learning. Taxon hypotheses were assumed to be more persistent and the possibility was noted that guidance and orientation hypotheses might differ in this regard. The persistence of a taxon hypothesis should be directly related to its strength, which in turn would be a function of the stereotypy with which it is deployed. Orientation hypotheses, by definition, are most likely to be used in a stereotyped fashion and could demonstrate the most marked persistence.

These postulated differences in the persistence associated with the various types of hypotheses suggest that shifts in persistence brought about by certain experimental treatments, including for instance brain lesions, might reflect changes in the use of particular hypotheses. Thus, any treatment inducing a shift from the use of a place hypothesis to a taxon hypothesis would be expected to increase persistence.

10.1.1. PERSISTENCE SHIFTS

A variety of experimental treatments affect the persistence with which behaviours are employed, some producing increases, some decreases. The stereotyped behaviour described by Maier (1949) in response to an insoluble problem is an example of an increase in persistence elicited by particular training conditions. Conversely, increased training can sometimes elicit a paradoxical decrease in persistence either in reversal, the *over-training reversal effect,* or in extinction, the *over-training extinction effect.* The bulk of experimentation aimed at an understanding of extinction and persistence phenomena has been concerned with another treatment which affects persistence–the use of random intermittent reward during training. In most situations such partial reward training serves to increase resistance to extinction, and a discussion of this phenomenon would serve as a means of exploring the possibility that persistence shifts can accompany changes in hypothesis behaviour.

10.1.1(a). The partial reinforcement effect. There are several major theories of the partial reinforcement effect, which is one of the most widely studied phenomena in experimental psychology:

(1) The *discrimination hypothesis* suggests that partial reward training is more similar to extinction than is continuous reward training; thus the onset of extinction cannot be easily discriminated. This notion is derived from the generalization decrement hypothesis of extinction.

(2) The *memory after-effects hypothesis* (Capaldi 1966), another application of the generalization decrement notion, suggests that behaviour during training becomes conditioned to the after effects produced by reward or non-reward. During continuous-reward training animals learn to respond only in the presence of reward after effects; thus extinction is relatively rapid. During partial-reward training animals learn to respond in the presence of both reward and non-reward after effects; thus extinction is prolonged.

(3) The *frustration hypothesis* (e.g. Amsel 1958) suggests that animals are frustrated by their failure to find food (or water) on non-reward trials, and that behaviour gradually becomes conditioned to the cues associated with frustration. Then, the frustration elicited during extinction is compatible with continued responding.

None of these theories, according to Mackintosh (1974), accounts satisfactorily for the partial reinforcement effect, though all the processes postulated could play some role in its development. In addition to these factors we would suggest another which could contribute to the unusual persistence seen after partial-reward training: a shift in the nature of the hypotheses underlying behaviour.

(1) In response to the frustration associated with unpredictable non-reward, taxon hypotheses could become increasingly stereotyped in the fashion described by Maier (1949). This factor could be partially responsible for the *partial reinforcement acquisition effect* which involves the gradual development of supra-normal performance during partial-reward training (but see (3) below). This is manifested as faster running in an alley or as increased response rates in the operant chamber.

(2) In response to the uncertainty associated with random reward place hypotheses might become inoperable. This follows from a set of connected considerations: *(a)* any variability in the environment elicits, via the locale system, exploratory behaviour; (b) random intermittent reward constitutes a source of continuous variability; (c) the continuous elicitation of exploration interferes with goal-directed behaviour; thus (d) partial-reward training biases strongly against the use of place hypotheses. If animals discard a place hypothesis in the face of variability this should happen within a few trials of non-reward and should lead to the use of a more persistent taxon hypothesis. This factor could account for the partial reinforcement effect observed after only a few training trials, a phenomenon which cannot be explained in terms of the gradual conditioning of behaviour to the cues provided by frustration.

(3) As a corollary to the shift from place to taxon hypotheses, the variability associated with intermittent non-reward could devalue the output from the locale mismatch system, even when taxon hypotheses are being used. When extinction begins the animal would not react immediately with exploration, and this would have the effect of increasing persistence.

The factor leads to the general postulate that any form of environmental variability during training could increase persistence, a postulate similar to one forwarded recently by Amsel (1972).* The importance of this idea is that it is the variability inherent in partial reward, rather than non-reward *per se,* which primarily produces increased persistence. It is important to note in this regard that the variability must be truly unpredictable in order to elicit increased persistence; neither regularly patterned partial reward nor a constant delay of reward, both of which could be considered frustrating, produce increased persistence.

Thus, the increased persistence seen during extinction after partial-reward training can be ascribed to several factors: (1) a shift in the hypotheses underlying behaviour occasioned both by frustration and variability, leading towards the use of more persistent hypotheses; (2) the neglect of the exploration system which normally acts to facilitate extinction; (3) the conditioning of behaviour to the context provided by non-reward. Certain of these would appear in hippocampal animals, while others would not, in that they depend upon the presence of the locale system. We shall return to the partial reinforcement effect, and its manifestation in hippocampal animals, later.

10.1.1(b). Extinction variables. Resistance to extinction can also be affected by conditions during extinction itself. Two such factors are of particular interest in the present context; the interval between repetitions of the trained behaviour, and the availability of alternative behaviours. In many situations it appears that the use of shorter inter-trial intervals facilitates extinction. This would seem to be a function of the rapid build-up of reactive inhibition which serves directly to inhibit the previously trained behaviour. Under these conditions a rest interval allows the inhibition to dissipate and the behaviour *spontaneously recovers.* According to our model both the effect of massed extinction trials and spontaneous recovery should be evident in hippocampal animals, in as much as they depend upon factors working within the taxon system. In fact, the normal animal might show less sensitivity to the effects of inter-trial interval during extinction when place hypotheses are being used for extinction would not depend upon the accumulation of reactive inhibition.

The availability of alternative behaviours can also affect extinction rates. Reversal can be viewed as an extinction situation (for one behaviour) which incorporates a highly likely alternative behaviour. The extinction of certain avoidance tasks, superimposed upon approach tasks, offers another example of a situation where suitable alternatives could help the animal extinguish old behaviours. It is obvious that the hippocampal animal, lacking exploratory mechanisms, might be crucially dependent upon the presence of such alternatives.

10.1.1(c). Extinction reconsidered. The foregoing discussion indicates that variations in persistence can result from several factors: (1) shifts between

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* According to this view

> 'the maintenance of any ongoing behaviour in the face of a stimulus that evokes an orienting-disruptive response increases resistance to extinction ... The mechanism responsible for this increased persistence is a more general form of the counterconditioning mechanism in the frustration theory of the partial reinforcement effect' (Amsel et al. 1973, p. 176)

This latter explanation becomes unnecessary when one assumes that in order to maintain behaviour in the face of stimuli which would otherwise elicit exploration, the animal must ignore its locale mismatch system.

hypotheses of varying persistence;* (2) the neglect of the misplace system; (3) the action of variables influencing the build-up of inhibition. The latter factor depends upon processes occurring within the taxon systems, while the former two factors depend upon interactions between systems. Place hypotheses are most easily extinguished; merely demonstrating the absence of reward should elicit exploration, the development of up-dated maps, and the abandonment of the now inappropriate place hypothesis. The phenomenon of latent extinction (eg. Seward and Levy 1949) represents this form of extinction in a relatively pure state. Taxon hypotheses are more persistent, though in the intact animal the existence of the locale system provides for exploration, which plays an important role in the extinction of these hypotheses partly by allowing for the development of competing behaviours.

Thus, the animal without a hippocampus suffers from a dual deficit in extinction situations. First, the use of minimally persistent place hypotheses during training is impossible. Second, the facilitating effect of exploration should be absent. This suggests that strong deficits in extinction could occur in lesioned animals. Further, it suggests an unusual dependence upon those factors affecting persistence within the taxon systems. On the other hand, a number of phenomena associated with extinction could be intact in hippocampal animals. That portion of the partial reinforcement effect unrelated to the shift from place to taxon hypotheses, or the blocking of exploration, should appear; spontaneous recovery should be normal; the action of reactive inhibition should be observed.

10.2. Extinction after hippocampal damage

According to our model the effect of hippocampal lesions should be to make the animal dependent upon the taxon systems both for the behaviour learned during training and the means by which this behaviour is changed once extinction has begun or when some other change in the reward contingencies has been instituted. We have already described some evidence for this; changes in the location of reward, or its associations with other objects, can be reacted to in a normal or abnormal fashion, depending upon the extent of locale involvement in the normal animal. The introduction of shock in the alley approach experiment shows both normal and abnormal behaviour developing in the hippocampal animal at the same time. The intact rat, behaving on the basis of a place hypothesis, reacts to the introduction of shock by altering its behaviour at a remove in the start area. The lesioned rat, using a taxon hypothesis, cannot inhibit its running behaviour in the start area but can avoid contact with the object associated with shock. Similarly, when reward is moved from one place to another, the intact rat quickly switches its behaviour to accommodate the change; the hippocampal rat cannot manage the same. Such examples predict that deficits will often, but not always, be seen in hippocampal animals during extinction.

The results of studies concerned with extinction in hippocampal animals are presented in Table A26. It is clear from the table that profound deficits are seen in a variety of situations. However, two important exceptions to this pattern emerge. First, in a few studies which have manipulated inter-trial intervals (eg. Jarrard, Isaacson, and Wickelgren 1964; Jarrard and Isaacson 1965) large deficits were observed only when spaced trials were used. Second, nearly normal extinction rates, with certain qualifications, are often seen in avoidance tasks, operant tasks, and classical conditioning situations. These facts indicate that extinction deficits are not an inevitable accompaniment of hippocampal damage. Before turning to a closer analysis of these exceptions we should note a few important observations concerning the nature of the hippocampal animal's behavioural response to the withdrawal of reward.

Cohen (1970) observed that while control rats sniff, scratch, and attempt to get out of the empty goal box, hippocampal rats remain placid, seemingly oblivious to the absence of reward. This failure to react to non-reward could be interpreted in several ways. First, it could imply that these animals generally lack a frustration response to the withdrawal of reward. We think this is unlikely, and shall discuss the question of frustration later. Here, it suffices to say that hippocampal animals display what appear to be either increased or at least normal frustrative reactions in a number of other situations. Second, the absence of a reaction to non-reward in the runway could indicate the absence of the specific expectation of reward in the goal box; that is, no mismatch occurs. This seems quite plausible to us, and it is consistent with the fact that hippocampal animals do not show the characteristic elevation of plasma corticosterone upon the withdrawal of reward (Coover, Goldman, and Levine 1971b), a function apparently associated with the occurrence of environmental mismatch (see pp. 360-1). The absence of such a mismatch, however, does not mean that extinction becomes impossible. In fact, in both the Cohen (1970) and the Coover *et al.* (1971b) studies the hippocampal animals showed extinction, albeit somewhat more slowly than did controls. The absence of the mismatch function does imply, however, that extinction might proceed in different ways in intact and hippocampal animals.

Kimble (1969) trained rats on a brightness discrimination task in a Y-maze for water reward. He ran the animals to a satiation criterion;

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* Aside from the partial reinforcement effect the over-training extinction effect might also be partially explicable in terms of shifts between hypotheses. In this case, however, the shift would be from a more persistent to a less persistent hypothesis. We cannot examine his possibility in detail here, but it is consistent with the fact that the over-training extinction effect is most prevalent in situations (eg. runways) allowing for a shift from taxon to locale hypotheses, presumably when considerable training has generated extensive reactive inhibition (cf. Kendrick 1958).

that is, he continued to train the rats until responding for water stopped spontaneously. Both control and hippocampal rats stopped drinking at about the same time. A few trials later the control rats stopped running; the hippocampal rats ran throughout 100 trials, though they did not drink. The same pattern of persistent responding was seen in the hippocampal rats when a standard extinction procedure was used. These data suggest that the hippocampal rats were not running for water *per se.* Their behaviour bore all the marks of a fixation which, initially dependent upon reward, had become autonomous. In this situation, as Kimble has shown, running could persist almost indefinitely. Another aspect of the data from the Kimble study confirms the suggestion that the hippocampal animals were responding on the basis of a persistent taxon hypothesis; they responded almost entirely to the cue that had been associated with reward. Similar fixation of choice during extinction in hippocampal animals has been reported by Niki (1965) and by Douglas and Pribram (1966). Thus, in situations where profound extinction deficits are reported, there are clear indications that hippocampal animals perform on the basis of persistent taxon hypotheses. This provides a basis for understanding the important effect upon extinction of those factors to which the taxon system is most sensitive: the rate of repetition of non-rewarded responses, and the availability of alternative behaviours.

10.2.1. VARYING INTER-TRIAL INTERVAL

For the normal animal in the runway inter-trial interval seems relatively unimportant during extinction; this is reasonable if we assume that extinction in these situations is based predominantly on changes in the locale system. For the hippocampal animal, however, inter-trial interval appears critical; massed extinction trials can lead to relatively normal extinction rates in hippocampals. We assume that these data reflect the way in which persistent taxon hypotheses can be altered; massed repetitions without reward lead to a rapid build-up of inhibition. Thus, these results parallel those presented for habituation (p. 249) and reversal (p. 382) in suggesting that when the same behaviour is repeated (and unrewarded) at short intervals it will drop out in hippocampal animals. It is important to note that the behaviour in question must not constitute a long chain of sequential acts. We noted in our discussion of habituation and hyperactivity that hippocampal animals decrease their activity at a seemingly normal rate only in small boxes where the same responses are repeated at a rapid rate, and often fail to decrease their activity in larger boxes where repetition rates for the same response are slow. A parallel to this effect in extinction is provided by the study of Jarrard and Lewis (1967); they showed that massed extinction trials in a complex maze failed to lead to normal extinction rates in hippocampal rats, presumably because the rate at which particular responses in the maze were repeated was too slow.*

Much the same analysis can be applied to extinction in operant situations. These can be viewed, as we have pointed out, as fairly pure examples of taxon function. Thus, we would expect that extinction in both normal and hippocampal animals might be relatively lengthy, but that no major differences should appear between the groups. Most operant situations leave response rate at the discretion of the animal; thus, inter-response times should be quite short during the early stages of extinction. Extinction in operant tasks typically proceeds at about the same rate in normal and hippocampal animals, with small deficits sometimes appearing in the lesioned subjects. The large deficits reported in the runway, where the normal animal can avail itself of place learning and its associated rapid extinction, find no parallel in the operant situation.

10.2.2. THE AVAILABILITY OF ALTERNATIVE BEHAVIOURS

As we have seen, the 'extinction' of responding to the previously rewarded stimulus in a non-spatial discrimination reversal task is relatively rapid in hippocampal animals (p. 283); they quickly adopt an alternative, though usually maladaptive, behaviour which is attractive in that it is at least sometimes rewarded (Silveira and Kimble 1968). This effect holds even when trials are spaced. In contrast, the total removal of reward in the same situation (Kimble and Kimble 1970) results in a profound extinction deficit which is characterized by abnormally stereotyped responses to the previously rewarded stimulus.

This comparison brings out the crucially important role that an alternative behaviour can play in extinction for hippocampal animals. The distinguishing feature of those aversively motivated tasks which have produced normal extinction rates in hippocampal animals is that they provide highly likely alternative behaviours for the animals. Thus, in the case of the conditioned emotional response, the aversively controlled behaviour (suppression) is superimposed upon an appetitive habit. When the aversive contingencies are removed the animal can re-engage in this appetitive behaviour; hippocampals do so at least as readily as do normals. In this situation the alteration in aversive contingencies is clearly signalled to the animal; shock is no longer presented after the conditioned stimulus. In most avoidance situations this is not the case; as long as the animal

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* A curious aspect of the data in the Jarrard and Isaacson (1965) study concerned the effect of switching the inter-trial interval in the course of extinction. Hippocampal rats extinguished initially with massed trials showed normal extinction rates; when they were switched to spaced trials the running behaviour recovered. Normal rats did not show this recovery effect. These data strongly suggests that the two groups had extinguished in different ways. We suspect that the recovery of responding in the hippocampal rats reflected the dissipation with time of some of the short-term inhibition built up during the massed extinction trials. The absence of this effect in normal rats indicates that extinction in these animals was based on something other than, or in addition to, this type of inhibitory effect.

continues to avoid it fails to detect the removal of shock at the onset of extinction. This, of course, partly accounts for the well-known difficulty associated with extinction of avoidance behaviours. Lovely *et al.* (1971) have shown how this can affect extinction rates in hippocampal rats trained on a two-way avoidance task. They found an extinction deficit in their lesioned rats when one considered the number of trials on which a response was made after shock had been made contingent upon response; this is consistent with what we have already seen concerning the learning of this task. However, if extinction was measured in terms of the number of responses made *after* an animal had once failed to run, there was no difference between the groups. Most of the hippocampal rats never responded again, after once performing this alternative behaviour.

10.2.3. EXTINCTION IN NORMALS AND HIPPOCAMPALS COMPARED

The foregoing has documented the case that hippocampal animals are often deficient at extinction, but that they can show `normal' extinction rates when given either massed unrewarded trials or a strong alternative response. This sensitivity to factors which often fail to influence the rate of extinction in normal animals indicates that extinction proceeds in different ways in the intact and hippocampal animal; this implication is strongly supported by a study reported by Warburton (1972). Rats were given repeated acquisition-extinction cycles on an operant task. The hippocampal rats had a small, but persistent, deficit in extinguishing this behaviour. Warburton then tested the effects of the intraperitoneal injection of an anti-cholinergic drug upon the extinguished behaviour. This drug elicited a recovery in the normal animals but had no effect upon the hippocampal animals. These data suggest that normals and hippocampals had extinguished in quite different ways, using brain systems dependent upon different neurotransmitters. They also support the suggestion, made earlier, that normal animals can extinguish through the action of the locale system when taxon mechanisms alone would not have supported extinction. Removal of the locale system releases the taxon systems and the behaviour 'recovers'. This analysis can be applied to an experiment reported by Niki (1962). He showed that hippocampal lesions produced after extinction can lead to the partial recovery of the previously extinguished behaviour, in the absence of any post-operative training. Here again, extinction within the locale system seems to have occurred before sufficient inhibition had been generated in the taxon system, and this disparity was brought out by the subsequent removal of the locale system.

The deficits reported in extinction in hippocampal animals, then appear to be a function of the different ways in which the locale and taxon systems respond to the withdrawal of reward. At the same time a few aspects of reasonably normal extinction performance can be observed in hippocampal animals, presumably reflecting functions mediated solely by the taxon systems. Thus, spontaneous recovery, assumed to reflect the dissipation of short-term inhibitory effects, remains intact in hippocampal animals (Schmaltz and Theios 1972). Similarly, the effect of work decrement on extinction, a factor directly related to the inhibition generated by the performance of a response, remains the same in normal and hippocampal animals (Van Hartesveldt 1973).

10.2.3(a). The partial reinforcement effect after hippocampal damage.

Several studies have been reported which investigated the role of the hippocampus in the partial reinforcement effect. We have suggested that this effect rests partly on shifts from less persistent place hypotheses to more persistent taxon hypotheses, partly on the shutting off of the exploratory system, and partly on the effects of frustration. Animals with hippocampal lesions should show a partial reinforcement effect, but should not show that part of it related to the persistence induced by hypothesis shifts involving the locale system, or that part due to the shutting off of the mismatch system. The absence of the locale system makes original learning more prone to persistence, even without partial reward. Thus, the partial reinforcement effect might, in hippocampals, be superimposed upon the deficit in extinction shown by continuously rewarded hippocampal animals.

The available data generally support this position. Bloom and McFarlain (1971) tested rats in a runway under continuous or partial (50 per cent) reward conditions. Although the hippocampal rats had a deficit in extinction they displayed a partial reinforcement effect. That is, continuously rewarded hippocampals extinguished more rapidly than did partially rewarded hippocampals. On the other hand, Franchina and Brown (1970) showed that hippocampal rats trained with either patterned or random partial reward in a runway extinguished at the same rate, which was roughly equivalent to that seen in intact rats given random partial-reward training. The authors concluded that the partial reinforcement effect was absent in the hippocampal rats; this conclusion would seem to us to be unwarranted. The control groups in this study learned to pattern their running speeds in the patterned partial-reward condition; they ran fast after non-reward trials and slow after reward trials. They did not show the partial reinforcement effect in this situation, as we have already noted. However, the hippocampal rats tested under this patterned partial-reward condition failed to pattern their responses in accordance with the alternating schedule. They ran rapidly on all trials, as did the hippocampal rats given random partial rewards. The fact that both these hippocampal groups showed extinction rates similar to the random partial-reward control animals raises a sticky interpretive problem. Did the hippocampals show a partial reinforcement effect in both cases, or did they simply have an extinction deficit and no partial reinforcement effect at all? Two points suggest that the former is the more likely explanation. First, the Bloom and MacFarlain study, and others to be noted, indicate that the partial reinforcement effect can be demonstrated in hippocampal rats. Second, Franchina and Brown used massed extinction trials (20 s inter-trial interval), a procedure which should have produced normal extinction rates in their hippocampal animals (see above). Thus, the prolonged extinction in both hippocampal groups would seem to reflect the presence of a partial reinforcement effect, rather, than its absence.

Williams (1971) trained rats in a runway under continuous or partial reward using spaced trials. Hippocampal rats showed the expected extinction deficit in this situation, whether they had been continuously or partially rewarded. However, in terms of the number of trials required to reach two different criteria of extinction (the first or the third extinction trials with a latency of 60 s), the partially rewarded hippocampals took reliably longer than did the continuously rewarded hippocampals. Thus, in the presence of an extinction deficit it is still possible to detect a partial reinforcement effect in hippocampal animals. More recently, Brunner *et al.* (1974) have reported a partial reinforcement effect in rats with X-ray-induced damage in the hippocampus.

The most convincing data have been provided by Amsel *et al.* (1973). These investigators showed that the increased persistence selectively elicited by stimulus variability during training (a tone presented at the end of an FR-21 chain) was absent in hippocampal rats. This is consistent with the notion that this increase in persistence is related to a shift in hypotheses, or to a shutting off of the exploratory system, and differs somewhat from the persistence elicited by the frustrative aspects of random non-reward. The latter, in view of the studies discussed above, appears intact in hippocampal animals.*

10.3. Reaction to reward change

Extinction, as we noted at the beginning of this chapter, represents a drastic case of a class of situations involving a change in reward contingencies. The abnormal persistence seen in hippocampal animals in extinction is often seen in other situations involving such changes, as we have noted at several points in this section of the book. In the remainder of this chapter we shall attempt to account for the general character of this persistent behaviour as it applies to any situation involving shifts in reward contingencies.

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* A study by Rabe (1968) showed that abnormal persistence in hippocampal rats *depended* upon frustrative non-reward in an operant situation. Rats were trained an FR-20 schedule. Following this, a second lever was introduced, responses to which were rewarded either on an FR-5, FR-10, or CRF schedule. Both normal and hippocampal rats showed considerable, though not different, persistence to the less rewarding FR-20 lever. This is markedly different from what is seen in an analogous runway task (Gaffan 1972) where normals switch but hippocampals do not.

Less drastic situations involving reward shifts include the shifts in operant schedules discussed earlier (p. 322) and changes in the quantity or quality of reward given for a particular response, amongst others. Through all these situations the common thread of frustration appears. This concept, discussed briefly above, is worth elaborating upon because it has been suggested that the hippocampus is centrally involved in frustrative reactions (eg. Gray 1970, Glazer 1974a,b).

Frustration is assumed to be mildly aversive, to energize behaviour, and to provide cues to which behaviours can be conditioned. The classical paradigm for demonstrating frustration makes use of its energizing property. Rats are trained to run in an alley with a goal box at the end and another goal box midway down the alley. Removal of reward from the initial goal box triggers frustration, which speeds up running to the final goal box (Amsel and Roussel 1952). Other tests for frustration include shifts in operant schedules and the partial reinforcement effect. The general problem of reactions to reward shifts, then, is often intertwined with the notion of frustration, except in those cases where a shift involves an increase in reward.

10.3.1. THE EFFECTS OF HIPPOCAMPAL LESIONS UPON FRUSTRATION

Notwithstanding the claim that the hippocampus is important to frustration mechanisms, the available evidence indicates that frustrative effects are largely intact in hippocampal animals. In the most direct test of this notion, Swanson and Isaacson (1969) showed that hippocampal rats were indistinguishable from control rats in the double-alley described above. In operant situations, as we have seen, frustrative shifts often elicit overresponding in hippocampal animals (e.g. Swanson and Isaacson 1967, Rabe and Haddad 1968). In fact, Swanson and Isaacson suggested that the

> 'hippocampal rats may have been uniquely sensitive to the frustrative conditions of reinforcement withdrawal in the sense that their frustration response was more intense'
> (p. 34).

In contrast to this demonstration of 'increased' frustration, a study of reward shift in a runway failed to find significant performance changes in hippocampal rats following either an increase or a decrease in reward quantity (Franchina and Brown 1971). Further, Niki (1962) has shown that hippocampal rats fail to decrease running speeds in an alley when a delay is interposed between response and reward,* while two studies

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* Mikulka and Freeman (1975) have shown that this condition retards the learning of a Y-maze position habit in hippocampal rats.

have demonstrated deficits in hippocampal rats in a situation requiring slow running (Posey 1972, Rickert and Bennett 1972). Similarly, Murphy and Brown (1970) failed to find a reliable contrast effect[*] in hippocampal rats switched from high to low-concentration sucrose solutions (however, see Gaffan (1973) for a study reporting normal contrast in fornical rats). Most of these studies demonstrate decreased frustration reactions in hippocampal animals. Thus, one can observe either increased or decreased frustrative reactions in response to various treatments. It seems unlikely that a direct effect upon frustration is one of the effects of hippocampal lesions.

10.3.2. THE BASIS OF ABNORMAL SHIFT BEHAVIOUR

The thread tying the various shift studies together is neither abnormal frustrative reactions, though these may sometimes occur, nor an inability to respond to altered reward contingencies, though this too is sometimes seen. Rather, it appears to be an inability to change certain forms of behaviour. As a number of studies have made clear, hippocampal animals are sensitive to different levels of reward when such differences are present from the onset of training (e.g. Murphy and Brown 1970, Franchina and Brown 1971, Kramarcy, Mikulka, and Freeman 1973) in that their asymptotic performance rates reflect these different reward levels. Thus, it cannot be the case that alterations in the quantity or quality of reward go unnoticed. Whether or not the hippocampal animal can alter its behaviour in the face of these changes, however, seems to depend upon the nature of the behaviour itself. Thus, appropriate shifts in behaviour have been reported in some operant situations, or in situations where consummatory responses alone are measured, or in some extinction tests. On the other hand, decreasing reward, or delaying it, in a runway often fails to elicit a marked decrease in running speed. This difference between spatially extended runways and operant situations has been noted before (see p. 342). Intact rats can alter their behaviour in the start box in such situations, while lesioned animals seem to have to wait until they catch sight of the objects of reward themselves, though this does not always lead to normal performance (particularly in extinction).

This difference between normal and hippocampal reactions to reward shifts shows up clearly in Posey's (1972) study of runway behaviour. His rats were first trained to receive reward regardless of runway speed. Following this, reward was only given on those trials on which a run took at least 5 s; this required slowing down. The hippocampal animals were poor at this task, but what is particularly interesting is the distribution of changes in running speeds within the runway. Lesioned rats were faster than controls in the start area, but slower in the goal area. This suggests that the control rats master the task primarily by hesitating in the start box, a feat beyond the powers of the lesioned rats.[*]

It seems clear that changes in reward are often noticed, if responded to inappropriately, in hippocampal rats. A study reported by Gaffan (1972) provides evidence that hippocampal disruption does not eliminate sensitivity to reward shifts, even in runways, though it might leave animals unable to alter their behaviour rapidly. Rats were trained to turn left on a T-maze to obtain four small food pellets. Following the achievement of stable performance on this task, eight pellets were made available in the right arm. Normal rats rapidly learned to turn right in order to get the eight pellets first; rats with fornix lesions never did this. However, they demonstrated clearly that they had learned about the eight pellets by decreasing their latency to the right arm (via the left arm and its four pellets) dramatically.[**]

In a recent study (Van Hartesveldt 1973) the effects of variations in reward size upon the acquisition, performance, and extinction of both continuous and partial reinforcement operant schedules were explored. When reward size was decreased on the CRF schedule both normal and hippocampal rats increased their response rates. Similarly, both groups responded similarly to the partial reward schedule (FR), including the number of sessions it took the rats to extinguish when the ratios was set at 105 responses per reward.[***] Van Hartesveldt suggested that abnormal increases in response rate were not seen in her hippocampal rats upon reward level decreases because the decreases occurred gradually. Increases were seen, but they did not differ from the increases observed in normal animals. As we noted earlier (p. 322) hippocampal rats adjust normally to shifts in operant schedules only when the shifts are not abrupt. Abrupt shifts are likely to elicit exploration in intact animals, which would militate against the marked response rate increases observed in hippocampal animals.

The data from all these studies suggest that the basis for persistence in hippocampal animals resides in their use of stereotyped taxon hypotheses, without the possibility of disrupting these through exploration. Normal animals either use place hypotheses, which are minimally

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[*] This refers to an unusually large downward shift in performance after a downward shift in reward such that shifted animals perform worse than controls always tested at the lower reward level (cf. Crespi 1942).

[*] The Hullian concepts of backward chaining and the backwards breaking down of an alley response during extinction might apply quite nicely to hippocampal rats, if not to normal ones.

[**] This study shows that the effects of hippocampal damage can be equally marked in reactions to increased, as well as decreased, reward. Thus, it constitutes *prima facie* evidence against a faulty frustration reaction interpretation.

[***] Perhaps the most important finding in this study was the fact that extinction rates were similar for the two groups. Extinction in this situation, as the author pointed out, was a function of the inhibition

> 'generated by increasing the amount of work required to obtain a reward' (p. 356).

This type of inhibition corresponds to that postulated for the taxon systems and it is clear from these data that this form of inhibition remains intact after hippocampal lesions.

persistent and easily extinguished, or they use taxon hypotheses which can be disrupted by exploration. Only when the experimental situation is arranged such that taxon hypotheses must be used and exploration has been eliminated, as in partial reinforcement training for instance, will the normal animal be reduced to the persistent behaviour seen in hippocampal animals.

10.3.3. CONCLUSIONS

The above discussion indicates that reactions to reward changes are often abnormal in hippocampal animals because they lack the locale mechanisms available to intact animals; their performance reflects solely the properties of the taxon systems. The obvious corollary to this position, as we just noted, is that intact animals should perform like lesioned animals when they use taxon hypotheses and when exploration has not been allowed.

The central point to be made concerning an understanding of extinction, both in normal and lesioned animals, involves what takes place prior to the removal of reward: how has the animal learned the task? In some situations a variety of hypotheses can be used, while in others performance requires the use of a specific hypothesis. A proper evaluation of extinction effects requires the prior analysis of learning. The means by which the former occurs will depend upon the hypothesis used during learning. The withdrawal of reward will be rapidly registered in the locale system, and this will lead to the rapid extinction of inappropriate place hypotheses. The cessation of behaviour, however, will still depend to some extent upon the strength of the taxon hypothesis mediating it, for most tasks incorporate habits requiring response hypotheses. One-trial extinction is relatively rare. The locale system contributes to the extinction of these taxon habits by triggering exploration, such that the full persistence associated with taxon hypotheses is rarely seen. This delicate interplay between locale and taxon systems accounts for the difficulty in providing a neat analysis of extinction, in normal as well as in hippocampal animals. Yet it offers the possibility of understanding the persistence often seen in hippocampals, not as a function of some inhibitory capacity of the hippocampus which has been disrupted but rather as a function of the properties of what remains after this disruption. As such, it might point the way to a more comprehensive theory of extinction, one which sees this phenomenon as occurring in any of several ways, involving both new learning and the inhibition of old learning.

11

Maintenance behaviours

IN discussing the effects of hippocampal damage upon various learning tasks we bypassed the question of the more general effects of brain damage upon such things as eating, drinking, and so on. We have assumed that none of these maintenance behaviours nor, for that matter, basic sensory and motor processes, are critically dependent upon the hippocampus. In our treatment of fear-based behaviour and aggression (see pp. 302-15) we concluded that any influence of hippocampal damage was indirect. In the present chapter we shall consider two broad areas of lesion effects: (1) influences upon drinking, eating, sexual, maternal, and social behaviours; (2) influences upon sensory, motor, autonomic, and endocrine function.

While the lesion data relating to these behaviours are reasonably clear, considerable data generated in studies using electrical or chemical stimulation techniques point to other, less clear-cut, conclusions. In the present chapter we largely restrict our discussion to the lesion data. Studies employing stimulation will be discussed in the following chapter.

11.1 Food and water intake, and related behaviours

In general, hippocampal lesions do not affect food or water intake in animals under either ad libitum (free access) or deprivation conditions (see Table A27).* Intake of sucrose, quinine water, and salt water (after adrenalectomy), seems normal in hippocampal animals. It is reasonable to assume on the basis of these data that the hippocampus is not critically involved in consummatory behaviours. However, Jarrard (1973) has proposed that the hippocampus does play a role in these and other such behaviours; the lesion data summarized in Table A27 would appear to render any general role for the hippocampus in consummatory behaviour highly unlikely.

Two recent studies have investigated the hoarding of food in hippocampal rats (Wishart, Brohman and Mogenson 1969, Wallace and Tigner 1972). This behaviour involves the transportation of food from

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* Thomka, Murphy, and Brown (1975) have shown that hippocampal rats drop more food through their cage floors than do normal rats; this might account for any reports of increased food intake. The authors also point out that weight loss after hippocampal lesions could result if all animals are put on a diet involving a fixed, small amount of food, and the lesioned animals lose more through spillage.

an area outside the animal's living cage into the cage and would appear to reflect the action of several factors: deprivation of food, the relative insecurity of the location outside the cage containing the food, and a tendency to transport objects (cf. Munn 1950, Bindra 1959, for discussions of hoarding). An interesting possibility raised by these two studies, as noted by Wallace and Tigner, is that

> 'a difference in security does not have the same effect on the hoarding of hippocampals as it does on that of control animals' (p. 941).

Hoarding in the lesioned rats, as in the controls, was affected by the deprivation of the animals. The absence of an influence of the security–insecurity dimension on hippocampal animals is consistent with the finding that these animals begin to eat more rapidly in strange environments (eg. Jarrard 1968) than do normal animals. The absence of such place fear agrees with our conclusions formed from a consideration of aversively motivated behaviour (pp. 302-15).

11.2. Social, maternal, and sexual behaviour

Both sexual and maternal behaviour can be affected by hippocampal lesions. Early work suggested that hippocampal lesions in male rats prolong the time to the initiation of the first mount (Dewsbury *el al.* 1968) but then decrease the inter-mount interval, such that hippocampal rats mount more often per unit time (Kim 1960b, Bermant, Glickman, and Davidson 1968). However, Michal (1973) has reported that hippocampal lesions decrease mounting frequency in male rats. These data taken together indicate that hippocampal damage neither increases nor decreases sexual drive in a direct manner.

Michal computed measures of the predictability of behaviour sequences in his rats and found that hippocampal rats were markedly more predictable than were control rats; that is, they tended to perform the same sequences repeatedly. This facet of their behaviour could account for both the increased and decreased mounting observed in the studies noted above. Michal measured sexual behaviour in his rats for only 10 min; no normal rat managed to mount to ejaculation in this time. Given that hippocampal rats take longer to initiate the first mount, it is conceivable that with a longer test period Michal could have found increased rather than decreased mounting frequencies. The common thread in all these studies is the stereotypy in behaviour, be it mounting as in the early studies or attention to the head and body of the female without mounting, as observed by Michal.

While an early paper suggested that hippocampal lesions do not influence social behaviour (Glickman *et al.* 1970), three later reports (Kolb and Nonneman 1974, Nonneman and Kolb 1974, Ely, Greene, and Henry 1976) have noted marked changes in rat, cat, and mouse, respectively.

In rats, hippocampal damage drastically reduced contacts between pairs of lesioned animals in both large and small fields. In cats, lesions seemed to make the animals submissive to intact cats. The authors commented on the fact that the lesioned cats seemed uninterested in other cats, preferring to eat if allowed to do so. However, when presented with a silhouette model of a cat, lesioned animals seemed quite interested in it, though their responses to this model do not decrement normally. In mouse, a failure to develop social dominance hierarchies was observed in conjunction with decreased aggression. All these data could, as Ely *et al.* suggested, be explained in terms of a loss of cognitive mapping. The possibility that the hippocampus is more directly involved in social behaviour must be left open. At least one aspect of (anti) social behaviour remains intact after hippocampal damage in rats: mouse-killing (De Castro and Marrone 1974, Kolb and Nonneman 1974).

Hippocampal lesions in female rats can affect various components of maternal behaviour, including nursing, retrieving, and nest building (Kim 1960a; Kimble, Rogers, and Hendrickson 1967), while in the cat these lesions affect oestrous behaviour (Peretz 1967). These changes are not directly predicted from the present theory, and we can only assume that they are attributable to other defects that are directly predicted. For example, the deficit in retrieving and nursing might be due to the mother's inability to locate the pups accurately. Thus, Kimble *et al.* give an example of a lesioned dam who

> 'executed perfect hovers, but several inches away from any of the pups' (p. 406).

11.3. Sensory and motor function

No gross motor or sensory defects of a permanent nature are seen in animals after bilateral hippocampal lesions.* However, two studies have suggested that unilateral lesions can lead to a subtle motor defect (Greene, Saporta, and Walters 1970, Saporta and Greene 1972). Rats with unilateral lesions were found to have both a turning and orienting bias ipsilateral to the lesion. This bias persisted in the visual mode after optic chiasm section, indicating that it rests on a motor, rather than sensory, effect. According to the present model the hippocampus has no direct motor role that would account for these results. Such biases are seen after many subcortical lesions, and it must remain a possibility that they are not directly attributable to the hippocampal locus of the lesion.

No particular bias has been reported after bilateral lesions, though one can assume that damage in these cases is often quite asymmetrical. What is often seen, on the other hand, is a strong preference for turning to one side or another in two-choice situations; these preferences persist for

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* We have already noted that the hippocampus can modulate sensory evoked potentials (see pp. 138-9) and that hippocampals might by hyper-reactive to intense sensory inputs (pp. 248-50).

abnormally long periods in hippocampal animals, as we have seen. However, it is not unusual for hippocampals to demonstrate one turning preference at an early stage and then switch to the opposite preference, maintaining this for a long time as well. Thus, it seems unlikely that the persistent turning tendencies so often noted in hippocampal animals are related to the effects reported by Greene and his colleagues; this possibility must be left open until further work clarifies the nature of the motor effects of unilateral lesions.

We have noticed some remarkable short-term motor-changes in rats with electrolytic or mechanical lesions of the dorsal fornix (O'Keefe and Nadel, unpublished observations).* For the first few days after the lesion these animals are profoundly catatonic. When placed on a table or turned on their backs and left unrestrained they will maintain the unnatural posture for several minutes. Abnormal limb postures are also maintained in this way. In a few of these animals extreme catatonia resembling the waxy flexibility described by Stein (1971) after injections of 5-hydroxydopamine is seen; these animals retained unusual standing postures for 15 min or more. The absence of whisker movements reported by Gray (1970) after lesions in the septum does not occur after fornix section unless the septum is also damaged.

This condition, it should be noted, resembles that reported by Bures, Buresova, and Weiss (1960) after bilateral placement of potassium chloride (KCL) crystals in the dorsal hippocampus of rats (e.g. the elicitation of spreading depression) and by MacLean (1957b) after direct deposition of crystalline carbachol in the hippocampus of the cat. MacLean noted that after his injections

> 'sometimes the animal may be draped into various postures, which it will maintain for minutes at a time (p. 130).

In our experience this condition clears within a few weeks of operation, although the experienced investigator can often tell the difference between normal and hippocampal rats when they are lifted up; the hippocampal rat appears 'looser' and more relaxed. The cause of the initial gross change is unknown; one could speculate that it results from surgical trauma and/or seizures associated with the surgical or stimulation procedure. Whatever its cause, this phenomenon would appear to be relatively unimportant in assessing the results of lesion studies, most of which are carried out after a recovery period during which this abnormality subsides.

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* De Castro and Marrone (1974) have described similar effects of hippocampal lesions. They reported the same changes noted above, as well as the fact that these effects are ameliorated by the passage of time. They argued that these changes did not underlie the behavioural change they were investigating (shock-induced fighting). The report of increased 'tonic immobility' in rabbits (Woodruff, Hatton, and Meyer 1975), after the lesioned animals were turned on their backs, is probably another example of this effect.

11.4. Autonomic and endocrine functions

Basic autonomic and endocrine functions appear to be intact in hippocampal animals. Thus, basal metabolic rate (Kim 1960a), heart rate Jarrard and Korn 1969), and galvanic skin response (Bagshaw *et al.* 1965) are all normal and there is

> 'no essential defect in pituitary-adrenal function in . . . hippocampus-ablated rats' (Coover et al. 1971b, p. 731).

The absence of 'essential' defects does not mean, however, that either autonomic, or particularly endocrine, functions are always normal in hippocampal animals. We have already noted that abnormal heart rate responses to supra-threshold shocks occur in these animals. Similarly, the evidence that certain hormonal systems are influenced by, and in turn can influence, the hippocampus is now quite strong. This evidence relates primarily to the pituitary-adrenocortical system (involving ACTH and corticosterone in the rat); there is little evidence that other hormone systems, such as those involving the gonadal hormones for instance, are modulated by the hippocampus.

The evidence concerning this hormone-hippocampus interaction falls into three categories: (1) the uptake of radioactively labelled hormone by the hippocampus; (2) the effects of systemic injections of various hormones upon the activity of hippocampal neurones; (3) the effects of hippocampal lesions or stimulation on pituitary-adrenal function. The studies providing this evidence are listed in Table A28.

11.4.1. UPTAKE STUDIES

In a series of experiments McEwen, Weiss, and their colleagues have demonstrated that the hippocampus is a potent site of uptake for corticosterone in the rat (e.g. McEwen, Weiss, and Schwartz 1969, McEwen, Magnus, and Wallach 1972, Gerlach and McEwen 1972); this finding has been replicated in other laboratories (Ford, Rhines, and Steig 1971, Knizley 1972). The binding of corticosterone, which is primarily nuclear (Gerlach and McEwan 1972, Warembourg, 1975), appears to be maximal about one hour after injection (McEwen *et al.* 1972, Ford *et al.* 1971). The intensity of uptake varies among the different hippocampal fields, with CA1 and CA2 showing the most intense labelling (Gerlach and McEwen 1972, Warembourg 1975); the latter study showed strong labelling in the fascia dentata as well. This strong uptake of corticosterone by the hippocampus can be contrasted with its general failure to concentrate gonadal hormones (Stumpf and Sar 1971, Zigmond and McEwen 1970, but see Luttge, Chronister, and Hall 1973 and Pfaff and Keiner 1973 for conflicting reports).

11.4.2. EFFECTS ON SINGLE UNITS AND EEG THETA

Pfaff, Silva, and Weiss (1971) have shown that corticosterone can reduce the firing rates of neurones in the dorsal hippocampus. ACTH, on the other hand, can increase hippocampal neurone firing rates; some units were found which were affected by both ACTH and corticosterone. These results were seen in chronic and acute preparations in both intact and hypophysectomized rats. The ACTH-induced increases usually began 3-10 min after injection and lasted 25-30 min, whereas the corticosterone induced decreases began 10-40 min after injection and lasted at least 2 h. In contrast, Dafny *et al.* (1973) found that the hippocampus was the least reponsive limbic area to injections of cortisol, a related corticosteroid. Whereas over 80 per cent of units in areas such as the anterior hypothalamus, ventromedial hypothalamus, and mesencephalic reticular formation changed firing pattern, only 13 of 28 units in the dorsal hippocampus were affected. Of these, seven increased activity, while five decreased activity.

Several studies (Martin, Moberg and Horowitz 1975, Urban and De Wied 1975, Urban *et al.* 1974) have reported an effect of adrenal hormones on hippocampal EEG. Urban *et al.* (1974) found that ACTH$_{4-10}$ significantly reduced the frequency of the hippocampal theta in the dog while the animal waited for a visual signal to press a lever for food reward. The effect was only seen under conditions of mild deprivation when the animal's performance was low and erratic and not during strong food deprivation when its performance was high and consistent. In another study from the same laboratory, Urban and De Wied (1975) found that ACTH$_{4-10}$ *increased* the frequency of theta elicited by stimulation of the reticular formation in unrestrained rats. The maximum changes occurred 60-120 min after the injection.

Martin *et al.* (1975) looked at the spontaneous and sensory elicited theta in the unrestrained rabbit. They found an increase in the amount of spontaneous theta 60-90 min after injection of corticosteroid but no change in the theta evoked by auditory stimuli. It is unfortunate that in the latter two EEG studies and in the chronic part of the Pfaff *et al.* unit study behaviour was neither monitored nor controlled, so that one cannot rule out an intermediary effect of the hormone on behaviour.

11.4.3 EFFECTS OF LESIONS OR STIMULATION

A large number of studies (listed in Table A28) have investigated the effects of lesions or stimulation of the hippocampus on pituitary-adrenal function. These can be split up into two categories: (1) those investigating the physiological functioning of the pituitary-adrenal system, that is, resting levels and/or cyclic variations; (2) those investigating pituitary-adrenal responses to various experimental treatments, typically involving stressful or novel stimulation.

11.4.3(a). Physiological functions. While a number of authors have claimed that fornix section or hippocampal lesions abolish the normal diurnal rhythm of plasma corticosteroids[*] (Mason 1958, Nakadate and de Groot 1963, Moberg *et al.* 1971, Endroczi 1972), there have been many negative reports (Galicich, *et al.* 1965, Endroczi and Nyakas 1971, Wilson and Critchlow 1973/4, Kearley, Van Hartesveldt, and Woodruff 1974, Lanier *et al.* 1975). Some of the confusion could relate to the time of testing after operation. Lengvari and Halasz (1973) have shown a disruption of the diurnal cycle with fornix lesions at 1 week, but not 3 week, postoperative testing. Most of the positive reports indicate that the effect consists of a smoothing out of the normal cyclic rhythm such that lesioned animals have abnormally high levels in the morning and abnormally low levels in the evening, though one study (Endroczi 1972) reported an absolute increase throughout the day. In most of these studies no attempt was made to correlate corticosteroid levels with behavioural activity, in spite of the known relationship between the two, and the fact that hippocampal damage usually alters activity levels, in particular attenuating cyclic activity changes (e.g. Jarrard 1968).

Chemical or prolonged electrical stimulation of the hippocampus has been reported either to increase basal ACTH secretion (Bohus, Nyakas, and Lissak 1968) or to decrease plasma corticosteroid levels (Mason 1959 Rubin, Mandel, and Crandall 1966, Kawakami *et al.* 1968), though there have been negative results (e.g. Endroczi and Lissak 1960). The extent to which these results are due to the spread of after discharges to other structures is not clear, as most of these studies failed to provide controls for this possibility. To add to the confusion, the effects of stimulation have been shown to be critically dependent upon frequency parameters; low-frequency stimulation inhibits ACTH release while high-frequency stimulation facilitates it (Endroczi and Lissak 1962). These data do not allow any firm conclusion concerning the nature of hippocampal influence upon resting pituitary-adrenal function. However, if we assume that low-frequency stimulation mimics physiological function while high-frequency stimulation acts as a functional lesion, these data are consistent with the idea that the hippocampus normally acts to inhibit ACTH release.[**]

11.4.3(b). Behavioural functions. While the data concerning resting levels are somewhat confused, a clearer picture is emerging from a consideration

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[*] This cycle consists in high levels in the evening and low levels in the morning, and is related to, though lagging behind, cyclic changes in activity levels (Endroczi 1972).

[**] This assumption, taken in conjunction with the rapid potentiating effect of ACTH on hippocampal neurones, suggests that there is a negative feedback loop maintaining resting ACTH levels.

of the kinds of environmental manipulations which elicit pituitary-adrenal activity and the way in which the hippocampus might exert its influence upon this hormonal system.

Much of the work concerned with pituitary-adrenal responses to environmental factors has concentrated upon physically unpleasant stimuli such as extreme cold, physical restraint, anaesthesia, electric shock, and so on. The view arose that pituitary-adrenal mobilization was nonspecific, related to any stressful situation (cf. Selye 1950). While there is little doubt that such extreme stimuli mobilize the pituitary-adrenal system, there is increasing evidence that other, less extreme, stimuli might also be able to mobilize the system. Thus, Mason (1971), in criticizing the traditional view, stated that:

> 'it appears highly advisable that we view with skepticism the conclusions drawn from all those past and future studies of the role of "physiological" or "physical" stimuli in endocrine regulation in which no rigorous efforts were made to minimize or assess the possible role of associated psychological reactions' (p. 238).

Among the psychological, or *neurogenic,* factors that seem important in mobilizing the pituitary-adrenal system several stand out as particularly interesting in the present context. There is little doubt, for instance, that fear provokes an endocrine response; ACTH and other pituitary peptides, as well as corticosterone, all have an influence upon behavioural reactions in fear-provoking situations (cf. de Wied *et al.* 1972 for a review). It is worth noting that this influence appears strongest in situations of 'mild' fear (e.g. Weiss *et al.* 1969, 1970), where mild fear is usually taken as *situational* or place defined.

Perhaps more pertinent, strong pituitary-adrenal responses are triggered by uncertainty, novel situations, and the failure to confirm expectancies. Thus, Mason (1959) showed that endocrine mobilization occurred in 'first-experience' situations, even when these did not involve pain or an obvious threat to the animal. Endroczi (1972) has shown that the pituituary-adrenal system responds strongly to unmet expectancies, such as the withdrawal of expected rewards or closing down an alley normally used to get to water in a maze. Similarly, Bohus (1973), Coover *et al.* (1971a) and Coover, Ursin, and Levine (1973) have all provided evidence that

> 'one of the conditions which will activate the neuroendocrine mechanisms leading to subsequent release of ACTH is a change in expectancies' (Coover el al. 1973, p. 174).

These studies indicate that environmental uncertainty, or novelty, is a crucial factor in endocrine release.

The fact that uncertainty and environmental mismatch can be singled

out as potent triggers for pituitary-adrenal mobilization brings our discussion back to the possible nature of the hippocampal influence upon this endocrine system. The one situation in which hippocampal rats fail to demonstrate normal plasma corticosterone elevations concerns the extinction of an appetitive response (Coover *et al.* 1971b). Thus, mismatch mobilizes the pituitary-adrenal system in normal, but not hippocampal, animals. This deficiency could account for many of the effects of hippocampal disruption upon pituitary-adrenal reactions to stress.

11.4.4. CONCLUSIONS

These data suggest that an important interaction exists between the hippocampus and the pituitary-adrenal system. One possible way of conceiving of this interaction is as follows: the pituitary-adrenal system is under direct control of portions of the hypothalamus (and other pituitary hormones), as well as being controlled from feedback through basal levels of ACTH and corticosteroids themselves. This system undergoes patterned circadian rhythms which are related to, and affected by, other circadian rhythms, including those involved with activity and temperature regulation. Triggering of this endocrine system, as part of a mobilization pattern in preparation for behavioural exertion, can be effected by several brain systems (cf. Krieger and Krieger (1971) for evidence that there are several brain systems capable of independently mobilizing the endocrine response). One particularly effective triggering system is connected with the hippocampal novelty detection-mismatch function; stress would be another trigger. As part of its output in mismatch situations, the hippocampus will activate the release of ACTH.

The presence of corticosterone-binding cells in the hippocampus and the predominance of these in CA1, coupled with the responsiveness of hippocampal neurones to ACTH and corticosterone, could all reflect part of a feedback loop maintaining basal levels of these hormones, whilst enabling the pituitary-adrenal system to respond to novelty. This simple version of an admittedly complex function is rather vague, but it would not seem particularly useful at present to specify this system in any greater

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[*] There are a number of interesting possibilities, none of which are supported by much evidence. These fall into two classes, concerned with short-term and relatively long-term effects. The pituitary peptides and the corticosteroids have been implicated in the modulation of behavioural responses to fearful situations; they can clearly influence performance in a variety of such tasks, as we noted above. McEwen (personal communication) has suggested that these effects are maximal when fear is related to places rather than objects. Thus, it might be possible to draw a connection between pituitary-adrenal modulation of place-dependent fear and the hippocampal role in the definition of such places.

Another, related, possibility is suggested by the time course of corticosterone binding in the hippocampus, on the one hand, and the effects of ACTH and corticosterone on hippocampal neurone activity on the other. While ACTH has a rapid potentiating effect on hippocampal neurones (and markedly increases the sensitivity detail. We are thus leaving open numerous questions, including those concerning specific effects of the pituitary peptides and corticosteroids on the neural elements themselves.* **

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of hypophysectomized rats to places in avoidance situations), corticosterone has little effect for 30-60 min, at which time it depresses neural activity in the hippocampus. This coincides with a period during which the previously learned avoidance behaviour can suffer from relatively poor performance, the so-called Kamin effect. The fact that corticosterone concentrates in cell nuclei during this time and has been implicated in protein synthesis (McEwen et al. 1969) raises the exciting possibility that this hormone is in some way involved in the plasticity underlying long-term memory changes in the mapping system. The failure to find an effect of hippocampal lesions upon the Kamin pattern (Klein et al. 1975) argues against this possibility.

** Several recent review articles treat the interaction between the hippocampus and various hormone systems (McEwen, Gerlach, and Micco 1975, Bohus 1975, McGowan-Sass and Timiras 1975, Van Hartesveldt 1975). The last of these gives a thorough view of the problem and should be consulted by interested readers.

12

Stimulation Studies

DIRECT stimulation of the hippocampus, by chemical or electrical means, is being used in an increasing number of behavioural studies. As a technique stimulation lacks several of the drawbacks associated with surgical lesions. Its effects are at least theoretically transient, so that the function of a neural region can be disrupted at any stage of a learning experiment at the whim of the investigator. Further, the problem of recovery of behavioural function after surgical lesions, whatever its basis (see pp. 235-6), need not arise with stimulation. Brain stimulation has also been used as a means of assessing the effects of initiating or disrupting normal physiological function upon various aspects of general behaviour, the latter with more success than the former.

These uses of stimulation all suffer from the possibility that, though applied locally, stimulation can elicit important effects in distant structures. This occurs, for instance, through propagated seizure discharges or the diffusion of injected drugs. The effects of electrical stimulation often last considerably longer than the stimulation itself (e.g. Gergen and MacLean 1961), particularly when seizures are evoked. In practice, this means that the electrical activity of other brain areas should be recorded during the course of stimulation in order to detect the propagation of seizure after discharges. As it is now well established that seizure thresholds decrease with repeated stimulation (cf. Goddard, MacIntyre, and Leech 1969), these control records should be taken throughout the course of any stimulation study. Few of the pioneering studies, and too few of the more recent studies, provide this important control.

Chemical stimulation suffers from fewer difficulties once the possibility of diffusion has been controlled. Local application, through implanted cannulae, of minute quantities of drugs can have marked behavioural effects, and this technique conveys the added advantage that one might be able to draw quite specific conclusions regarding the pharmacological basis of these changes. These advantages, however, only hold when the injection site is limited and the quantity of drug is small. Few of the studies presently available fulfill these criteria.

Both electrical and chemical stimulation techniques are continually being improved, and more recent studies employ controls against many of the objections raised above. For this reason it is worth devoting a chapter to the discussion of studies using these techniques. However, our discussion cannot be exhaustive. The interested reader is referred to Table A29 for a list of studies using stimulation techniques and to recent review articles which present these studies in a more complete fashion (e.g. Izquierdo 1975).

In view of the paucity of 'clean' data we have not relied upon stimulation experiments in the development of the present theory. Here, we shall attempt to interpret stimulation studies in terms of the theory and shall discuss three different types of study: (1) studies investigating the general behavioural effects of stimulation, seen either as an initiator or disrupter of normal activity; (2) studies investigating the influence of stimulation during the performance of a previously trained behaviour; (3) studies investigating the effects of stimulation, given either during or after learning, upon learning and/or retention.

12.1. General effects

In describing any effect of stimulation it is essential to separate those effects seen during stimulation from those seen after stimulation has ceased. This is most easily done with electrical stimulation where the dividing line between the two is under the direct control of the investigator. This differentiation between *on* and *off* reactions seems particularly important in regard to hippocampal stimulation where quite different effects can be seen in the two phases. Thus, the most commonly observed effect of mild stimulation of the hippocampus is an alerting, or *arrest*, reaction (e.g. Kaada, Jansen and Andersen 1953, MacLean 1957b, Bland and Vanderwolf 1972b*) during stimulation, while active exploration can be seen as an off reaction (Milgram 1969a). Associated with the alerting response one can observe cortical desynchronization, respiratory acceleration, and heart rate increases (Kaada, Feldman, and Langfeldt 1971). Direct application of cholinergic drugs (typically carbachol) can also elicit this alerted state (Grant and Jarrard 1968), but is often followed by seizures and a catatonic state (MacLean 1957a,b) as we noted before. Local application of anticholinergic drugs (e.g. neostigmine or methylscopolamine) leads to a decrease in exploratory behaviour (Van Abeelen *et al.* 1972), as does strong electrical stimulation involving after discharges in the hippocampus (Leaton 1968). All of these effects are consistent with the notion that hippocampal integrity is crucial to exploratory behaviour. Van Abeelen *et al.* concluded that

> 'the mouse hippocampus contains a cholinergic mechanism which regulates exploratory tendencies' (p. 474).

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* According to Bland and Vanderwolf this arrest pattern is only seen with stimulation of 8 Hz or more.

Less easy to understand are the observations of eating and drinking elicited by hippocampal stimulation (electrical: Milgram 1969a,b, Oliver, Firestone, and Goodman 1973, Huston *et al.* 1974, Milgram, Grant and Stockman 1975; chemical: Fisher and Coury 1962, Coury 1967, Grant and Jarrard 1968, Mountford 1969, Huston *et al.* 1974, Siegfried *et al.* 1975). The present model holds that the hippocampus is not directly involved in the control of these behaviours, and we have already seen that hippocampal lesions do not generally affect eating or drinking. Hippocampal stimulation-elicited eating and drinking differ from that seen with hypothalamic stimulation in two ways. First, they typically occur some time after stimulation, either as a rebound effect upon cessation of electrically induced after discharges (Milgram 1969a; Oliver *et al.* 1973, Milgram *et al.* 1975), or spreading depression elicited by KCl (Huston *et al.* 1974, Siegfried *et al.* 1975), or after a seizure discharge has subsided (Mountford 1969). Second, the eating elicited by stimulation can be conditioned either to a signal (CS) or to the experimental situation itself (Siegfried *et al.* 1975, Milgram *et al.* 1975).

There is strong evidence that the stimulation-elicited drinking is an indirect effect. Routtenberg (1967) suggested that carbachol-elicited drinking was generally a function of diffusion of the drug into the third ventricle; activation of cells bordering on the ventricle would then be responsible for the consummatory pattern. While Mountford (1969) disputed this claim, Simpson and Routtenberg (1972) provided convincing evidence that the crucial site involved in elicited drinking is the subfornical organ; this has been confirmed by other investigators. Elicited eating, on the other hand, does not submit to this simple analysis, though it must be indirect in the sense of being a rebound effect. Our theory does not preclude such effects, but nor does it provide a clear basis for predicting them.

There are at least two ways to account for the rebound elicitation of eating (or drinking) after hippocampal stimulation. First, it could be assumed that the hippocampus has outputs to (presumably) hypothalamic sites driving these consummatory patterns and thus can modulate such behaviour. The fact that these specific behaviours are generally elicited after stimulation in CA1 (Grant and Jarrard 1968, Milgram 1969b, Jarrard 1973) suggests the possibility that this region of hippocampus normally blocks specific consummatory patterns, perhaps as a corollary to its primary function of driving exploration. The decreased latency to eat observed after hippocampal lesions (see p. 256) is consistent with this notion. Second, it could be assumed that the effects of hippocampal stimulation are non-specific in that they appear only when propagated seizures have been elicited. The rebound nature of the behavioural effects is consistent with this idea, as are data from studies of the effects of hippocampal stimulation upon hypothalamically elicited attack behaviour (Siegel and

Flynn 1968, Vergnes and Karli 1969, Nagy and Decsi 1974). Vergnes and Karli showed that

> 'the inhibitory effect of a hippocampal seizure discharge seems to be due to its propagation to other nervous structures, in particular to amygdala and hypothalamus' (p. 889).

Another effect which might be explained in this way concerns the self-stimulation sometimes (Ursin *et al.* 1966, Milgram 1969a, Brown and Winocur 1973, Oliver *et al.* 1973) but not always (Stein 1965, Margules and Stein 1968, Milgram 1969a, Livesey and Wearne 1973) seen with hippocampal placements. A study by Jackson and Gardner (1974) demonstrated that hippocampal stimulation could effect hypothalamic self-stimulation, suggesting that self-stimulation in the hippocampus works through the hypothalamus.

It is not possible to choose between these two alternatives, partly because they are not genuinely opposed. The very presence of such elicited effects indicates that activity started in the hippocampus can influence consummatory patterns. What must remain at issue is the specificity of this influence and the role of seizure after discharges in the triggering of behaviour. Should propagated seizures be essential to the effects then we can conclude that the hippocampus is not central to the control of consummatory patterns. Certainly, the lesion data provide no reason to assume otherwise.

12.2. The effects of stimulation upon performance

Hippocampal stimulation has been used as a functional lesion to assess the effects of disruption upon the performance of previously learned behaviours. The aim of much of this research was the determination of the role of the hippocampus in the long-term memory for such behaviours. The major problem with this use of stimulation concerns the propagation of seizure discharges to other brain structures. Thus, strong electrical stimulation can disrupt simple classically conditioned responses (e.g. Flynn and Wasman 1960, Vanegas and Flynn 1968), the learning of which is unaffected by hippocampal lesions. In most of the studies reporting this effect seizures were routinely elicited. The possibility that such effects upon performance owe to interference with structures other than the hippocampus is supported by the fact that decrements are usually elicited only by intense stimulation. With parameters specifically chosen to avoid at least overt behavioural seizures, or with spreading depression confined to the hippocampus, a different picture emerges. No disruption was seen in the performance of go–no-go discrimination, learning set and delayed alternation (Weiskrantz, Mihailovic, and Gross 1962), nor in a position habit (Olds and Olds 1961), nor in a T-maze brightness discrimination (Grossman and Mountford 1964), nor in lever-press avoidance (Margules and Stein 1968). In the last study more intense stimulation, which likely evoked after discharges, did interfere with performance. Similarly, Nakao (1966) has shown that performance of an escape response motivated by hypothalamic stimulation was disturbed by hippocampal stimulation when after discharges invaded the amygdala. Thus, interference with performance in these studies could be dependent upon disruption of activity in other brain areas. Andy *et al.* (1968) provided strong support for this interpretation when they showed that the debilitating effect of hippocampal stimulation upon performance of a passive avoidance task was ameliorated by fornix lesions.

On the other hand, carbachol injected into the hippocampus interferes with the performance of an operant go–no-go alternation task (Overstreet, Vasquez and Russell 1974), while hippocampal spreading depression has been shown to interfere with one-way active avoidance (Bures *et al.* 1960), spatial alternation (Henderson, Henderson, and Greene 1973), and lever-press avoidance (Erickson and Chalmers 1966). In the active avoidance study deficits appeared to be due to the loss of place information; the authors suggested that the rats

> 'had lost the ability to differentiate the safe side of the apparatus and remember its position' (p. 223).

The deficit in the lever-press avoidance task is in disagreement with both lesion results and the data from a study using electrical stimulation (see above).* In agreement with lesion results, however, is the finding that electrical stimulation facilitates two-way active avoidance (Stein 1965) and disrupts performance on a VI-45 operant schedule (Oliver *et al.* 1973).

Most of these studies of the effects of stimulation upon performance have used tasks which, we can assume from the results of lesion studies, do not require hippocampal participation. The inappropriate conclusion has been drawn that the hippocampus is not involved in permanent memory storage, though it might be involved at some early stage of learning since stimulation does not disrupt performance in many tasks. We can only suggest that tasks should be chosen which demand the involvement of the hippocampus if one is to assess the effects of stimulation properly.

12.3. Effects of stimulation upon learning

Stimulation has been used to study the role of the hippocampus in learning in basically two ways. First, it has been used more or less as a

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* A possible explanation for this discrepancy lies in the technique used to elicit spreading depression. Bures *et al.* (1960) note that when crystalline KCl is used depression is typically confined to the hippocampus, but that with the use of liquid KCl depression can easily spread. Erickson and Chalmers used liquid KCl and did not check for the spread of depression to neighbouring areas.

functional lesion, present during the entire course of learning but usually not during retention. Second, it has been used in a much more precise fashion, applied only at some critical time during/after learning or before/during retention. Here, the interest resides in the role of the hippocampus in some circumscribed stage of the learning/memorization process.

12.3.1. STIMULATION THROUGHOUT LEARNING

This application of stimulation was used in most of the early studies, unfortunately in conjunction with the learning of tasks not normally affected by surgical lesions. Flynn and Wasman (1960), for instance, showed that stimulation involving continual after discharges interfered with the performance of a classically conditioned response during the learning session but that retention was nearly perfect when stimulation ceased. This study shows the danger of ignoring the effects of seizure propagation; it is clear that the hippocampus is not involved in either the learning or the retention of such tasks, but that seizures elicited in this structure can interfere with performance. Stimulation below seizure threshold (Correll 1957), or below the threshold for eliciting overt behavioural responses (Weiskrantz *et al.* 1962), did not interfere with the learning of appetitive approach responses or simultaneous discrimination, respectively.

On the other hand, disruption of the hippocampus interfered with passive avoidance learning (Henderson *et al.* 1973, but see Bresnahan and Routtenberg 1972)[*], reversal of a position habit (Olds and Olds 1961), extinction of an approach response (Correll 1957), the learning of a delayed response task (Hirano 1966), spatial alternation (Greene and Lomax 1970, Henderson *et al.* 1973) or jump avoidance (Whishaw and Deatherage 1971). Most of these results are in accordance with the lesion data, as is the finding that procaine injections into the hippocampus facilitate two-way active avoidance learning (Weiss and Hertzler 1973).

12.3.2. STIMULATION AT CRITICAL STAGES OF LEARNING

The most promising application of stimulation involves its use only at some critical stage of the learning, memorization, or retention process. This application typically rests on the assumption that the storage of memory depends upon processes extended in time after the learning experience and that these processes might be separable into several distinct components. The simplest use of this methodology involves stimulation just after learning and the measurement of retention as an indicator of any hippocampal role in the storage process. More sophisticated, and more recent, applications involve quite discrete stimulation given for brief periods. In either case retention can be tested at various times after learning and/or stimulation, thus distinguishing between the effects of the treatment upon short-term and long-term memory processes.

Many of the early studies using stimulation in this way are subject to the criticisms lodged earlier: the use of inappropriate tasks, the propagation of after discharges, and so on. This makes some sense of the early work, in view of the likelihood of seizure propagation to the amygdala and the now widely accepted role of this structure in most of the tasks used in these studies (cf. Goddard 1964, Bresnahan and Routtenberg 1972). Kesner and Doty (1968), for instance, found deficits in passive avoidance learning only when their post-trial stimulation in the dorsal hippocampus caused after discharges in the amygdala. Stimulation in the ventral hippocampus, even when it produced after discharges in the dorsal hippocampus, did not influence learning. The authors concluded that

> 'while the amygdala thus seems to have a critical role in the mnemonic processes pertinent to the present study, the hippocampus probably does not' (p. 65).

Nyakas and Endröczi (1970) reached the same conclusion, as did Vardaris and Schwartz (1971).

In several studies spreading depression was elicited in the hippocampus just after training a conditioned emotional response (Avis and Carlton 1968, Hughes 1969, Kapp and Schneider 1971). In all of these studies liquid KCl was used, and propagation of depression beyond the borders of the hippocampus likely. Considerable seizure activity was noted in the first and third studies, which provided EEG records, while Hughes reported that 17 per cent of his subjects died from the injection. Though purporting to demonstrate that the hippocampus is involved in the processes underlying long-term memory storage, these studies actually show little more than the dangers inherent in the poorly controlled use of stimulation techniques.[*]

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[*] These results accord well with the lesion data. Bresnahan and Routtenberg used a step-down passive avoidance task, which rarely elicits deficits in lesioned rats. Henderson *et al.* used the runway task known to produce mixed effects. They report only latency data which often indicate a deficit after lesions, but do not report upon the number of contacts with the water spout, which is rarely increased in lesioned rats.

[*] Numerous difficulties arise in the interpretation of studies involving the injection of puromycin, an inhibitor of protein synthesis (e.g. Cohen and Barondes 1967, Cohen, Ervin, and Barondes 1966). Interference with memory storage seems related to the epileptogenic action of the drug, rather than to its suppression of macromolecular synthesis. In a carefully controlled series of studies Nakajima (1969, 1972) showed that deficits induced by actinomycin-D injected into the hippocampus, in a T-maze position habit, were related to the seizures induced by the drug, rather than to its effects upon RNA synthesis. Thus, deficits were dependent upon the injection-test interval rather than on the training-injection interval. Maximal disruption appeared only after four days, when seizure activity begins to appear in the hippocampus. RNA synthesis, on the other hand, was suppressed within a few hours of injection, but this did not seem to influence learning or retention. Nakajima (1969) suggested that propagation of seizures into the amygdala might be an important factor in the decrements observed. Such an explanation is even more compelling with regard to those early studies in the mouse which limited injections merely to the temporal area *in toto* (e.g. Flexner, Flexner, and Stellar 1965). We do not deny the likelihood that RNA and/or protein synthesis are

While these early studies, and others we have not discussed, were subject to basic methodological flaws, more recent work incorporates some of the necessary controls. A consensus seems to be arising from these studies that stimulation of the hippocampus has disturbing effects only at certain stages of the memorization process. Most of this work has concentrated upon one-trial passive avoidance tasks; an unfortunate choice, for the involvement of the hippocampus in such tasks, as we have seen, is rather variable.

Several studies employed step-down passive avoidance, a task usually unaffected by hippocampal lesions (see Table A23). Wilson and Vardaris (1972), using currents above threshold for eliciting seizures, found only a small deficit in this task. Zornetzer and Chronister (1973) and Zornetzer, Chronister, and Ross (1973), however, found that bilateral stimulation in the fascia dentata at subthreshold intensities could interfere with learning. Lastly, Kapp, Kaufman, and Repole (1974) report that step-down passive avoidance is *not* affected by post-trial stimulation, even when the current level is quite high and when there are bilateral fascia dentata placements. Clearly, no firm conclusions can be drawn from these data.

Sideroff *et al.* (1974) used a different form of passive avoidance, akin to a discriminated step-through task, and found that stimulation given either 10 s or 3 h after a single electric shock caused retention deficits, but only in terms of latency to respond. In two studies directly aimed at differentiating between short-term memory and long-term memory Kesner and Conner (1972, 1974) trained rats to lever press for continuous reward and then shocked the final lever press of the session, after which the lever was withdrawn and hippocampal stimulation applied. Retention after 1 min was good, but after either 4 min or 24 h there were deficits. This was expressed in terms of the number of lever presses and did not show up in the measure of latency to the first lever press.

In a recent extension of this work Kesner *et al.* (1975) showed that while deficits are present with stimulation-test intervals of up to 60 min, performance is normal at 180 min. If stimulation was applied a week after learning, then deficits were not seen at any stimulation-test interval. These data were all obtained in the lever press passive avoidance situation. In an active avoidance task only limited effects of stimulation were reported, and then only up to one day after stimulation. In a purely appetitive task, the acquisition of lever pressing, deficits were elicited by stimulation given one, but not seven, days after completion of training. It must be stressed that in all these studies the stimulation employed inevitably elicited hippocampal after discharges.

These latest results invert the conclusions reached from the earlier work and suggest that hippocampal seizures can interrupt some intermediate memory stage. However, without controls providing information about the spread of these seizures it is impossible to state that the effects are due to an interruption of hippocampal function, as Kesner et al. (1975) do.[*]

Another study investigating the effects of post-trial stimulation upon avoidance learning is worth mentioning. Landfield, Tusa, and McGaugh (1973) trained rats on one-way active avoidance and stimulated them 5 s after a response; in a second study a discriminated avoidance task was used. In both studies the stimulated rats were significantly better than non-stimulated controls with electrodes implanted and the authors use this difference to justify the conclusion that hippocampal stimulation facilitates the consolidation of memory. However, there was no difference between stimulated rats and unoperated controls in either task. There seems to be no justification for their conclusion.[**]

Finally, Livesey and Wearne (1973) reported large deficits in rats trained on a simultaneous brightness discrimination and stimulated either just after a response or throughout the training trials. This deficit was related to the use of a maladaptive position habit, and is thus consistent with results seen in several lesion studies. Livesey and Meyer (1975) have replicated this effect using more restricted stimulation conditions; the maximally effective treatment involved stimulation during the choice period of each trial. All rats were then trained to criterion without stimulation and retested with stimulation. Decrements were seen in some, but not all, the subjects. The authors noted, but failed to comment further upon, this dichotomous result. Once again, deficits were related to the adoption of perseverative position habits. Livesey and Bayliss (1975), however, have shown that stimulation in the fascia dentata does not interfere with the learning of this task, though it can disrupt reversal. Here, the deficit was similar to that produced by lesions; no difficulty in giving up the old response, but the adoption of a maladaptive position habit.

Though much of this stimulation work is intriguing, it is plain that

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involved in long-term memory storage, nor that the hippocampus undergoes such changes. In fact a wide variety of experiments had demonstrated biochemical changes in the hippocampus correlated with learning; many of these are discussed by Nakajima (1975). Precise study of the hippocampal role in long-term memory *demands* the use of behavioural tasks requiring locale participation and control over all the effects of drugs used, and very few of the studies presently available fit this description.

[*] Zornetzer and Chronister (1973a) have reported the use of a food-finding task which seemed to involve place learning. Here, stimulation after a trial caused a clear deficit. It would be useful to have some data on the short-term and long-term memory characteristics of this effect.

[**] Zornetzer, Boast, and Hamrick (1974) have also shown that the mere implantation of an electrode in the hippocampus can lead to deficits in one-trial passive avoidance. It is possible that stimulation merely alleviates the irritating effects of electrode implantation.

clear-cut results are not yet available.[*] [**] While we would agree that stimulation techniques can be useful and that real effects may have already been demonstrated, we would argue that at present there are too many unknowns in these studies to allow for any straightforward conclusions. In constructing a theory of hippocampal function based on the effects of its disruption it would seem better to rely upon lesion data at present.

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[*] Deutsch and his collaborators (e.g. Deutsch, Hamburg, and Dahl 1966, Deutsch and Leibowitz 1966, Wiener and Deutsch 1968) have reported that various changes in retention are a function of changes in cholinergic systems, which can be manipulated by anti-cholinergic and anti-cholinesterase drugs injected into the hippocampus. In a recent study George and Mellanby (1974) have shown that the carrier used in such studies (peanut oil) can, by itself, affect memory. This throws the earlier work into some doubt, and shows once again the problems associated with stimulation and injection techniques.

[**] Many of the studies discussed in this chapter are explored at greater length in the review article by Nakajima (1975).

13

Long-term memory

The hippocampal syndrome derives from two sources: (1) the loss of those functions depending upon the locale system; (2) the forced dependence upon taxon mechanisms. The former contributes a specific deficit in place learning and exploration, the latter a bias towards persistence and stereotyped behaviour. In considering the lesion data as reflecting these two factors we have limited our remarks to the learning (or extinction) of various behavioural tasks and have not explicitly discussed the question of the long-term storage of place information. Several experimental approaches address this question, which we can now take up in the final chapter of this section; this will form an introduction to the final section of the book, which is concerned with the functions of the hippocampus in humans.

The suggestion that the hippocampus is involved in long-term memory storage derives primarily from work with humans, but it has also been forwarded as an explanation for considerable work with infra-humans. As we have seen, both electrical and chemical stimulation studies have implicated the hippocampus in memory processes, though there are problems with the interpretation of many of these studies. In this chapter we shall consider several types of lesion experiment which provide clues to the hippocampal role in long-term memory storage.

13.1. Long-term memory storage in the locale system

The study of memory function in the hippocampus (and many other brain structures) has often proceeded under the implicit assumption that memory is a unitary phenomenon. While few investigators would state this assumption explicitly, it is none the less present, often specifying certain cortical areas as the site of permanent memory. Rather fewer researchers hold the other traditional view, associated with Lashley, that memory cannot be localized in this way. The trend of much recent research indicates that neither the non-localization nor the limited localization theories are completely acceptable. Instead, it appears that there are different types of memory, relating perhaps to different kinds of information, and that these are localized in many, possibly most, neural systems.

We have hinted at this view by emphasizing the notion that behaviour, viewed at the molar level, consists of the use of particular hypotheses, each type resting on a different form of information. The memory for these would be stored in different neural areas, corresponding perhaps to those areas responsible for specific forms of information processing.

Under this assumption there is no such thing as *the* memory area. Rather, there are memory areas, each responsible for a different form of information storage. The hippocampus, for instance, both constructs and stores cognitive maps. Behaviour which is based on place hypotheses should require hippocampal participation during initial learning as well as during subsequent retention. Behaviour which does not utilize place hypotheses can be learned in the absence of the hippocampus, and it should not be surprising to find that experiments involving the removal of the hippocampus after such learning fail to interfere with retention. These assumptions can only be tested by using tasks whose behavioural basis (in intact animals) is well understood. To demonstrate that hippocampal lesions fail to interfere with the retention of, for instance, a simultaneous visual discrimination task says nothing about the long-term memory for places.

In the course of evaluating the possibility that the hippocampus has a long-term memory function we can also briefly discuss the related issue of *recovery* of *function.* As we noted earlier (see p. 235), serially produced lesions sometimes fail to have the same effects as lesions produced in a single operation. We suggested that this phenomenon partially resided in the fact that serial lesions did not produce some of the non-specific effects, such as surgical trauma, which accompanied single-stage lesions. It was also noted that this attenuation could result either from a *substitution* or *restitution* process. The latter would involve a genuine *take over* of function, such that learning would proceed in the same way as before the lesion, that is, utilizing the same hypothesis. The former, substitution, process would involve the solution of the task by a different hypothesis, thus using different neural systems *in their normal way.* We shall argue that any take over of function after hippocampal lesions results from substitution, not restitution. Such take over should be impossible when purely place-learning tasks are involved.

13.1.1. RETENTION STUDIES

Table A30 presents the results of studies concerned with the retention of tasks trained prior to the placement of hippocampal lesions. These studies provide a mixed picture: the retention of some tasks is affected, that of others is not. Generally, tasks demanding locale function, e.g. maze learning, elicit retention deficits, while tasks not requiring locale function, e.g. simultaneous visual discrimination, are retained normally. However, this neat classification, which strongly supports the suppositions

made above concerning multiple memory storage sites, does not account for all the studies listed. We can consider the exceptions in more detail.

Studies of the retention of one-way active avoidance have produced mixed results. As we have already seen (pp. 306-8), this task can be solved with either place or taxon hypotheses. Retention tests should reflect this fact. In Niki's (1962) study hippocampal rats were impaired neither in post-operative acquisition nor in the retention of pre-operative learning. In the study by Olton and Isaacson (1968a) hippocampal rats were impaired in both. This suggests that the two experimental situations were differentially loaded in favour of taxon and place hypotheses, respectively. It is significant that identical results were obtained in both acquisition and retention.

The possibility that the hippocampus is differentially involved in particular stages of the memory process has already been raised in discussing stimulation studies; these failed to produce any clear-cut conclusion. Several lesion studies have also approached this question, again with inconclusive results. Uretsky and McCleary (1969) and Glick and Greenstein (1973) both report that the interval between learning experience and lesion can influence the results of retention tests. In the first study combined lesions of the fornix and entorhinal area 3 h, but not 8 days, after learning induced deficits in active avoidance retention. In the second study impairments in retention of a passive avoidance task were only produced with immediate lesions, not when surgery was delayed for one hour after training. These studies were interpreted as indicating an involvement of the hippocampus in some form of short-term or temporary memory, but not in the long-term memory for the same material.*

Boast, Zornetzer, and Hamrick (1975) addressed the same issue in a different way and obtained markedly different results. Lesions were made prior to training on a passive avoidance task, but retention was tested either 15 min or 25 h after training. Lesions anywhere in the hippocampal system produced deficits at 24 h, while only lesions in the fascia dentata produced deficits at 15 min. These data are difficult to interpret and are complicated by several factors. Uretsky and McCleary (1969) only obtained deficits when the entorhinal cortex was damaged; Glick and Greenstein (1973) and Boast et al. (1975) used tasks which do not necessarily involve the hippocampus. It is not possible to say anything conclusive about a differential involvement of the hippocampus in short-term and long-term memory on the basis of this work.

One final type of experiment should be discussed in this context.

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* The possible involvement of the hippocampus in short-term memory processes has been investigated with an interference paradigm in several studies. Rats were trained a task, and then an 'interfering' task was interpolated between successive trials of the first task (Walker and Means 1973, Alexander, Broome, and Means 1974, Jarrard 1975). The results of these studies were mixed; the first and last study showed abnormal interference effects, but the second did not.

Animals are taught an alley approach-avoidance task, and lesions are made at various stages of this training sequence. For instance, in Fried and Goddard (1967) the sequence consisted of approach, intermittent punishment, and then continuous punishment. Intact rats respond to this sequence in the following way: they slow down during the initial stages of intermittent punishment, then speed up again, then slow down drastically during continuous punishment. Hippocampal lesions were made either (1) before any training, (2) when the rats had slowed down during intermittent punishment, or (3) after intermittent punishment had been terminated, when the rats were running rapidly again but before continuous punishment had begun. Rats which received lesions at either the first or third stage showed poor avoidance when continuous punishment was introduced, which is in agreement with other studies of this type of passive avoidance. However, those rats which received lesions at the second stage did slow down when continuous punishment was introduced. The authors suggested that

> 'the dominant emotional state of the organism at the time of the lesion is of prime importance in determining the effect of that lesion on subsequent avoidance behaviour' (p. 329).

An alternative interpretation of these data is possible, one which brings them into line with our earlier discussions. The intermittent punishment used in these studies has many of the properties of intermittent reward (cf. Banks 1966), in that it produces persistent stereotyped behaviour. We can assume that this represents the action of variability upon the selection of behavioural strategies, leading to the substitution of taxon for place hypotheses. The slow running stage after the introduction of intermittent punishment would partly reflect the conflict between a place and a taxon hypothesis–running through a dangerous place to get reward in another place. The increase in running speed could then reflect the ascension of the running hypothesis and the abandonment of the conflicting place hypothesis. Continuous shock overcomes this taxon hypothesis and leads to the suppression of running. In the absence of a conflict between a place and a taxon hypothesis the recovery of running after the introduction of intermittent shock should be quite rapid, as Fried and Goddard report, but the strength of the ensuing taxon hypothesis might be less than in the case where a conflict existed. This could account for the better passive avoidance shown by those animals lesioned prior to conflict resolution. Our explanation here is quite speculative and not meant to suggest a final interpretation of these data. The results of these studies are quite variable, the task itself is particularly confusing, and it is perhaps best to leave such studies aside in any analysis of the hippocampal role in memory storage.

13.1.1(a). Conclusions. The general thrust of the data from retention studies is that the hippocampus is involved in at least the long-term storage of place information. Further, there does not appear to be any mechanism for replacing this function in the absence of the hippocampus. This suggests that `recovery of function' after hippocampal lesions, which we discuss in a moment, resides not in a direct take over of function, but rather in the finding of new solutions based on taxon hypotheses if possible.

The conclusion that the hippocampus is integral to the long-term storage of locale information gives this structure a central role in memory mechanisms. As such, it brings the animal data into line with data gathered in the clinic during studies on patients with known or putative hippocampal damage (see Chapter 15). The important point is that this is not a generalized long-term memory defect; it relates specifically to information stored in the locale system. This specificity might explain the failure of previous investigators in pinning down a memory function for the hippocampus. The particular type of information stored in the hippocampus must remain the central question. Our assumption that locale information is involved indicates that the hippocampal memory store would be critically important for both the detection of novelty and for the utilization of place hypotheses.

The notion that the same task can be solved in any of several ways underlines the need for extreme caution in both the planning of experiments and the interpretation of results. Too often, negative results have been taken as proof that the hippocampus had no role in long-term memory in animals. This conclusion was clearly unjustified, as later work has shown.

13.1.2. SERIAL VERSUS SINGLE-STAGE LESIONS

Additional data bearing on the problem of alternative solutions to behavioural tasks comes from those studies concerned with the `take over of function' problem. In its simplest form this area of research has been concerned with the long-term after effects of surgical procedures, making the assumption that animals suffering brain damage might, with time, acquire the ability to solve problems which were initially retarded by the operation. It has often been assumed that such recovery would reflect some reorganization of cerebral function, such that remaining brain areas were performing the function of excised areas.

Another approach to such recovery is suggested by the assumption that many behavioural tasks can be solved in more than one way. Take over of function might represent a change in the means of behavioural solution, rather than an actual reorganization of brain capacities. Such a shift would accord more comfortably with what is known of the specific anatomical and physiological properties of most extra-cortical structures as we have already pointed out.[*]

Very little work has been reported concerning the recovery of function following hippocampal lesions. In one recent study Mahut and Zola (1973) report that two years after fornix section monkeys had improved their performance on certain discrimination tasks. Any conclusions regarding this form of recovery await further long-term research.

Somewhat more data are available from studies contrasting the effects of single-stage and serial lesions. Traditional views of functional recovery would suggest that serial lesions might attenuate expected deficits in that neural reorganization would proceed more steadily. Further, a psychological statement of the same order might argue that multi-stage lesions afford the animal an opportunity to transfer the learning from one structure to another. When the target structure is totally ablated, other structures will be capable of subserving retention. It is implicit in this argument that no brain structure has a unique capability which cannot be `taken over' by another.

On the other hand, a view which emphasizes the uniqueness of particular brain structures in conjunction with the possibility of alternative solutions to the same problems would state that serial lesions will only attenuate deficits in those cases where the lesioned structure is not essential to learning. That is, tasks demanding

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[*] Another approach to the question of recovery of function is provided by the use of neonatal lesions. Here, it is possible that the brain can reorganize itself during development such that no specific deficits would be produced by the lesion. There has been little work to date on neonatal hippocampal lesion effects and what there is seems rather confusing (e.g. Isaacson et al. 1968, Nonneman and Isaacson 1973, Moorcroft 1971, Molino 1975). The first two reports, involving the testing of cats some time after neonatal lesions, suffer from the `filling in' of the lesion during development and from the fact that neocortical lesions had many of the same effects as did the aspiration hippocampal lesions. None the less, there were indications that neonatal lesions could affect a number of functions usually impaired by adult lesions, though some tasks, such as runway extinction, were unaffected by the early lesions.

Moorcroft's study does not shed much light on the recovery question, as his rats were tested only 3 days after lesions were made. For this very reason, however, these data provide information on the maturation of hippocampal functions. As we have pointed out already, there is considerable post-natal neurogenesis in the hippocampus, and the structure does not appear to be fully functional in the rat for about 3 weeks. Given this, it should be possible to demonstrate the developmental onset of locale system functions with growth. Moorcroft showed that the hyperactivity usually induced by hippocampal lesions only appears when lesions are made at 16 days or older and the rats tested at around 20 days. Of course, these data do not say much about the onset of locale function; it would be better to study normal rats at different ages in situations such as spontaneous alternation, exploration, and so on (see e.g. Douglas et al. 1973). Altman el al. (1973) have pointed out that neonatal rats bear a strong resemblance to adults with hippocampal lesions, a suggestion with which we concur, with certain reservations (see Nadel et al. 1975). Finally, Molino (1975) tested rats with either dorsal or ventral lesions made in infancy; he also observed some `filling in' of the lesion. Neither lesion affected the development of a CER, which was somewhat retarded by adult ventral lesions. Similarly, neither infant lesion facilitated two-way avoidance; only the adult ventral lesion had this effect. It should be noted that the different groups were all tested 60 days postoperatively, thus at quite different ages. The extent to which this procedure was responsible for the atypical adult data remains to be determined.

locale solutions should not benefit from serial lesions, while those which can be solved either through place or taxon hypotheses might benefit.

Five studies of recovery of function after serial lesions in hippocampus have been reported (Isaacson and Schmaltz 1968, Stein *et al.* 1969; LeVere and Weiss 1973, Dawson, Conrad, and Lynch 1973; Greene, Stauff, and Walters 1972). In two of these some recovery was seen, while in the other three serial lesions yielded the same results as did one-stage lesions. In the Stein *et al.* report the animals were tested on several tasks, in all cases after all surgical treatments had been effected. Included were tests of successive discrimination and passive avoidance, both examples of tasks in which either place or taxon hypotheses could conceivably be used. In these cases the authors report that two-stage lesions yield normal performance, while one-stage lesions yield deficits. In that the animals in this study were not given any training in the interval between the two operations, one cannot argue that the recovery of function depended upon an initial displaced learning which survived the second, and final, operation. As such, these data support the notion that serial lesions, without intervening training, are less deleterious than are single-stage lesions. Dawson *et al.* compared one-stage and two-stage lesions and provided an important control lacking in the Stein *et al.* study; their rats were tested at equivalent times after the completion of surgery. They concluded that

> `there are no distinguishable differences in the behavioural deficits produced by single-stage as compared with two-stage hippocampal lesions' (p. 275).

Thus, in their study, perseveration and hyperactivity were a constant feature of bilateral hippocampal damage, whether the lesions were made serially or in a single operation.

In the Greene *et al.* study the two-stage lesion group received some training between the two operations. Hippocampal animals with serial lesions were less impaired on a spatial alternation task than were one-stage animals. However, the serially lesioned rats were still significantly worse than were control animals. Thus, while serial lesions can overcome part of the deficit they apparently cannot eliminate it. Finally, LeVere and Weiss have provided the strongest evidence that the nature of the task and the particular brain structure involved put unique constraints on the extent of `recovery' following serial lesions. They used a task which almost inevitably produces deficits in hippocampal animals: spatial discrimination reversal. No difference was seen between animals receiving one-stage or two-stage lesions in this situation, both groups being impaired relative to controls.[*]

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[*] Douglas (1975) reported on some hitherto unpublished work demonstrating that serially produced hippocampal lesions abolish spontaneous alternation.

14

An extension of the theory to humans

14.1. Introduction

Any model of hippocampal function must account not only for data from the rat, cat, and monkey, it must make sense of data gathered in the clinic as well. For over 20 years it has been known that the hippocampus is essential in human long-term memory function, and the inability of investigators to find adequate parallels to this function in other species led to the unfortunate tendency to assume that there were major species differences regarding hippocampal function. With the advantage of hindsight we can see that this discrepancy between clinical and experimental work resulted from two related factors: (1) an inaccurate conceptualization of the nature of the memory deficit consequent upon hippocampal damage in humans, embedded within a similar set of misconceptions concerning the nature of normal human memory; (2) the use of inappropriate paradigms in work with infra-humans, which generally failed to assess the same aspects of memory affected by hippocampal damage in humans.

We have argued elsewhere (Nadel and O'Keefe 1974) that there is no valid reason to assume that differences of large magnitude exist between mammalian species in regard to the function of specific brain areas, and we are thus in strong agreement with the recent tendency to bring the clinical and experimental work together under the same theoretical umbrella (cf. Isaacson and Pribram 1975).

In the first section of the book we established the basis for the theory of cognitive mapping by considering data from a variety of species, including humans. Impressive evidence for the existence of spatial mapping comes from recent work with humans, while the philosophical background to the problem of space perception rests almost entirely on considerations of the proper basis for human knowledge. The cognitive map in infra-humans should be viewed as a spatial map in which representations of objects experienced in the environment are ordered within a framework generating a unitary space. However, the central property of the locale system is its ability to order representations in a structured context. The development of objective spatial representations is not the only possible use for such a system. We hope to show, in this final section of the book, that mapping structures can represent verbal,

as well as non-verbal, information. For both of these forms the locale system will be shown to be central to a particular form of memory: that concerned with the representation of experiences within a specific context. We shall argue that memory comes in two basic varieties: (1) memory for items, independent of the time or place of their occurrence; (2) memory for items or events within a spatio-temporal context.*

In this first chapter of the final part of the book we shall consider two theoretical issues. First, we must consider the problem of spatial orientation in humans in more detail, in order to define precisely the nature of the spatial representation supposedly subserved by the locale system. This is necessary primarily because there exists considerable confusion in clinical neuropsychology concerning the various forms of spatial representation and their neural bases. Much of this confusion results from the empiricist bias in theories of space perception, as we noted earlier (p. 52). Second, we shall discuss, in broad outline, the general problem of human memory. Amongst the reasons which make it possible to draw together the clinical and animal experimental research is that theoretical conceptions of human memory have changed radically in recent years. In discussing memory, we shall touch upon the various forms it takes, and the need to see long-term memory, for certain kinds of information, in terms of holistic structures.

14.2. Neural correlates of human spatial representation

We have contrasted two forms of space perception: absolute, or non-egocentric, and relative, or egocentric. The former was seen as a unitary, objective space in which the position of the organism did not affect the distribution of objects represented within that space. The latter, on the other hand, consisted in a variety of spaces defined by the relation between the organism and external objects, or between different parts of the organism. The spatial mapping function proposed for the hippocampus is concerned with the former, non-egocentric, space; though it receives inputs from systems concerned with relative spaces, the hippocampus does not store these egocentric representations.

According to traditional empiricist psychology objective space is generated from relative space, and this position has been incorporated into much of the clinical work in the form of the assumption that the same brain areas are responsible for both. Thus, Teuber and his colleagues

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* This distinction is similar to that drawn by Tulving (1972) between *semantic* and *episodic* memory. Many other writers have agreed that there is an important difference between memory which is essentially context free and that which incorporates, and is defined by, context. There are important differences in the detailed specification of these various dual-memory systems and we do not want to identify ourselves with any particular version of the theory at present. The dichotomy between route structures and map structures, as elaborated in Chapter 2, captures the essential differences between the context-free and context-dependent memory. As we shall see shortly (p. 398), much recent work on human memory has concentrated on those forms of memory storage which go beyond the restricted possibilities of association-based list, or route, structures.

(e.g. Teuber 1963, Semmes et al. 1955, 1963) assume that both forms of space, which they label intra- and extra-personal space, are subserved by the parietal area; this assumption seems to be unwarranted.*

In a review of the broad area of spatial disorders in clinical patients, Benton (1969) noted that

> 'Most patients who show defective absolute or relative localization of stimuli in external space do *not* show concomitant disturbances in more general topographical orientation or topographical memory ... This dissociation testifies to the essential independence of those two broad types of disorder' (p. 217).**

The fact that localization of objects in space relative to the position of the observer can be dissociated from topographical, or geographical, orientation attests to the independence of egocentric and non-egocentric space, that is, the separation between relative space and spatial maps.

While Teuber (1963) argued that disorders of both forms can be seen after parietal damage, more recent evidence suggests instead that the parietal area is involved with purely egocentric space, and the related process of recognizing objects in different perspectives (e.g. Butters and Barton 1970, Ratcliff and Newcombe 1973). Damage to the parietal area yields perceptual disturbances as well, and these, according to Benton, are not correlated with disorders of geographic memory.

From our point of view it is important to stress that the parietal area is not central to spatial mapping for two reasons: First, the fact that there is extensive post-natal development of the parietal area has been taken as evidence in support of the view that objective space cannot become available in humans until as many as 6-7 years after birth. This position, acceptable to Piaget, fails to account for the facts of spatial perception and development, as we have already seen (pp. 78-9). Second, there is a strong correlation between disorders of geographic orientation and general memory (e.g. Benson, Gardner, and Meadows 1976, Van Buren and Borke 1972); this makes little sense if one places the former in the parietal region and the latter in the hippocampus.

Thus, we allocate to the hippocampus only that aspect of spatial representation concerned with non-egocentric space. We would not expect to see, in association with hippocampal damage, deficits in body schemas, or left-right differentiation, or localization of objects relative to the organism. As in other animals, where a specific deficit in place learning was postulated, we suggest only that humans with hippocampal damage would be deficient in the representation and use of total, geographic environments.

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* Benton (1969) stated that 'there has been much theorizing to the effect that spatial knowledge of one's own body (particularly right-left differentiation) underlies the development and maintenance of the apprehension of spatial relationships among objects or events in the external environment and therefore that there must be a necessary and intimate relationship between disorders of the body schema and disorders of spatial orientation ... these theories make pleasant reading but they are not altogether in accord with the facts of clinical observation' (p. 223). Semmes *et al.* claim to have found just such an association, between two-point tactual discrimination and spatial orientation, but their claim that the latter disorder was multi-modal, and therefore a general one, is not supported by the data they present.

** Benton defines 'absolute' localization with respect to the body axis; thus it is part of what we term egocentric space. It is not to be confused with the notion of absolute space.

14.3. Human memory

14.3.1. INTRODUCTION

Any item entering the human memory system gets routed through a series of stores, some organized hierarchically, others in parallel, each analysing it and storing it in a particular form. Some of these stores form what we have called the taxon systems; others form the locale system. We cannot provide an extensive review of the literature pertaining to these processes; the interested reader is referred to many recent books for further discussion.* Rather, we shall concentrate upon those areas of central importance to an understanding of the amnesic syndrome consequent upon hippocampal damage. We shall do this by first describing the properties of human memory within the taxon and locale systems, and then attempting to bring out the correspondence between these properties and those specified by other approaches to memory.

14.3.1(a). Memory in the taxon system. As we noted earlier, there are several stages of memory within the taxon systems. The initial effect of the activation of any representation within the taxon systems is a lowered threshold in the neurones coded for that representation. During this phase some kind of temporal-order information can be preserved in the form of the relative strengths of the traces of recently activated representations. However, since the long-term effect of such activations summate, so that the most frequently experienced items have the lowest thresholds, order information is lost within the long-term taxon memory system. This is another way of saying that storage within the taxon systems cannot take temporal context into account.

Taxon memory stores are governed primarily by the principle of *category inclusion.* Each taxon category is interested in, or coded for, different aspects of items, and any given item is potentially specifiable in terms of the set of taxon categories it activates. Similar items activate similar sets, sharing many of the same neural elements. The extent of neural overlap in these representations is a measure of the similarity between the items represented; it also provides the basis for understanding the *interference*

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* Some good books are Neisser (1967), Norman (1969, 1970), Tulving and Donaldson (1972), Steinberg and Jacobovits (1971), Anderson and Bower (1973), Melton and Martin (1972), Norman and Rumelhart (1975), Weimer and Palermo (1974), and Bobrow and Collins (1975).

seen between related items, when context is not taken into account.[*]

The entry into taxon storage of several similar items results in maximum threshold reductions for those features which all items hold in common, with progressively less change in threshold for the less popular features. This gives rise to the *abstraction* of a *prototype,* which may be different from any specific item experienced but which nevertheless captures the central tendency of the group of items involved (e.g. Reed 1972, Rosch 1973, 1975, Smith, Shoben, and Rips 1974).[**]

Thus, taxon memory is characterized by several basic properties: (1) categorization by feature similarity; (2) the abstraction of prototypes representing the central features of the category members;[I] (3) the absence of spatio-temporal context coding; (4) the consequent absence of order, time and place information;[II] (5) incremental storage; (6) substantial decay with time.

14.3.1(b). Memory in the locale system. The locale system can be pictured as a long map extending from the past into the future, segments of which contain groups of representations in a fixed spatial (and hence temporal) relationship. These representations can be drawn from any of the taxon category stores and might consist, for example, of objects forming a spatial map or, of semantic categories, yielding an idea (see below, pp. 401-10).

The properties of storage within the locale system have already been enumerated; (1) preservation of spatio-temporal context; (2) single occurrence storage; (3) minimal interference between different representations of the same item; (4) multiple channels of access for the retrieval of any, or all, of the relationships embodied in the map. These properties, as we have seen, convey to the map features lacking in taxon stores, such as a freedom from dependence upon specific input or output modes. Thus, any of the relationships stored in the map can be retrieved from it by activating any portion of the map, whether or not these relationships were noticed at the time of input. This follows from the fact that the members of any map segment form the context for each item within that segment.[*]

An important difference between the rat and human locale systems lies in the development in the latter of optional *strategies* for placing items in the map, manipulating those that are already there, and relating different segments of the map to each other. This development is unlikely to have been precipitous, in the sense of the sudden emergence of language, but rather to have evolved gradually, with transitional stages seen in the cat and monkey.[**] In humans, items in the map can be moved from one location to another, generating new configurations which provide information about possible actions. The new feature provided by evolution, which possibly occurs in the cat, is the dissociation of this predictive mode from actual movements.

Further, items, and sets of items, not related to the present sensory array can be called up and manipulated. Thus, items which are never related in reality can be juxtaposed, substituted for each other, or related in other ways to create new patterns. This possibility, in addition to others allowing for the comparison of different map segments, provides the basis for operations which might be central to some of the mental activities usually referred to as thinking, imagination, and creativity. Finally, the existence of such optional strategies for information manipulation within the map, aside from enabling humans to imagine things they have never experienced (such as an elephant in a tree), lay the basis for a liberation of the mapping system from its connection with space; the relationship between items in a map need no longer represent an inevitable `real-world' spatial relationship. This feature will become obvious when we discuss the way in which the map `spatially' represents semantic relationships (pp. 401-10).

14.3.2. A MODEL OF HUMAN MEMORY

While we cannot discuss the entire range of work on human memory, or even a fraction of it, we can broadly outline the main features of memory. The distinctions we have just drawn between the memory processes

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[*] There are taxon stores based on various aspects of input items: physical form, meaning, etc. Some differentiation between items similar in one respect can be achieved by reference to a different storage system, where they might not be similar at all. One of the advantages of multi-attribute coding (cf. Underwood 1969) lies in this possibility. It appears that the activation of the different taxon stores occurs in rapid sequence, and that any representation, even the most primitive, can be maintained through active attention (cf. Posner and Warren 1972); we shall discuss these processes in the context of an analysis of short-term memory (pp. 386-9).

[**] These categories contain information about the extent of similarity between the members and about the relationship between each member and the prototype; the latter relationship is asymmetrical. That is, the `distance' from the prototype to any member is different than from that member to the prototype. These asymmetries are reminiscent of what is seen in subjectively loaded mental maps (p. 77) and indicate that, for both verbal and non-verbal information, the overlap model we have postulated is somewhat oversimplified. It is interesting to note that Leibniz's model, which does not seem to work for the locale system, could be applicable to the taxon systems.

[I] As Palmer (1975) pointed out, such central tendency prototypes are not useful in the description of all categories and other principles might be involved in the establishment, for instance, of a category concerned with items which do not vary continuously over a range of possible values.

[II] We are referring here to long-term information.

[*] The similarity between our map formulation and other recent proposals concerning long-term memory (e.g. Norman and Rumelhart 1975) will be brought out more clearly in later discussion (p. 398).

[**] We suspect that this is mirrored by changes in the role of the frontal cortex, portions of which exert more and more direct control over the hippocampus with phylogenetic development, culminating in direct fronto-hippocampal connections in the monkey (see p. 131). There is, as yet, insufficient information to extend our analysis, though it seems likely that the middle portion of the sulcus principalis is the crucial area (cf. Van Hoesen, Pandya, and Butters 1972). This area projects to the septum in some species and indirectly to the entorhinal cortex or hippocampus in others. Another indication of this shift is seen in the correlates of theta activity, which serves as the locative input (or coding strategy) in the mapping system. Correlated with movement in the rat in most instances, theta becomes more and more dependent upon non-movement processes in higher species, as seen in the shift towards a predominance of low-frequency theta. Finally, the increasing importance of the CA1 field (see p. 106), as documented by Stephan (1975), also attests to the shift in capabilities of the hippocampus with phylogeny. This field is responsible for the misplace system driving curiosity and information-seeking behaviour.

mediated within the taxon and locale systems will be presented in a more familiar context in what follows.

Norman (1970) summarized what can be taken as a representative view of the various forms of human memory:

> 'First, newly presented information would appear to be transformed by the sensory system into its physiological representation . . . and this representation is stored briefly in a sensory information storage system. Following this sensory storage, the presented material is identified and encoded into a new format and retained temporarily in a different storage system, usually called short-term memory. Then, if extra attention is paid to the material, or if it is rehearsed frequently enough, or if it gets properly organized, then information is transferred to a more permanent memory system' (p. 2)

though it is considerably out of date in some respects. Three sequential stages are postulated in this type of model: immediate memory, short-term memory, and long-term memory. Most traditional memory models, including those based on clinical observation, have emphasized the way in which inputs flow through a variety of passive information processors. More recent models, on the other hand, have moved away from

> 'rigid boxes of stores and towards a flexible system with a large part played by such optional control processes as attention, rehearsal and strategies' (Craik 1971, p. 236).

This shift reflects a profound change in thinking about the nature of long-term memory systems, accompanied by an interest in the active 'control' processes required for gaining access to, and effecting retrieval from, these systems. The distinction we have drawn, along with many others, between context-free taxon category memory and context-dependent locale memory is but one manifestation of this change.

Traditional associationistic models of human memory failed in much the same way that S-R explanations of animal behaviour failed.[*] Two related areas of research, concerned with the process of imagery and the basis for language comprehension, have combined to lay the basis for new conceptions of human memory. Before describing these developments we can briefly outline the properties of the earlier stages of memory referred to by Norman. These, it will be seen, correspond largely to what we have labelled taxon memory.

14.3.2(a). Short-term memory processes. The first physiological representation of an input, usually referred to as iconic (visual) or echoic (auditory) memory, can establish a persistence which outlasts the duration of the stimulus by approximately 150 ms if actual stimulus duration is brief. Extensive analysis takes place during this time, resulting in a list of physical features which define the input; this presumably occurs in neurones like those described by Hubel and Wiesel (1962) and numerous other workers. This processing occurs automatically, unaffected by prior learning.[*] Interactions between separate inputs can take place, on the basis of physical (spatial and/or temporal) overlap, such that adjacent items affect one another's processing, as seen in studies of masking and metacontrast (cf. Weisstein 1969). The information provided by iconic and echoic processing allows the observer to *construct* the environment. First, the total stimulating field is segmented into coherent units as in figure-ground separation or stream segregation (e.g. Bregman and Campbell 1971). This segmentation precedes identification; evidence from cataract-removal patients (von Senden 1932) and from clinical studies (Macrae and Trolle 1957) supports this distinction between what Hebb (1949) called *figural unity* and *figural identity*. Second, a set of transitional stages leads to the development of identifiable *physical traces* (e.g. Crowder and Morton 1969) which could persist for as long as 10 s (Phillips and Baddeley 1971, Entus and Bindra 1970).[**] At more or less the same time, the 'meaning' of the input is encoded; that is, the neural representation within the *semantic* category to which items belong is activated. Thus, a word is coded both for its physical appearance (and/or acoustic properties) and its meaning within a brief period after input (cf. Posner and Warren 1972).

All these 'traces' constitute short-term activations within taxon category stores,[I] and the subsequent fate of the information stored in this way depends entirely upon the active control processes alluded to above. Though activation of a category representation will have long-term consequences, it is not sufficient to preserve spatio-temporal contextual information. This latter requires the establishment of memory within a different system; most control processes can be viewed as potential mediators of this mechanism.[II]

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[*] Anderson and Bower (1973) recapitulate this history and propose a neo-associationist theory which improves upon, but does not ultimately save, this approach.

[*] We are excluding the developmental effects described by Hirsch and Spinelli (1971), Blakemore and Cooper (1970), and others. Should these prove to be true, they would be applicable only to an early critical stage in the development of the organism.

[**] The persistence elicited in these trace systems presumably underlies such phenomena as `priming' (e.g. Leeper 1935, Epstein and Rock 1960, and others), where the presentation of an item raises, for a brief period, the likelihood that it will be perceived upon subsequent presentation.

[I] We shall not discuss the evidence concerning hemispheric specialization at any length. Work with normal subjects, patients with unilateral brain damage, and patients with hemispheric disconnection has demonstrated that in humans the two hemispheres are largely specialized for different functions (cf. White 1969, Milner 1971, Gazzaniga 1970). Thus, taxon stores for verbal information would be located in the left hemisphere and those for non-verbal information in the right hemisphere. This specialization is preserved when one moves to locale memory, as we shall see later (p. 424).

[II] A number of memory-improving strategies depend upon manipulating inputs in ways which reduce the load on these context-free stores, though they still fail to preserve context in the spatiotemporal sense. Thus, nonsense syllables can be recoded as words (BLK-BLACK), acronyms can be used (International Workers of the World-IWW), individual words can be combined into a chain (natural language mediation, Prytulak 1971), and complex reduction rules can be applied to material as well as various substitution codes used, as described in the popular literature (e.g. Lorayne 1968). Such procedures can serve to decrease interference, but fail to achieve context-specific memory. In

The short-term memory resulting from activation of category representations has the characteristics of limited capacity, short duration, and the preservation of item and order information. This applies to both verbal and non-verbal information; however, there appears to be an important difference between the two. Aside from the various stores noted above, verbal information is apparently represented in a separate short-term system coded for the articulatory features of words.* This system possibly functions in both the comprehension and generation of speech, as we shall see later (p. 401).

Information can be maintained in the short-term memory state through the active process of rehearsal, which amounts to a recirculation of information and which might be restricted primarily to verbal items and to the articulatory code just mentioned. Rehearsal seems to be necessitated by the limiting features of the short-term memory process. The high reliability of retention for recent inputs and the strong order information available for such inputs indicates that access is achieved simply through 'testing' memory locations for recently used representations; those most recently used will have lowered thresholds. The capacity of the short-term memory system, in this view, is a joint function of the signal-noise detection capability of the system, the decay rate of the threshold change, and the rate at which representations can be activated. Rehearsal counteracts decay, but cannot enlarge the capacity of the system.**

Aside from its role in short-term retention and any specialized function in language comprehension and generation, short-term memory functions to maintain items in a state of accessibility until they can be encoded into a contextual long-term memory system. The set of items in short-term memory, which could include items called up from prior experience, must be encoded into long-term memory in such a way that activation of any member of the set activates retrieval of the remainder; in our view this involves locating the set in the locale system's cognitive map structure.

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the same way the property of category inclusion can be utilized to improve recall, as shown in the work of Mandler (1967), Tulving (1966), and Bower *et al.* (1969), where knowledge of the categorial relationship between items in a list can serve to reduce the amount of information that needs to be retrieved. By themselves however, these reduction procedures would probably have minimal effects, unless they were made available at the time of recall (see p. 417). Their usefulness likely relates to the fact that they also reduce the amount of material that must be encoded into a contextual memory store, a process which requires time, and would therefore benefit from any such reduction in situations where speed of encoding is important. In contrast to these reduction strategies, techniques which facilitate contextual coding, as we shall see shortly, are useful when presented at the time of information input and are considerably more effective.

* Warrington and Shallice (1969) and Shallice and Warrington (1970) have provided strong evidence that this is a separate short-term memory store, located in a discrete neural area (the posterior parietal region), and not part of any causal short-term memory to long-term memory chain. The clinical syndrome of *conduction aphasia* represents the selective loss of this capability, with preserved long-term memory capabilities.

** Continued rehearsal will yield a 'better' long-term trace in the taxon system; the marginal effectiveness of pure rote learning is presumably due to the potentiating effects of sheer repetition.

We assume that finding and applying an appropriate coding strategy for a set of items takes time, and that short-term memory and rehearsal provide this time by maintaining the items in an accessible state.

*14.3.2(b). Contextual long-term memory.** Two areas of research attest to the central importance of context in long-term memory: (1) work on imagery (e.g. Bower 1970) indicates that storing information in an interactive, relational form vastly improves retention; (2) research in psycholinguistics indicates that *(a)* long-term memory for connected discourse (sentences, narrative) takes the form of retention of the meaning, or sense, of the discourse, typically with a loss of specific lexical elements and syntactic information (e.g. Johnson-Laird 1970), *(b)* analysis of the solely surface elements of language will not suffice to explain either the infinite generative possibilities of language or its comprehension, and (*c*) it appears necessary to postulate some semantic *deep structure* which incorporates the meaning of discourse without preserving lexical elements. Most contemporary investigators are in agreement in assuming that such a deep structure necessitates an abstract interactive system characterizing the relations between stored elements (cf. Weimer and Palermo 1974, Norman and Rumelhart 1975).

We would argue that a map structure can provide the basis for understanding the above facts and that it could serve as the form within which deep structure is articulated. Some discussion of work on imagery and language will support this position.

The function and nature of imagery. The demonstration that concrete words are recalled better than abstract words (cf. Paivio 1971) gave credence to the possibility that the chance to encode memory in imaginal form would improve long-term memory.** The independence of some form of imaginal coding from verbal coding had already been established (e.g. Brooks 1968) and the study of imagery was on its way. Subsequent work has verified the facilitatory effects of forming images between paired associates (Bugelski, Kidd, and Segmen 1968),*** the importance of an interaction, preferably spatial, within the image (e.g. Bower 1970), and the low interference seen between items remembered through the use of imagery coding.

The importance of spatial context in these effects is brought out clearly in the study of 'the method of loci', an imaginal technique known

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* By contextual memory we mean coding within a spatio-temporal framework.

** Miller, Galanter, and Pribram (1960) had previously described the facilitating effects of a memory strategy dependent upon imagery, the one-bun, two-shoe technique.

*** Facilitation in this study depended upon a certain minimal inter-pair interval; this presumably relates to the time needed to find an appropriate image. This result has been replicated in other work.

to the ancients and described by Yates (1966) in her book *The art of memory* as well as by Luria (1969). In this technique the subject memorizes the layout of some building, or the arrangement of shops on a street, or any geographical entity which is composed of a number of discrete loci. When desiring to remember a set of items the subject literally 'walks' through these loci and commits an item to each one by forming an image between the item and any distinguishing feature of that locus. Retrieval of items is achieved by 'walking' through the loci, allowing the latter to activate the desired items. The efficacy of this technique has been well established (Ross and Lawrence 1968, Crovitz 1969, 1971, Briggs, Hawkins and Crovitz 1970, Lea 1975), as is the minimal interference seen with its use.

Results such as these had the initial effect of misleading some investigators into an oversimplified view of the nature of imagery (cf. Neisser 1972), epitomized perhaps by Paivio's assumption that imagery was quite distinct from language, that it involved a different form of coding than did the latter, and that it worked through the storage of something like a picture of the items to be remembered (cf. Paivio 1971). This latter assumption has been strongly criticized by Pylyshyn (1973), who suggested instead that some abstract structure generated in the process of imaging is stored in long-term memory, that this structure represents, though not literally, the relations between the elements in the image, and that the image must be reconstructed upon recall. Work with both visual images (Neisser and Kerr 1973, Kosslyn 1975) and sentence memory (Kosslyn and Bower 1974, Begg 1971, Anderson 1972) supports this constructivist view, indicating that the facilitative effects of imagery are not due to the long-term storage of a literal image, but rather to the storage of some deep structure analogue which can re-establish the image if required. In view of this, the facilitative effects of the 'method of loci' can be seen to derive from the ease with which the overall spatial image can be reconstructed. In addition, these newer data and the interpretation put upon them by most investigators seem to invalidate Paivio's notion that language and imagery work on different coding structures; both apparently demand some abstract deep-structure representation.

To recapitulate, imagery seems to work through the long-term storage of an abstract information set which captures the relationships between an interacting group of elements and from which the image can be easily reconstructed. This reconstruction process can start anywhere in the 'scene' and the image itself can be scanned in any direction (Lea 1975). This is consistent with the notion that the deep-structure analogue stored in long-term memory is one in which all the elements are interrelated and simultaneously available,* e.g. something like a map.

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* Kosslyn's (1975) study indicates that the literal image is reconstructed within the taxon system.

14.3.2(c). Language. The assumption that some abstract deep-structure base characterizes long-term memory first arose within psycholinguistics. We can briefly review this development, concentrating on evidence demonstrating that there are two stages in language processing, one involving the serial ordering of a linear string of symbols, the other consisting of an underlying non-linear structure from which this ordered string is generated. The similarities between these processes, and those just described for imagery, will become clear in the course of our discussion. As there is considerably more information available concerning deep structure in language than there is for imagery, we can go into some more detail here; the features of deep structure demonstrated for language are probably also to be found in the imagery system.

The existence of a deep structure. One approach to language, as to any form of behaviour, is to attempt to explain it solely on the basis of its observable linear structure. This approach, associated with the behaviourist school, suggests that each element of a sentence is generated in response to preceding elements, or in response to a stimulus in the environment, and that the whole sentence can be thought of as a Markov chain. Given the first word of a sentence, any other word has a finite probability of being produced, depending upon the number of times in the past that the word followed the first. Thus, a word like `smelly' would be followed quite often by `feet' or `cheese', less frequently by `music' or `airplane', and virtually never by `for' or `thinks'. Higher-order Markov chains would take into account not just the previous word but the previous two words, three words, and so on. Language, on this model, is generated solely by a system which produces strings of symbols in an ordered left-to-right linear sequence. Highly practiced sequences would be run off without recourse to decisions, ideas, etc. From this point of view there is nothing unusual about Lashley's colleague who claimed that '... he could arise before an audience, turn his mouth loose and go to sleep. He believed in the peripheral chain theory of language' (Beach *et al.* 1960, pp. 510-11).

Another aspect of language emphasized by the behaviourists is the

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* There was some evidence in Lea (1975) that certain starting sites in a spatial array are more easily accessed than others; this seemed related to either top-to-bottom scanning methods or to some subjective impact of the objects located at those sites. This is, as Lea points out, an area requiring further research. Lea also failed to find any relationship between the reaction time required to scan from one site to another, and the real-world distance captured by the image of those sites; this is in disagreement with results reported by Kosslyn (1973). It is not necessarily the case, however, that a failure to find an increase in reaction times indicates that the image does not represent increased distances. There is no reason to assume, within a neural mapping structure, that real-world distances would be correlated with neural distances in a fashion which would produce orderly changes in reaction times with changes in imaged distances.

referential nature of meaning; that is, the way words refer to things or events and appear to derive their meanings from this reference. This connection comes about as the result of a simple conditioning process; a sound experienced in the presence of an object will, when later heard by itself, call up the same, or some of the same, responses as the object itself. The meaning of a sequence of sounds or words would then be given by the sum total of the conditioned meanings of each individual sound or word. In the face of harsh criticisms, to be mentioned below, this strong position has been progressively modified and weakened. In one recent formulation (Osgood 1971) meaning was seen as dependent upon some sort of internal response (r_m) which was derived from the total external response to the object. Words are not conditioned to the external responses but to these r_ms. The meanings of more abstract words, such as justice, are derived in a secondary fashion from the r_ms associated with actual objects or events.

This simple behavourist approach, which emphasizes the observable aspects of language, does seem to explain adequately many of the stereotyped features of language and some of the simpler referential features of meaning. It fails, however, in the language sphere in exactly those places where it fails in its explanation of behaviour in general, human or infra-human; it ignores or denies the purposeful variability and originality of behaviour, the novel behaviour not obviously due to generalization, the flexible use of behaviour learned in one situation but applied for a different purpose in another, and the underlying similarity amongst superficially different behaviours. These aspects of behaviour become acutely obvious in language and it is here that the deficiencies of the behaviourist account are most glaring. As pointed out by Lashley (1951), Chomsky (1957 and elsewhere), and Fodor (1965):

(1) Novel sentences constitute a large proportion of all utterances.
(2) The related words in a sentence often are not contiguous. The sentence 'the man who lived in the house sneezed' derives its meaning from the noncontiguous elements 'the man ... sneezed' and not from the contiguous elements 'the house sneezed'.
(3) Superficially different sentences such as the active `the boy hit the dog' and the passive `the dog was hit by the boy' have the same meaning.
(4) The same sound can have more than one meaning.*

It is hard to see how reference to any response, or partial response, or hidden partial response will remove the ambiguity associated with the use of sounds with two different meanings. Disambiguation almost always depends on the context within which the sound occurs. It is to explain the existence and importance of these features of language that Chomsky and most subsequent writers on linguistics have postulated the presence of a deep structure in addition to the more superficial one which generates the left-to-right temporally ordered pattern of the observed behaviour.

The form of the deep structure. The deep structure was designed to account for the creative aspects of language, the connectedness between non-contiguous surface elements, the different meanings of a sound depending on its context, and the relationship between such superficially different sentences as the active and passive forms. In Chomsky's (1957, 1965) systems the deep structure consists of a complex set of rules which operate on symbols or strings of symbols, as well as the structures which the application of the rules generates. Chomsky's grammar has three sets of rules: the most important of these are *syntactic,* the others are *semantic* and *phonological.* The syntactic rules were seen as the creative part of the system, generating the basic sentence structure; the other rules acted passively on the inputs they received from the syntactic component to generate meaning and phonological representations.

Let us briefly consider the rules of the syntactic component of Chomsky's grammar and the structures that they generate. There are two different types of syntactic rule, *phrase-structure* rules and *transformational* rules. The phrase-structure rules operate upon symbols for grammatical categories such as noun phrase (NP) and verb phrase (VP), rewriting them as strings of symbols. Fig. 33 gives some examples of the operation of these rules. Note that these rules operate on individual symbols, and that no account is taken of the history of that symbol or of the derivation of the string in which it is embedded. A noun phrase receives the same treatment wherever it appears. The structure generated by the operation of the phrase structure rules is often portrayed as a tree diagram called a *phrase marker* (see Fig. 34); this seems to be viewed by Chomsky as a static structure, all parts of which exist simultaneously in the base component of the grammar.

Transformational rules were introduced when it was seen that although phrase-structure rules could generate simple active sentences, by themselves they could not account for such things as passive sentences or questions. The transformational rules are applied to the deep structures generated by the base component and differ from the phrase-structure rules in that they apply to strings of symbols, are applicable only in a fixed order, and take into account the history or derivation of the string upon which they operate. This last property means that, in a sense, they operate upon whole phrase markers. By allowing for the optional (Chomsky 1957) or obligatory (Chomsky 1965) addition, deletion, or re-ordering of elements within a string, they easily provide for the transformation of sentences from, for instance, active to passive or declarative to interrogative (see Fig. 35).

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* It is not clear whether one should speak of one word with two meanings or two words which sound alike (homophones).

Rewrite SENTENCE as NOUN PHRASE (NP)+VERB PHRASE (VP)
S ⟶ NP + VP

Rewrite NOUN PHRASE as DETERMINER (DET)+NOUN (N)
NP ⟶ DET + N

Rewrite VERB PHRASE as VERB (V)+NOUN PHRASE (NP)
VP ⟶ V + NP

Rewrite NOUN (N) as dog, boy
N dog, boy

Rewrite DETERMINER (DET) as the
DET ⟶ the

FIG. 33. Examples of phrase-structure rules for the sentence `The dog bites the boy'.

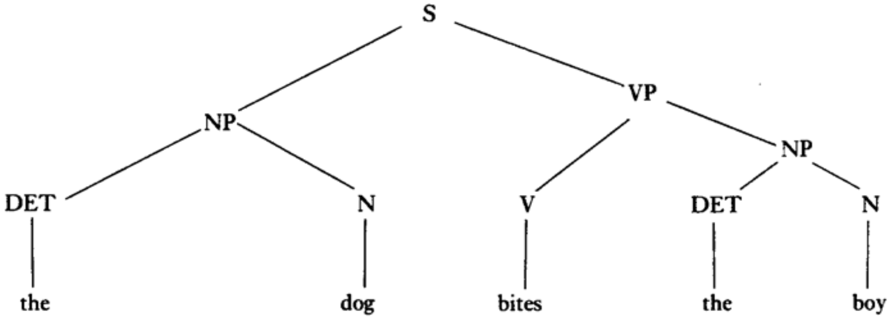

FIG. 34. An example of a phrase marker for the same sentence as in Fig. 33.

$\text{DET}_I + \text{N}_I + \text{V} + \text{DET}_2 + \text{N}_2 \longrightarrow$

The dog bites the boy

$\text{DET}_2 + \text{N}_2 + \text{BE} + \text{V} + \text{EN} + \text{BY} + \text{DET}_I + \text{N}_I$

The boy is bitten by the dog

FIG. 35. An example of the transformation rule for converting active to passive sentences. Same sentence as in Fig. 33.

In Chomsky's theory the meaning of a sentence is assigned to it by a separate component, the semantic component. This acts passively on the terminal string of the phrase marker, fitting meanings to each of the elements. Meanings are fully determined by the nature of the input from the syntactic component to the semantic component. Katz and his colleagues (Katz and Fodor 1963, Katz and Postal 1964, Katz 1972) have constructed a theory for this kind of semantic component, envisaging it as composed of two parts: a dictionary of meanings, and a set of projective rules allowing for, and providing meanings of, combinations of items.*

Chomsky's system succeeds in doing what it set out to do. It accounts for many of the interesting features of languages which fall outside the province of simple behaviourist models; it permits the generation of an infinite number of sentences from a finite number of rules; it explains why distant elements of a sentence can have strong relationships; it answers to our intuitive feeling of a similarity between syntactically different sentences by identifying a common deep structure. However, as Chomsky himself noted (1965, p. 162), it fails in one important respect; it does not capture the still deeper semantic relationships which can exist between syntactically different sentences. Thus, the grammar fails to capture the similarity between sentences A and B, or C and D:

(A) I liked the play.
(B) The play pleased me.
(C) John bought the book from Bill.
(D) Bill sold the book to John.

This failure to account for *paraphrases* would appear to be due to the narrow definition of the semantic component of the system. As we have seen, it is purely a passive feature of the grammar, whose function is to ascribe meaning to the deep structures generated by the base component. Intuitively, this seems to be an unnecessary restriction on the role of the semantic component. The meaning of a sentence is *not* only the sum total of the meanings of the words but includes the way in which they are put together. In this broader sense of semantic the base structure itself should be included in the semantic, and not in the syntactic, component.

This broader usage of semantic requires some elaboration, for it embodies an important shift in thinking about language comprehension. The behaviourist emphasis upon the elements of speech meant that most research was concerned with individual items; how they were processed,

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* The dictionary operates on the basis of *componential analysis*, specifying the meaning of a lexical element as the set of categories within which it is included (*semantic markers*) together with those features which separate it from other lexical items in the same categories (*distinguishers*). The technique of componential analysis has also been applied to verbs, in particular by Bendix (1966, 1971). He examined a number of verbs and showed that they could all be paraphrased by combinations of a few basic verbs such as have, cause, change, etc.

stored, interpreted, and generated. Thus, standard experiments involved the learning of lists of words or paired associates. When organizational factors were allowed (cf. Mandler 1967), they were generally restricted to the meaning relationships between isolated words. This accounts for the notion of *categories* and the host of experiments on the role of categorial relations in the learning of word lists and paired associates. Chomsky's critique partly embodied the notion that lexical items were not the central elements in language comprehension. However, in moving to higher-order units Chomsky did not expand the semantic component to include the meaning of these larger units.

Most of the recent work concerned with semantic deep structures, then, concentrates upon the mechanisms for comprehending and storing these higher-order verbal units, beginning with the recognition that what is remembered of sentences, paragraphs, or even stories is the sense of the discourse as a whole. Before turning to a brief discussion of some of this work it is worth digressing momentarily to discuss Tulving's (1972) notion of semantic memory. In view of the shift we have just described, it is unfortunate that Tulving chose to apply the term semantic in its older usage to a system representing the meaning of individual words, independent of context. The confusion arising from this usage has led some (e.g. Schank 1975) to reject the notion of semantic memory entirely in favour of a system including only the lexicon and episodic memory. According to Schank, the meaning of individual words is stored in the lexicon, while any relations between individual items must be stored in terms of some event in which they took part. We cannot agree with Schank on this point, though we find his model for semantic deep structure (see p. 398) one of the most attractive in the field. While we do not accept Tulving's separation *in toto,* we think there is strong evidence for a separation between some form of context-free memory, using (in the old sense) semantic categories, and a context-dependent memory, using something like a spatio-temporal framework. As we shall see in the next chapter, the data from amnesic patients supports this distinction.

Semantic deep structure. The work of Bransford, Franks, and their colleagues (e.g. Bransford and Franks 1971, Bransford, Barclay, and Franks 1972, Bransford and Johnson 1973, Bransford and McCarrell 1974, Franks 1974) provides important clues to the nature of memory for higher-order verbal units. Their early work demonstrated that, given a set of related sentences, subjects formed something like a prototype sentence which, though never actually seen, was more readily recognized as familiar than sentences which had been seen. Later work extended this observation by showing that the remembered representation for a sentence depended upon the context within which it was seen, as well as upon various inferences and assumptions the subjects could make about the material, presumably based on some prior knowledge of the contexts within which the events described could obtain. In fact, given an inappropriate context, a sentence which would have been understood in isolation was often judged incomprehensible. Similarly, they argued, some sentences which would be meaningless in isolation can be given some sense by the context within which they occur.

This work on sentence comprehension requires a model which provides for some deep structure that codes the relationship between the various elements in the sentence or between several sentences. Studies of semantic deep structure have concentrated upon such models, in the hope of specifying the form within which these relationships could be coded such that the meaning of a sentence as a whole could be stored, paraphrases of that sentence recognized, sentences could influence one another's representations, and prior information could be brought to bear on comprehension of inputs (and, hence, the meaning attached to these).

Early work on the basis for a semantic deep structure (e.g. Bendix 1966, 1971, Fillmore 1968, 1971, McCawley 1968, 1971, Lakoff 1971) spoke primarily to the first two of these requirements, concentrating upon sentence comprehension in isolation. Though superseded by later models, we shall describe Fillmore's system as it presents some of the basic features of those which superseded it. According to Fillmore, a *case system,* in which items were unordered though identified as to function, would provide a more appropriate base than the ordered set of grammatical categories proposed by Chomsky. In Fillmore's system the sentence is represented by its *modality,* which specifies such conditions as tense, negation, and mood of the sentence as a whole, and *proposition*, which identifies the verb and its permissible cases. These latter are given as an unordered set, with each case defining the relationship between the item in that case and the verb. Fillmore specified eight deep-structure cases:

(*a*) Agent—the instigator of the event
(*b*) Counter-agent—the resistance against which the action occurs
(*c*) Object—the entity acted upon or under consideration
(*d*) Result—the entity that ensues from the action
(*e*) Instrument—the immediate cause of the action
(*f*) Source—the place from which some entity moves
(*g*) Goal—the place to which some entity moves
(*h*) Experiencer—the entity receiving, accepting, undergoing, or experiencing the effect of an action

In the sentence *John opened the door with the key,* John is the agent, door the object, and key the instrument. The deep structure of each simple sentence would consist in a verb plus its obligatory and optional cases. *Open,* for example, always requires an object, but takes an agent and an instrument as options. The transformational rules in Fillmore's system, as in Chomsky's, are concerned with generating surface sentences from deep structures. However, since the cases in Fillmore's semantic deep structure are unordered, there is no need for rules which transpose elements. Instead, the rules establish a hierarchy amongst the cases associated with a verb, specifying which grammatical role each case will play in the surface sentence. For *open,* the instrument is the subject if it occurs alone, but the object of a prepositional phrase (with the *key)* if there is an agent. Fillmore's grammar does require deletion rules, because cases are represented in the deep structure by prepositional phrases which, in most circumstances, do not appear in the surface structure. Thus, the agent in our example would be represented in deep structure as *by John.* The preposition would survive in the surface sentence only in the passive case; in the active form the *by* would be deleted by a transformation rule.

This type of semantic deep structure, important for its emphasis upon functions and actions, can account for many of the facts of sentence comprehension. However, it remains silent on the more complex problems delimited by Bransford, Franks, and others, and those represented by the retention of the sense of paragraphs or entire stories. Three recent models which are specifically pitched at this level seem particularly interesting, those of Schank (1972, 1975), Norman and Rumelhart (1975) and Jackendoff (1976). Common to these approaches is the assumption that the deep-structure representation for language is some form of propositional or conceptual network which codes meaning through the interaction of elements. Thus, for Schank (1975) the basis of human memory is the *conceptualization,* which is 'action-based with certain specified associative links between actions and objects' (p. 259). Similarly, for Norman and Rumelhart (1975) the basis is the *active structural network,* which is a semantic network representing the underlying propositions in any stored event. Both systems rely on a set of primitives which define the forms of interaction between the elements in the memory structures; here they follow in the path of Bendix's componential analysis of verbs. Further, both argue that sentence after sentence can be 'added' to the memory structure, in some cases being influenced by what is already there, in other cases influencing it. Thus, they provide models for the comprehension of sets of sentences. More recent work by Rumelhart (1975) attempts to provide the basis for a representational network which would describe the structure of an entire story without building sentence upon sentence.

While we cannot explore these models in detail, it is worth emphasizing the fact that they insist upon a network-like propositional representation where the elements within the network are related to one another through the action of a primitive set of operators. The meaning of such a network, or conceptualization, is the totality of the relationships embodied within it. We find it particularly heartening that Norman and Rumelhart

emphasize the essential non-linguistic character of their networks; they apply their analysis to imagery phenomena as well as to linguistic deep structure. Here, they also stress the view that imagery depends, not on a pictorialization within memory, but rather upon some propositional deep structure which captures the relationships embodied in the image and from which the image can be reconstructed.

We will conclude this section on deep structure models with a discussion of Jackendoff's system (Jackendoff, 1976) which is, for us, the most interesting and exciting of the recent proposals. Jackendoff, expanding an original suggestion by Gruber 1965, has proposed that all sentences have deep semantic structures which are formally analagous to the subset of sentences describing events or states of affairs in physical space. First he shows how an analysis similar to the one by Fillmore described above will provide a deep structure for sentences about the location and movement of entities in physical space and, second, he shows how modifications and extensions of this purely spatial system can account for the meanings of non-spatial sentences.

In his analysis of spatial sentences, he starts with examples like:

(1) The train travelled from Detroit to Cincinnati
(2) The hawk flew from its nest to the ground
(3) The rock fell from the roof to the ground

and shows how their meanings can be captured by a deep structure which specifies the thematic relations between the verb and the nouns or noun phrases. Thus (1) would be represented by the deep structure function, GO; the theme of the function, *train;* the source or place from which the movement started, *Detroit;* and the goal or place where the movement ends, *Cincinnati.* Notice the similarity to Fillmore's case system described above. Spatial sentences (2) and (3) above would have similar deep structures with suitable additional information such as the manner of the motion. Other spatial sentences such as

(4) Max is in Africa
(5) The cat lay on the couch
(6) The bacteria stayed in his body
(7) Bill kept the book on the shelf

describe not the motion of the object or theme but its location and are represented by the deep structure function BE (4 and 5) or STAY (6 and 7).

Thus all states of affairs and events in physical space can be represented in Jackendoff's system by three functions GO, BE, and STAY, together with the things and places which these functions relate. Agency and causation are added to the deep structure by the higher order functions CAUSE and LET which apply not to entities but to events. Thus if sentence (3) above would be represented by GO (THE ROCK, THE ROOF, THE GROUND), then

(8) Linda threw the rock from the roof to the ground
(9) Linda dropped the rock from the roof to the ground

would be represented as CAUSE (LINDA, GO (THE ROCK, THE ROOF, THE GROUND)) and LET (LINDA, GO (THE ROCK, THE ROOF, THE GROUND)) respectively.

At this point Jackendoff takes a crucial step. He claims that nonspatial sentences have exactly the same representation except that the functions GO, BE, and STAY do not refer to the spatial location of entities but to the possessive, identificational, or circumstantial 'location' of entities. Let us look at possessive GO. While spatial or positional GO signifies the movement of an entity from one physical location to another, possessive GO signifies the movement of an entity from one possessive location to another. The sentence

(10) Harry gave the book to the library

is represented as possessive GO (THE BOOK, HARRY, THE LIBRARY). Similarly

(11) The book belonged to the library
(12) The library kept the book

are represented by possessive BE (THE BOOK, THE LIBRARY) and possessive STAY (THE BOOK, THE LIBRARY). The analysis of sentences about continuing states of identity or changes of identity, or continuing or changing circumstances, are given a similar treatment. Jackendoff shows, for example, that the semantic analysis of the sentence

(13) Linda kept Laura from screaming

is exactly parallel to the sentence about physical prevention

(14) Linda kept Laura (away) from the cookie jar

except that the avoided location is a circumstance in example (13). Thus, for example, the same rules of inference allow us to conclude that Laura did not scream (13) nor did she get to the cookie jar (14).

Jackendoff has not extended his analysis into the domain of completely abstract concepts nor to verbs referring to internal states or beliefs, but he sees no insurmountable obstacle to such a programme, nor do we. It is also reasonable to assume that this type of analysis can be extended to deal with units of speech longer than a sentence, thus incorporating the recent work on discourse and narrative comprehension. In summary, Jackendoff says:

> 'I consider it a striking property of the present system that simple principles, framed in terms of physical space, can be stated formally in such a way as to generalize to domains that bear no *a priori* relation to physical space' (Jackendoff, 1976 p. 121).

On linguistic grounds, then, it appears necessary to postulate the existence of several different mechanisms underlying the production and understanding of language. In addition to those mechanisms which select the appropriate words and sentence frames to produce the temporal left-to-right ordered structure of the surface aspects of language, there must be a deeper, more abstract level which carries the sense of a sentence or a set of sentences.* The common element that all deep structures share is their non-temporal aspect; put another way, they can all be represented by purely static spatial structures. The sense of an item is derived from its relation to other items within the structure, the overall sense of the sentence follows from the total configuration, while the interaction between such configurations allows for higher-order messages such as stories.

In terms of our model all of these deep-structure elements are identified with maps in the locale system (or their activation). The surface structure of the grammar, transformational processes, the syntactic structures, and the lexicon are those parts of the taxon system which provide the means by which maps in one person's locale system are transferred to another's.** These taxon systems are analogues of the route systems of lower animals. They are based on the operations of categorization and the formation of linkages between frequently associated items to yield route statements. These routes, which take discourse from one substantive to another, would appear to be the basis for the tone groups described by Laver (1970, see footnote below). The rules which govern the generation of a set of routes from the underlying semantic map are analogous to the transformational component of a Chomsky-type grammar. As we shall see these rules can be much simpler than those specified by Chomsky or Fillmore, since the form of the surface sentence can be read directly from the directions traversed in the map.

Types of semantic maps. In developing the notion of a semantic map we shall build on Jackendoff's insights and follow his general methods. First we shall describe how semantic maps can be used to provide semantic deep structures for sentences about entities and events in physical space. Using one of these spatial semantic maps as an illustration,

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* Studies of language generation, which occurs in small segments called tone groups according to Boomer and Laver (1968) and Laver (1970), indicate that there are three separable processes involved (Goldman-Eisler 1968): (1) an idea or determining tendency; (2) the transformation of this idea into a sequential chain of symbols; (3) the selection of appropriate lexical items. One function for the verbal short-term memory system noted before (p. 388) could be the retention of a group of surface elements during the elaboration of the entire tone group. In contrast to the appearance of lexical elements in a short-term holding system, both at input and output, long-term memory for language is clearly concerned with the meaning of an utterance (e.g. Johnson-Laird 1970, Bransford and McCarrell (1974).

** From an evolutionary point of view, language could have developed as a means of transferring information about the spatial aspects of the environment: how to get somewhere, how to find food, etc.

we shall outline some of the syntactic transformation rules for transcribing all or part of this map into a surface 'route' sentence or phrase. Following Jackendoff's method, the next step will involve a discussion of how non-spatial maps similar to these spatial maps can be formed and how the same transformation rules operate to generate sentences about non-spatial entities and events. Instead of mapping physical space, these non-spatial maps depict surfaces on which the locations represent possession (or, as we shall call it, influence), identity, and circumstances. In this section we will also introduce the notion that maps or parts of maps can be 'named' and these names can be entered into locations on other maps.

A semantic map for spatial sentences. Sentences about the location of objects or the occurrence of events in physical space have an obvious and natural representation within a spatial map structure. Let us use Jackendoff's sentence (3) as an example

(3) The rock fell from the roof to the ground

The map of this event has three phases (see Fig. 36). The first (a) depicts the unstated presupposition that an entity (E) *the rock* was in a place (P) *the roof* up to some unknown past time t_1. The second (b) depicts the action at time t_1 in which an entity *the rock* moves from the place *the roof* to the place *the ground.* In the third phase (c), *the rock* is in the location *the ground* from time t_1 onwards. Thus there are two places, an entity which either stays in these places or moves from one to the other, and time markers which specify the time of the movement and the beginning and end of the period spent in a location. These time markers may refer to times attributed to the external world or they may be entirely internal to the map. The entire map is shown in Fig. 36(d).

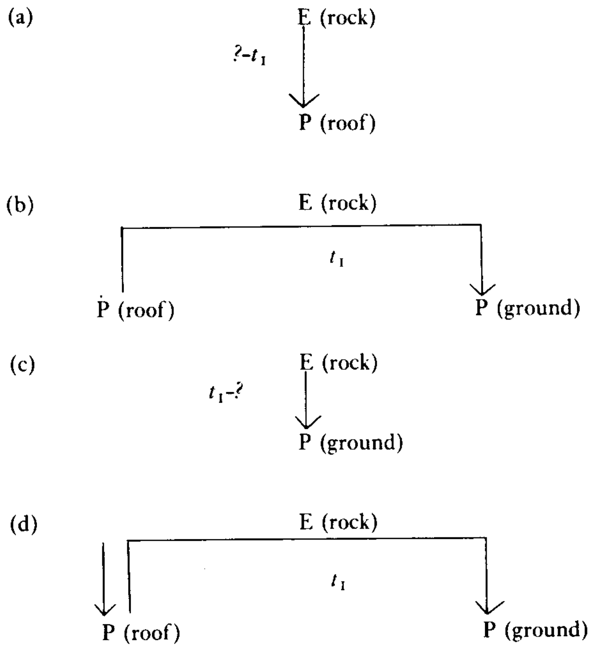

FIG. 36. Schematic for a semantic map of the physical spatial sentence 'The rock fell from the roof to the ground'.

Notice how the mapping system incorporates the three fundamental functions of Jackendoff's system, GO, BE, and STAY. BE is represented by the location of an entity in a place without a time marker. If there were no time marker on the first phase (a) of our three phase representation, this would depict the BE function: the rock is on the roof and, as far as we know, always was and always will be. The STAY function is represented by the third phase (c) where the time marker t, limits the duration of the state in the past direction but there is no time marker to limit it in the future direction. The second phase of our semantic map (b) represents Jackendoff's GO function, the movement of an entity from one location to another at some specific time.

A variety of sentences can be generated from our simple spatial semantic map by a set of transformation rules. We will assume that there are no obligatory points of entry into a semantic map nor are there obligatory directions of movement within the map. Thus although the map may have been originally constructed from a simple active sentence, it can be entered at any entity, event, place, or movement and read in part or in whole in any direction. The order of reading and the relationships between the successive items read determines the syntactic role of each item in the surface sentence. Maps containing nothing but spatial entities and events can only generate sentences in the active voice. We shall discuss the passive voice shortly. If the map of our example (Fig. 36) is entered at the entity *rock* and this is read first, it becomes the syntactic subject of an active sentence. If the movement is read next it becomes the verb, and the place of origin and the place of termination of the movement are made the objects of the prepositions *from* and *to* respectively. With this order of selection, we have generated our active sentence from which the map was created.

(15) The rock fell from the roof to the ground

If after reading the entity *the rock,* we had read first the place of origin *the roof* and then the movement and place of termination, our sentence would read:

(16) The rock was on the roof and (then) it fell to the ground (from there)

Similarly:

(17) The rock was on the ground where it had fallen from the roof

The natural expression of the relationship between an entity and its location is

$$(\text{Entity}) \text{ is } \left.\begin{matrix}\text{in}\\ \text{on}\end{matrix}\right\} \ (\text{location})$$

We might have entered the map at one of the places and read the entity next.

(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

The natural expression of the relationship between a location and its entity is

(Location) *have* (entity)

Similarly:

(20) The roof had something fall from it on to the ground and that was the rock
(21) From the roof, the rock fell to the ground

Finally the map can be entered at the movement itself

(22) The falling of the rock from the roof to the ground

In which case the movement is nominalized as a gerund or a noun (fall) and the entity becomes the object of the preposition *of* as a subjective genitive. It is clear, therefore, that any of the parts of a map can be the subject of the sentence derived from that map and that the parts of the map can be read in any order or direction. Finally the syntactic function of a representation in the map is determined partly by its role in the map and partly by the order in which the parts of the map are read.

The simple physical semantic map can embody all three of the deep structure functions (GO, STAY, BE) which were discussed in the previous section. Note that there are no rules necessary to derive inferences from the deep structure since these are built into the map when it is constructed in the first place. Our map of sentence (15) contains the unstated information that the rock was on the roof for some undetermined time, that it fell through the places between the roof and ground before falling to the ground, and that it remained on the ground for some undetermined time after the event.

Semantic maps for non-spatial sentences. Sentences about entities and events in physical space constitute only a small proportion of the language. Non-spatial sentences represent notions such as possession, causation, responsibility, identity, and category inclusion to name a few. Jackendoff introduced the higher order functions CAUSE and LET into his linguistic system to deal with causation and permission in spatial sentences. More importantly he suggested that non-spatial sentences conveying information about possession, identity, and circumstances had the same formal structure as spatial sentences. We will draw upon this insight and introduce the notion of non-spatial maps. Following Jackendoff, we will consider three types of non-spatial map: maps of influence or possession surfaces, maps of identity surfaces, and maps of circumstance surfaces. We do not have room here to go into great detail about each of these non-spatial maps so we will concentrate on the influence or possession type and only briefly comment on the other two.

One important concept not captured by a purely spatial mapping system is that which is common to the notions of causation, control, power, instrumentality, and possession. These notions represent a relationship between entities and/or events in which one is under the influence of the other. Some of these relationships are represented in semantic systems such as Fillmore's by the deep semantic cases *agent* and *instrument* (see above). We will postulate that all of these relations are represented on one surface which we shall call an influence surface. Influence relations on this surface are represented by entities in particular locations and changes in influence are portrayed as movements between places. Expanding on our previous notation, places in our influence map will be labelled P_{infl} while places in our physical spatial map will be labelled P_{phys}.

Entries in different maps (entities, places, and movements) which have the same name are considered to be connected so that the activation of one entry also activates all the other entities. For example, if *Harry* were the name of a place in influence space and an entity in physical space, activation of one would activate the other.

Before we discuss an example of a map portraying relations and events in an influence space, it will be useful to introduce the concept of *map nesting* or *embedding.* Maps or parts of maps can be labelled with names and these names can then be represented as entities or locations in other maps. The names of maps can appear not only in maps of the same type but of other types as well. Thus the name of an influence map could appear as an entity in a physical spatial map. This notion of map embedding will become clear in the next example which illustrates both the movement of an entity in an influence space and the embedding of this influence event in a second influence map. Our example is sentence (10) taken from Jackendoff (see above).

(23) Harry gave the book to the library

This means that the book moved into the possession of the library and that this event was caused by Harry. In the system we are proposing both the transfer of possession and the causation of that event would be represented in an influence map. The transfer of possession is represented as a movement from some unknown location into the location (the library). This event is given the name *transfer of possession* and entered into the location (Harry) in the influence map. The interpretation given to the relationship in an influence map between a location and its content depends on the nature of the content. When the content is a primitive entity drawn from a taxon store such as *the rock* or *the book* then the relationship is interpreted as a possession where the location possesses the content. When the content is the name of another map (i.e. an event), then the relationship is one of causation, the event is caused by the location. Entities possess other entities, entities or events cause events.[*] We will leave open for the moment what the interpretation of an entity in an influence location of an event might be.

Fig. 37 shows the influence map of sentence (23). Notice that the sentence does not specify whether the book belonged to Harry before the event described or whether the book actually physically moved to the library. These are left ambiguous. Consider the related sentences:

(24) Harry gave his own book to the library

which disambiguates the book's former possessor.

(25) Harry gave the book to the library- after it had been displayed on loan there for several years.

and

(26) Harry gave the book to the library but won't be sending it to them until next year.

The map representation of the event in sentence (23) is given in Fig. 37 (a)-(d). Notice the similarity to Fig. 36 except that the relationships and the event take place in an influence space. In order to represent the rest of the sentence, namely, that Harry caused the event portrayed in Fig. 37(d), this influence map is given a name and entered into the influence location called Harry (Fig. 37(e) and (f)). Fig. 37(g) shows the map of the whole sentence. As we stated earlier, entities *are possessed* by influence locations (e.g. Fig. 37(c)) but events *are caused* by influence locations (e.g. Fig. 37(f)).

More than one entity can be represented in an influence location. For example, if our influence map had represented sentence (24) instead of

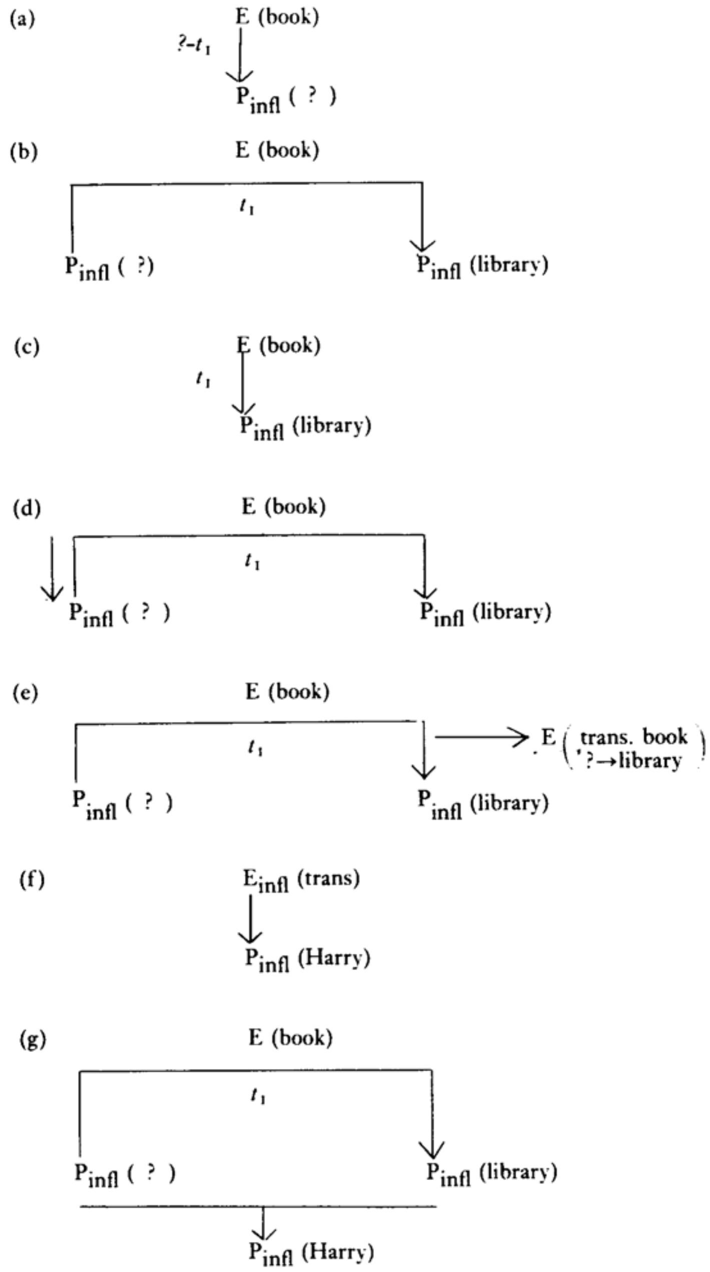

FIG. 37. Schematic for an influence map of the sentence 'Harry gave the book to the library'.

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[*] Strictly speaking, only events *cause* other events. When an event is entered into the influence space of an entity this is interpreted as an instrumental relationship. We will not go into this complication but assume for the present that agentive entities can cause events. It does not change the basic arguments set out here.

sentence (23) then the location *Harry* would have been substituted for the unknown location of the book in Fig. 37(a) and this location would have contained both the book until time t_1 , and the transfer at time t_1.

Let us look at some of the transformation rules for reading route sentences from influence maps. Here we will continually refer to the rules developed for physical spatial maps to show the similarities. As with physical spatial maps, influence maps can be entered at any point and read in any direction. Let us start with the event in influence space and compare it with the event in physical space mapped in Fig. 36. Consider the following pairs of sentences:

(15) The rock fell from the roof to the ground
(27) The book went from (the possession of) someone to (the possession of) the library
(16) The rock was on the roof and (then) it fell to the ground
(28) The book was in the possession of someone (or was someone's) and (then) it went to the library
(17) The rock was on the ground where it had fallen from the roof
(29) The book was in the possession of the library whence it had come from some unknown person

The natural syntactic expression of the relationship between an entity and its location in influence space is the same as in physical space. Conversely the natural expression of the relationship between an influence location and its content is

(Location) *have* (entity)

(18) The roof had a rock (on it) and (then) the rock fell to the ground
(30) Someone had a book and (then) the book went to the library
(31) Harry had the book given to the library
(32) Harry had someone give his book to the library

As examples (31) and (32) show the *have* transformation holds irrespective of whether the content of the influence location is an entity possessed or an event caused.

Two important syntactic features which must be introduced into the transformation rules for influence maps do not exist for physical spatial maps. These are the active/passive voice distinction and the genitive of possession. Both of these are necessary to transcribe readings in which the contents of an influence location are read before the name of the location itself. Thus, in our example, reading the locations first in Fig. 37(c) and (g) gives, respectively

(33) The library's book
(34) Harry had the book given to the library

while reading the contents of those locations first gives

(35) The book of the library
(36) The book was given to the library by Harry

If the influence location is read after its contents, it becomes the object of the preposition *by* in a passive sentence or the object of the preposition *of*. Notice that instead of (34) one could have read (37) or (38):

(37) Harry's giving (of) the book to the library
(38) Harry's gift of the book to the library

In addition to a mode expressing possessional relations and changes in possessional relations, Jackendoff postulated *identificational and circumstantial* modes whose syntax was analogous to the physical spatial mode in much the same way that that of the possessional mode was. We have not attempted to work this out in detail or to construct maps of these spaces* but see no insurmountable problem in doing so. Note, for example, the similarities between the identificational sentence (39), the circumstantial sentence (40), and the physical sentence (15) above.

(39) The rock went from smooth to pitted
(40) The librarian went from laughing to crying

To conclude this section, let us briefly mention what seems to us the main weakness of the semantic map idea. Maps of physical space which use a Euclidean metric allow inferences to be drawn about the relationships amongst entities in those maps which go beyond the usual laws of logic. This was, of course, what Kant meant when he called space a *synthetic a priori.* Thus we can say that the rock in its fall from the roof to the ground must pass through the intervening places. Our map did not specify whether these places were occupied or not. If it had then we could conclude that the rock must bump into, or pass through, whatever occupied those places. Some of the work of Bransford and Franks and their colleagues seems to be based on inferences of this type. Now it is not immediately obvious that the same kinds of inferences can be drawn from our non-physical semantic maps. Part of our difficulty here is that we do not know what the axes of the non-physical semantic maps are or even whether the maps can be considered Euclidean. These objections notwithstanding we think that the work of Jackendoff opens up exciting possibilities for investigating the use of maps of physical and non-physical spaces as the basis for deep semantic structures.

14.3.3. CONCLUSION

The above sections outline a possible way in which the cognitive-mapping system could function as a deep structure for language. The representations in this deep structure have all the properties we have attributed to maps.

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* Whether these maps are irreducible primitives or will on further examination be found to be reducible to various combinations of physical spatial and influence maps remains to be seen.

Items are entered into a semantic map not on the basis of the order in which they are received or their position in a left-to-right linear string but in accordance with their semantic relationships to other items in the map. Maps do not incorporate information about the way that they were constructed or the 'route' surface sentence from which they were constructed. Items in the map and their relationship can be read in any order and a large number of different sentences can be generated from the same map. Once a semantic map exists, additional information can be added or changes in the existing information can be made. The resulting large semantic structures embody the ideas expressed in paragraphs or whole stories. We locate these semantic maps in the left human hippocampus. Taken together with the representations of physical occurrences in the right hippocampus they form the basis for what is generally referred to as long-term, context-specific memory for episodes and narratives. In the next chapter, this assertion will be 'tested' against the known facts of the amnesic syndrome.

15

The amnesic syndrome

THE correlation between hippocampal damage in humans and some form of generalized memory defect has been established for over two decades (cf. Scoville and Milner 1957). Considerable research in the intervening years has failed to characterized this defect with any precision and has consequently failed to determine the exact nature of hippocampal function in humans. We have discussed, in the previous chapter, a model of human hippocampal function derived in part from work with infra-humans and in part from a consideration of the special features of human information storage and memory processing. In this chapter we shall apply this model to the data generated from the study of amnesic patients in the hope of coming to some understanding both of the syndrome of organic amnesia and of the role played in it by hippocampal damage.

The organic amnesia resulting from hippocampal damage, and a variety of other causes to be discussed shortly, is not the only form of memory loss known in the clinic, nor should it be in view of our comments on the non-unitary nature of memory processes. In accordance with the proposed properties of the taxon systems and the nature of its memory stores, damage in a variety of extra-hippocampal areas yields disorders which can be viewed as *limited amnesias* (cf. Whitty and Lishman 1966); examples include *agnosia, apraxia,* certain forms of *aphasia, prosopagnosia,* which is a specific deficit in face memory, and perhaps the selective deficit in verbal short-term memory associated with conduction aphasia (Warrington and Shallice 1969). Patten (1972b) has analysed some of these modality specific amnesias in detail.

Amnesia consequent upon bilateral damage to the locale system, in contrast, appears to be neither modality nor material specific. Unilateral damage, as we shall see, does lead to some specificity: left-hemisphere damage affecting verbal memory, right-hemisphere damage affecting nonverbal memory. Still, there is no modality specificity in these defects; what is important is the form of the to-be-stored information, not its channel of input. Looking ahead briefly, there are both *retrograde* and *anterograde* components to the organic amnesia syndrome. That is, there is both a loss of previously stored information and a loss in the ability to store new information. The fact of central importance in the syndrome, which has only recently become clear, is its selectivity. All old memories are not lost; some new information can be stored. The task of any theory of hippocampal function in humans is to provide the basis for understanding the selective memory loss,* which contemporary research is slowly defining. This is parallel to the problem presented by the study of hippocampal damage in infra-humans, where the selectivity of the behavioural effects also constitutes the central problem for theory.

Before turning to an analysis of the syndrome elicited by hippocampal dysfunction there are several methodological problems which must be considered. First and foremost we must consider the evidence relating the organic amnesia syndrome to hippocampal damage. In some cases the damage is known and there is no interpretive problem; in others the damage is unknown and subjects are classified solely on the basis of behavioural observations. Determining the extent to which hippocampal damage is responsible for the observed behaviour is a necessity. Following this we shall briefly describe some of the testing procedures used in the clinic, concentrating on those which assess memory function. Our purpose here is simply to provide the reader with some background concerning the terminology to be used, as well as an idea of the kinds of information which can be derived from the use of particular experimental procedures. Finally, we shall, on the basis of our theory, provide a set of 'predictions' concerning the effects of hippocampal damage. These 'post hoc' statements do not represent attempts at verification of the theory; such verification must come from future research aimed at a finer analysis of the syndrome.

Following these methodological and predictive sections, we shall review the clinical data, touching first upon data pertinent to the functions of the taxon systems which should provide an indication of the minimal learning capabilities of amnesic patients. Then, we turn to data concerned purely with the spatial mapping function of the (right) hippocampus and with its role in exploration and curiosity. Finally, we consider the central problem of the defect in memory storage, which has primarily been studied within the context of verbal learning paradigms. Here, we hope to show that there is a specific deficit in the processing and storage of deep-structure maps, pertinent to both imaginal and language content. This should manifest itself most obviously in the loss of context-specific memory.

15.1. The role of hippocampal damage in organic amnesia

The absence of precision in prior characterizations of human hippocampal function arises not only from the use of inadequate psychological models for memory; frequently the focus of interest was the clinical syndrome of

organic amnesia, rather than a particular brain structure, and it was impossible to assign functions to a given structure. The syndrome of organic amnesia, which arises after hippocampal damage, has also been associated with several other clinical states, not all of which can be easily characterized as to precise locus of brain damage. To some extent this has confused matters, at least from the perspective of an interest in hippocampal function. It is important to discuss, then, the nature of the patient population under study, both in terms of possible sites of damage and in terms of specific clinical manifestations that obviously have little to do with hippocampal damage.

15.1.1. VARIETIES OF ORGANIC AMNESIA

Our knowledge of the organic amnesia syndrome comes from the study of a variety of patient populations: (1) those with Korsakoff's psychosis, typically as a sequella to prolonged alcoholism or encephalitis (Korsakoff 1889); (2) those with mesial temporal lobe damage, resulting either from an accident, from vascular occlusion or a tumour, from temporal lobe epilepsy, or after an operation to alleviate focal epileptic seizures. These varied populations present somewhat divergent clinical patterns and it thus becomes important to try to establish the extent of hippocampal dysfunction in each of them. The perfect subject for study would have damage limited strictly to the hippocampus in an otherwise normal brain; few, if any, such cases exist.

There are two questions, in practice, which must be answered: (1) to what extent is hippocampal damage responsible for the defects seen in diseases known to involve diffuse damage, such as Korsakoff's psychosis? (2) to what extent is the amnesia seen after temporal lobe surgery for epilepsy a function of the prior existence of an epileptic focus?

15.1.1(a). The role of hippocampal damage in organic amnesia. The operation which left H.M., Scoville's (1954, 1968) classic patient with a profound memory defect involved the removal of much of the mesial temporal lobe, including most of the hippocampus, uncus, and amygdala, but spared the lateral temporal neocortex. Subsequent to the discovery of H.M.'s memory defect, Scoville and Milner (1957) examined some of the patients who had previously undergone similar, though often less drastic, removals. Generalized memory defects were seen in all those patients whose removals included portions of the hippocampus. More pertinent, perhaps, is the fact that no generalized memory defect was apparent when damage was limited to the uncus and amygdala. Sawa *et al.* (1954) and Terzian and Dalle Ore (1955) have also noted that resections limited to the amygdala do not impair general memory function. Thus, it seemed likely, though not proven, that hippocampal damage was crucial for the memory defect. Milner (1968) correlated right temporal lobectomy with

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* Marslen-Wilson and Teuber (1975), in analysing the defect in an amnesic patient, state that his behaviour 'underscores the perplexing quality of severe amnesia: it is not all or none . . . and it does not readily lend itself to those interpretations of amnesia which speak of generalized failure either of storage or retrieval' (p. 362).

memory defect and found that there was a strong correlation between the extent of hippocampal removal and the gravity of the memory loss. Finally, in what appears to be a definitive answer to this question, Corsi (1972) tested an extensive series of unilateral temporal lobectomy patients with varying degrees of hippocampal damage and found that the amnesic condition was directly related to the loss of hippocampal tissue. As we shall see later these losses were material specific: verbal defects were seen after left hippocampal damage and non-verbal defects after right hippocampal damage. Thus, it now seems established that the generalized amnesia resulting from temporal lobectomy is a direct function of hippocampal damage.*

The question is considerably more complex in the case of patients with Korsakoff's psychosis. This syndrome is defined operationally, so that any number of different pathological states can be associated with it. Most commonly it is seen in the following cases: (1) as the chronic phase of the Wernicke-Korsakoff disease, consequent upon prolonged alcoholism and dietary insufficiency; (2) as the aftermath to an attack of herpes simplex encephalitis; (3) as the aftermath of a stroke, or occlusion of the posterior cerebral arteries (Glees and Griffith (1952) and Victor et al. (1961) report such cases); (4) with certain brain tumours, as in the cases reported by Sprofkin and Sciarra (1952); (5) in association with Alzheimer's disease.** Associated with the last four of these precipitating conditions is clear-cut involvement of hippocampal dysfunction; considerable damage to the hippocampus has been reported in most cases that have been described (cf. Whitty and Lishman 1967). However, in the Wernicke-Korsakoff syndrome, based on chronic alcoholism, pathology has been traced most prominently to the mammillary bodies and the dorsomedial thalamus (Brierley 1966, Victor 1964, Victor, Adams, and Collins 1971). To be sure, these are target areas for outputs from the hippocampus and subiculum, yet one would feel more comfortable if changes were seen in the hippocampus itself. It remains possible that more detailed histochemical investigation at autopsy would show up subtle changes in the hippocampus; only future research can provide an answer to this possibility. We are forced to conclude that the amnesia seen in Korsakoff's syndrome is associated with damage either to the hippocampus itself or to its output systems.

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* While the involvement of hippocampal damage in the amnesia produced by temporal lobectomy is well established, it is not always the case that damage to the hippocampus, or more precisely the fornix, always results in amnesia. There are several reports in the literature (e.g. Woolsey and Nelson 1975) of cases involving fornix section without memory loss. We, along with others, have no good explanation for these anomalous reports; it should be kept in mind however, that the subiculum and not the hippocampus is the origin of the fornix.

** This list is by no means exhaustive. Other precipitating factors include: (1) the aftermath of electroconvulsive shock therapy (cf. Squire 1975); (2) operation for anterior artery aneurysm (cf. Lindqvist and Norlen 1966); and (3) concussion (cf. Yarnell and Lynch 1973). We will not be discussing data from such cases in this review, but they do not differ either in the possibilities they offer for study or the problems attendant with such use from the examples offered in the text.

However, we must bear in mind that Korsakoff patients, who comprise the majority of amnesic patients studied in the clinic, typically have extra-hippocampal damage of a diffuse nature. Though most investigators are careful to exclude patients with general dementia from their studies, the presence of diffuse damage could mean that the presenting syndrome for Korsakoff patients would be different than that seen in other patients, and, in fact, several studies have demonstrated deficits in sensory thresholds in these patients (e.g. Oscar-Berman, Goodglass, and Cherlow 1973, Jones *et al.* 1975) which are not apparent in patients with focal hippocampal damage. It is also important to note that there might be different forms of Korsakoff's disease (cf. Lhermitte and Signoret 1972), varying in the extent of damage to different structures or within the two hemispheres. Finally, Korsakoff's psychosis is not a static condition involving a fixed amount of damage. The same patients could vary from study to study, and different patients could vary within a study. Some assessment of the ongoing intellectual deterioration of such patients is important in order to facilitate comparisons between studies. This problem has plagued the study of Korsakoff's psychosis, possibly lying at the root of seemingly contradictory results reported by two groups of investigators as we shall see later (p. 427).

15.1.1(b). The contribution of the epileptic focus. The Montreal series of temporal lobectomy patients all underwent operations for the relief of focal epilepsy. The question of the contribution of epilepsy itself to the amnesic syndrome thus becomes an important one. In the epileptic patient grand mal, and often petit mal, seizures are accompanied by transient amnesias; direct stimulation of the hippocampus can have the same effect (e.g. Bickford *et al.* 1958). In these cases amnesia results from a `functional lesion' induced by the seizure or stimulation. Consistent with this view is the fact that seizures are associated with automatisms of behaviour which we would relate to the residual functioning of the taxon systems and which bear a striking resemblance to the stereotypies seen in animals with hippocampal lesions.

On the other hand, when areas afferent to the hippocampus are stimulated, 'memories' can often be elicited, as in Penfield's studies (e.g. 1958). This activation can be compared to the syndrome seen in the acute phase of the Wernicke-Korsakoff disease, described by Torda (1969) as a 'state of continuous hallucinosis' during which the patients continuously 'heard and/or saw their thoughts' (p. 279). A similar state has been described as the immediate, though transient, sequella to anterior cingulectomy, and was attributed to a discharging lesion producing downstream effects in the hippocampus (Whitty and Lewin 1960). All these effects can be ascribed to an activation of the hippocampus, during which the normal mechanisms for activating cognitive maps are bypassed.

When the epileptic tissue is removed, generalized amnesia can result if hippocampal tissue is resected. This defect is not apparent prior to operation, except during overt seizure. The fact that amnesia is present post-operatively only and that it is critically linked to hippocampal damage makes it rather unlikely that amnesia depends upon the pre-operative epileptic condition, as was suggested by Isaacson (1972). Perhaps most convincing in this regard is the existence of amnesic patients with known hippocampal damage but without prior history of seizures (e.g. Walker 1957, Rose and Symonds 1960, Victor et al. 1961, Dejong 1973). These cases indicate that amnesia is associated with the disruption of normal hippocampal function and is not dependent upon the establishment of an epileptic focus.

15.1.1(c). Conclusions. Most forms of organic amnesia seem to involve hippocampal dysfunction. Differences between the various patient populations reside in the locus and extent of extra-hippocampal damage. Korsakoff patients suffer from the greatest amount of such damage and the syndrome must be viewed in this light. It would perhaps be simplest to ignore the data generated from the study of these patients, but this is both impractical and unnecessary, the former because a considerable number of studies have utilized Korsakoff patients, and the latter because there are certain constant features in all varieties of organic amnesia and these can be assumed to define the functions of the structure central to the syndrome, the hippocampus.

15.2. Some clinical tests

Most of the testing methods used in the clinic to assess the amnesic syndrome are concerned with the problem of learning and retention of that learning after a variable interval. A major difference between the various retention tests resides in the amount and form of information provided to the subject during retention.

15.2.1. FREE RECALL

A typical free-recall paradigm might go as follows: A subject is shown a set of words in serial order. Following the presentation of this list and some delay interval, retention is assessed by asking the subject to recall as much of the material as possible. With such a test, the probability of correct responses being emitted by chance is usually quite low.

Free-recall tasks can be used to assess the short-term or long-term retention of any form of material. When the delay between item presentation is short, on the order of 5-20s, one can say that short-term memory is being assessed. Delays beyond that assess long-term memory performance, but only if the subject is prevented from rehearsing the test material. The means of preventing rehearsal vary from study to study; we shall see later that inadequate control over rehearsal could lead to misinterpretation concerning the status of a subject's short-term memory capability.

The importance of optional input strategies can be assessed in free-recall tests by giving the subject explicit instructions, for instance, to use natural-language mediation (see pp. 387-8n), or to form an image between several of the items, and so on. Similarly, the role of organizational factors, such as category inclusion, can be assessed by using test materials which either explicitly convey that organization (as in lists with category-related words blocked together) or contain the possibility of being organized into chunks by the subject. In the latter case the role of instructions could be important in determining the usefulness of having material presented in this chunkable form.

When several free-recall lists are presented in succession one can measure the extent and form of interaction between the lists–this gives some idea of the way in which memories interfere with one another. By varying the form of similarity between the items in successive lists the nature of coding can be assessed. For instance, if acoustically similar words are confused one can conclude that the storage system being tapped depends on an acoustic code and that systems based on other codes are not functional at that time or in that subject. By shifting the form of interaction between lists one can measure the specificity of interference effects; we shall see that such tests have proved quite useful in assessing the nature of the amnesic defect.

The most important feature of free-recall tasks is that the subject is not given any cues at retrieval.* Thus, these tasks measure the way in which the subject spontaneously encodes information and the availability of that information under what might be considered normal retention conditions. In fact, it will be seen that considerable information which has been registered, and stored, during free-recall presentation is not retrieved by most subjects in the absence of some form of retrieval cue at the time of retention. This indicates that the information has been stored in ways that do not allow for or actively interfere with its subsequent retrieval. Given that, the role of instructions during presentation of the test material and of specific cues during the retention test can be assessed in order to determine the nature of coding and the ways in which access to stored material can be improved. Such 'cued' recall tests have proved to be quite important in the assessment of the amnesic defect, as we shall see shortly.

15.2.2. PAIRED ASSOCIATES

A standard psychological test involves the presentation of pairs of stimuli, the first term being the `stimulus' and the second the required 'response'.

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* With the exception of the location in which the task is performed.

During retention the subject is given the 'stimulus' and must provide the 'response'. This task, termed paired-associate learning, provides the possibility of assessing the effects of the relationship between 'stimulus' and 'response' terms, between 'stimulus' and/or 'response' terms in successive lists, and so on. This paradigm has also been used extensively in the study of imagery processes, with subjects being instructed to form images between the 'stimulus' and 'response' items.

Unlike free recall, in the absence of retrieval cues, the paired-associate task incorporates retrieval information, the 'stimulus' items. Though it is a less demanding task than free recall, it allows for the controlled study of the efficacy of different forms of retrieval cues.

15.2.3. RECOGNITION

Recognition tasks involve a judgment on the part of the subject as to whether or not a test item has been experienced before. Usually, test items are familiar objects or words, and recognition consists in judging whether these items have been seen in a particular context, the test situation. Thus, this paradigm can be used as a sensitive measure of context-dependent memory. Less frequently, test items are unique configurations, e.g. a complex picture or a sentence, and recognition can be based solely on whether or not that configuration had ever been experienced, that is, some manner of familiarity judgment. These two situations, as we shall see, produce strikingly different results in amnesic subjects.

There are two basic forms of recognition test: yes-no recognition and forced-choice recognition. The first form involves the presentation of individual items during retention, with the subject responding either yes (the item is recognized as part of the test material) or no. The second form involves the presentation of many items simultaneously at retention, half of these being drawn from the test material, half not. The subject must choose those which were in the presentation list. In both forms the probability of making a correct response by chance is 50 per cent and some correction for guessing is important. By varying the nature of the 'filler' (non-presented) items one can manipulate the 'false' recognition of similar items. This provides a means of determining the nature of coding used during learning. For instance, the consistent false recognition of acoustically similar words tells us something about the form in which items have been stored.

15.2.4. OTHER TESTS

Aside from these standard psychological paradigms which can be used with either verbal or non-verbal materials, a variety of other tests have been utilized in the study of amnesics. Tests for spatial abilities, for instance, include mazes similar to those used in rats in their complexity, and these can be performed with either visual or tactual information. Tests for motor learning, such as rotary pursuit and mirror-drawing, have been used as well. We shall describe the features and procedures of these tests when analysing the performance of amnesics.

15.3. Predicted effects of hippocampal damage in humans

The present model provides three sets of overlapping predictions concerning the amnesic syndrome. The first is based upon the functions and properties of the hippocampal locale system, and includes those things which the amnesic patient ought not to be able to do under any condition. The second and third sets are based on the functions and properties of the extra-hippocampal taxon systems, the second including abnormalities in behaviour resulting from a forced dependence upon these systems, and the third including those forms of behaviour which the taxon systems can mediate and which should appear relatively intact in the amnesic subject.

15.3.1. PREDICTIONS FROM THE LOCALE SYSTEM

In view of the properties of the locale system, the amnesic patient should be characterized by the following.

(1) A lack of initiative, curiosity, and incidental learning.
(2) A deficit in maze learning and other situations requiring the use of a 'spatial' map of the environment.
(3) A deficit in those learning situations which are dependent upon retention of contextual information.
(4) An inability to utilize techniques based on spatial imagery.
(5) The loss of language functions dependent upon the deep-structure properties of the locale system, e.g. the recall of narrative.
(6) Selective deficits when unilateral hippocampal damage is involved: verbal defects with left hippocampal damage; non-verbal defects with right hippocampal damage.

15.3.2. PREDICTIONS FROM THE TAXON SYSTEMS

The forced use of the taxon systems should lead to two basic abnormalities in behaviour: (1) a tendency to repeatedly attend (and respond) to the same stimuli; (2) considerable proactive and retroactive interference in the learning of similar materials, which follows from the form of information storage in taxon systems. However, the taxon systems can mediate certain forms of memory, as we have seen both in discussing the animal data and in analysing the properties of human memory. Thus, the amnesic patient should (1) learn simple discrimination tasks, (2) learn motor tasks,

(3) be able to utilize such taxon strategies as rehearsal, category inclusion (chunking), natural-language mediation, and so on, and (4) show normal identification and abstraction, within the limits defined by the large interference effects inherent in the use of taxon storage, limits which could be expanded through the provision of cues which act against these interfering effects.

15.4. Taxon tasks

The first formal indication that hippocampal dysfunction did *not* lead to a complete loss of memory storage came from the study of simple motor tasks, examples of behaviours which we would allocate to the taxon systems. Adequate learning has been demonstrated in rotary pursuit, bimanual tracking, mirror drawing, tapping, and other tasks in both Korsakoff and temporal lobectomy patients (Talland 1965, Corkin 1968, Milner 1968, Cermak *et al.* 1973). These motor tasks do not require the participation of the locale system; in addition, they are not particularly subject to interference effects, and this enables amnesics to use taxon learning mechanisms with some efficiency.

Several aspects of the motor learning of H.M. (Scoville's patient, mentioned above) are worth noting. Though capable of showing long-term improvements, H.M. is still deficient at these tasks when compared with control subjects. In both rotary pursuit and bimanual tracking H.M. seems capable of improving only up to a certain point, at which his performance asymptotes; controls continue to improve beyond this point. The early improvement, in both H.M. and controls, is due to a steady reduction in the amount of time required to get back on the target, once off it (in these tasks the subject must maintain contact with a changing target). Later, the controls begin to spend less and less time off the track; this is not evident in H.M.'s error pattern (Corkin 1968). Corkin suggested that H.M.

> 'might have been less efficient in predicting the irregular shifts in the tracks encumbent in the bimanual task, although the extent to which this strategy was employed by either normal subjects or H.M. was not determined' (p. 264).

Much the same thing was seen in the rotary pursuit task. Thus, improvements dependent upon reducing the duration of errors can be seen in H.M. However, no improvement in staying on the target appears. This inability to benefit from some memory of the sequential changes occurring in a moving target is in contrast with H.M.'s performance on a task involving the learning of an internally generated sequence; Corkin showed that H.M.'s improvement in a tapping task parallels that of control subjects. In a sense, this pattern resembles the difference between maze-learning deficits and sequential-response facilitation seen in hippocampal rats (e.g. Jackson and Strong 1969).

Another type of learning that appears normal in amnesics is simple discrimination. Korsakoff patients can form what seems equivalent to a passive avoidance response (Talland 1965).* Sidman, Stoddard, and Mohr (1968) tested H.M. on several types of discrimination. Although unable to verbalize the nature of the task he was performing, H.M. showed normal discrimination between a circle and ellipses of varying degrees of similarity to the circle. Talland did not, unfortunately, test his Korsakoff patients on similar tasks. Finally, Gaffan (1972) has shown, in a single amnesic patient, that the learning of a simultaneous colour discrimination is normal, though the patient was quite certain during the retention tests that the task had never been seen before.

It is worth repeating that Korsakoff patients have difficulties in certain sensory tasks which reveal no impairment in temporal lobectomy patients (e.g. Oscar-Berman *et al.* 1973, Jones *et al.* 1975). Such functions clearly have nothing to do with the hippocampus, and the defects seen in their performance by Korsakoff patients are a reminder of the diffuse damage associated with that disease.

In sum, amnesics can learn certain tasks in a normal fashion. These are analogous to those simple tasks which hippocampal animals can learn and are a function of the operation of the taxon systems. As with the animal data, there are indications that, though able to learn certain things, amnesics often learn in a different way. Some further examples of normal learning in amnesic patients will be presented in following sections in the context of our discussion of the verbal memory defect.

15.5. Exploration and spatial mapping

15.5.1. INITIATIVE, CURIOSITY, AND RELATED PHENOMENA

It was early recognized that Korsakoff patients lack initiative, are apathetic about their surroundings, and are generally passive. Meggendorfer (cited by Zangwill 1966) noted that a lack of initiative was an almost invariant aspect of Korsakoff's psychosis, Burger-Prinz and Kaila (cited by Lidz 1942) noted that Korsakoff patients 'let the world come to them and do not go to meet it', and Talland (1965) stated that an 'abnormal measure of inertness and the absence of spontaneity have been regularly associated with the amnesic anomalies' (p. 115).

Talland has documented the essentially respondent nature of the Korsakoff patient; his attempts to engender social intercourse among groups of four or five patients were futile. All questions were answered without elaboration, and elicited at most a single additional comment from another

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* Talland cites a comment by Claparede concerning one of his Korsakoff patients. While shaking hands with a female patient he jabbed her with a pin hidden in his hand. A few minutes later, though unable to recall this incident, the patient refused to shake hands with him.

patient before silence was resumed. On the other hand, given a boring task such as drawing circles, these patients can continue at length without interruption.

Much the same thing has been reported following hippocampal damage. H.M. has a placid temperament and has been described as hypoactive (Milner 1966); he also can continue for long stretches of time at boring, repetitive tasks. The patient discussed by Sweet, Talland, and Ervin (1959), who underwent bilateral section of the fornix, was described as apathetic and lacking in spontaneity. Finally, the patient described by Victor et al. (1961), who sustained bilateral hippocampal damage following several strokes, 'tended to remain quiet for long periods of time and rarely would initiate a conversation' (p. 30).

Formal tests of exploratory capacity indicate a profound lack of interest in investigating uncertain or novel situations in Korsakoff patients. Talland has shown that such patients do not resume unsolved problems if disrupted. Another manifestation of this uninterest is a lack of incidental learning. A jigsaw puzzle composed of parts of a newspaper was given to the Korsakoff patients; solution required that they check to see that the assembled words made sense. Contrary to the control subjects, the Korsakoff patients remembered very few of these words.

Overall, the human amnesic patient shows little spontaneous behaviour, little interest in exploring the environment, and few signs of responding to items which are ‘out of place’. This picture compares quite well with what is seen in the rat, cat, and monkey after hippocampal damage.

15.5.2. SPATIAL MAPPING

The absence of exploration is assumed to result from the inability of the amnesic patient to develop cognitive maps. Before turning to more specific data on this point it is worth repeating the distinction we drew in the previous chapter; the locale system is involved in non-egocentric, objective spatial representation, not in egocentric spatial representation. The latter function, involving among other things the recognition of objects in different perspectives and orientations, appears intact in amnesic patients (Talland 1965), though it is disturbed by parietal lesions, as we have seen. The correlation between generalized memory defects and deficits in geographic orientation (Benton 1969) is an indication of the form of spatial representation we assign to the hippocampus.

Anecdotally, there are indications of profound spatial disorientation in amnesics. It takes Korsakoff patients considerable time to learn their way about a hospital, while some never manage this. H.M. shows even more profound disorientation; he appears virtually lost when more than two blocks from his home, often going instead to his old house from which he had moved some years before. Another patient (D.C.) described by Milner (1966) could not find his way about the hospital in which he had been resident for 11 years. Similar descriptions of disorientation for place are included in most reports of amnesic patients (e.g. Dejong 1973) and will not be enumerated here. These reports provide ample reason to suppose that deficits in maze learning and similar tasks would be seen in amnesics.

Talland's patients were profoundly impaired in learning a relatively simple tactual maze consisting of six choice points in a regular pattern. Further, these patients showed no savings when the maze was rotated. He noted that ‘those who discovered the pattern of turns formed but a precarious visual-kinesthetic map’ (p. 229). Cermak et al. (1973) have also reported a profound defect in maze learning in Korsakoff patients. H.M. and other bilateral temporal lobectomy patients have been tested on a variety of maze problems. Again, a profound impairment was seen in several modalities, indicating that the underlying disability is a general one. On complex mazes the deficit is nearly total; H.M. failed to solve a 28-choice visual stylus maze within 215 trials. In fact, no improvement was seen, in terms of errors, during this training. The same results were reported by Corkin (1965) for the learning of a tactual maze with 10 choice points. H.M. showed no improvement whatever over 80 trials, though he did show a reduction in the time required to move through the maze, replicating a result noted in the 28-choice maze. This decrease in error time, without an improvement in choice performance, would seem to reflect the same factors as H.M.'s performance in bimanual tracking (see p. 420): the possibility for a reduction in the time spent doing the incorrect thing, but no decrease in the number of times the incorrect thing is done.

H.M. has been tested on shortened forms of these two mazes (Milner, Corkin, and Teuber 1968). Initially, he was given 125 trials on an eight-choice visual maze; he showed little sign of learning during these trials. When the maze was further reduced to six choice points H.M. managed to reach the criterion of three errorless runs in succession after 155 trials. Once having learned the maze he showed considerable retention, even after an interval of six days. The pattern of errors made by H.M. on this maze are of considerable interest. The authors noted that there was a 'high build-up of anticipatory errors ... rather than a random distribution of wrong choices' (p. 226). H.M. did not make repetitive errors within a single trial, which argues against any simple response perseveration interpretation of his defect, as the authors pointed out. This pattern of errors bears a striking resemblance to that seen in rats (cf. our discussion of maze errors in rats, pp. 286-90).

H.M. was also tested on a shortened tactual maze containing five choice points. Though he did not reach criterion within 300 trials there was a steady reduction in errors in the course of this training. The results on both the shortened visual and tactual mazes suggest that H.M. can learn simple mazes, but only when there are sufficiently few choice points that the learning can be mediated by remembering the entire series of choices.

Talland tested his amnesics on a modified form of the Rey-Davis test, which evaluates both incidental and deliberate spatial learning. This task consists in finding the loose peg among a set of pegs on a board. Successive sets are given to the subject, who must then find the loose peg in each set. When coloured pegs are used the subject can find the loose peg by attending to the colours of the pegs (a guidance hypothesis); the loose peg is always coloured differently. When coloured pegs are not used, however, the problem must be solved by noting *where* the loose peg is located. About half of the 16 Korsakoff patients were able to master this task when given colour cues; all but one failed without these cues. When they had been trained with the coloured pegs and then were presented with the same series of peg placements, but using uncoloured pegs, they performed poorly. This transfer test shows that the patients solved the coloured-peg problem through the use of the colours and had not learned about the position of the loose peg. This pattern of results is similar to that seen in hippocampal rats in mazes with and without cues at the choice points (e.g. Leaton 1969, Winocur and Breckenridge 1973).

The maze-learning data provide information relevant to the question of hemispheric specialization in the hippocampal system. For both the visual (Milner 1965) and the tactual (Corkin 1965) mazes, selective deficits were seen after right, but not left, hippocampal damage. These data are consistent with the view that the right hippocampus in humans is concerned with spatial mapping. Milner (1965) pointed out that many of her left-hippocampal patients were markedly dysphasic and certainly could not have learned the visual maze through verbal mediation; they were obviously able to learn it solely through spatial, non-verbal mediation. On the other hand, the right-hippocampal patients resorted to numerous verbal devices, but were nevertheless severely impaired. Further evidence on this point comes from studies by Corsi (1972) and Milner (1971). Subjects were required to tap the members of a spatial array of blocks in a sequence demonstrated by the experimenter immediately beforehand. This non-verbal, spatially cued task is analogous to a digit-sequence test. Corsi embedded a recurrent sequence within the series, making the task logically equivalent to the verbal recurrent-series task described by Hebb (1961). In this situation subjects typically show a learning curve for the recurrent sequence. The right-temporal group with radical hippocampal damage was deficient on this task, showing virtually no learning of the recurrent sequence. H.M. was similarly impaired. Left-temporal patients, with or without hippocampal damage, performed quite well. This picture was reversed when a verbal test was used; right temporals performed well and left temporals were impaired. Korsakoff patients performed poorly on a formally similar task (Meissner 1966). The subjects had to press buttons in a prearranged sequence; amnesics performed poorly at any sequences

beyond one or two buttons. Finally, Samuels et al. (1971) tested Korsakoff patients on the retention of visual 'scenes' involving the presence of a set of figures in a pictorial background. Performance on this task was profoundly impaired, indicating that the amnesics had trouble remembering items in a context.

In sum, Korsakoff patients, temporal lobectomy patients with right hemisphere damage, and those with bilateral damage are deficient in learning even the simplest of mazes. The pattern of responding in the maze is strikingly similar to that seen in the hippocampal rat. These data are consistent with the assumption that the right hippocampus in humans subserves spatial mapping.*

15.6. The memory defect

15.6.1. INTRODUCTION

Until recently it was felt that amnesics could retain, in the long term, nothing beyond the simplest of motor or discrimination tasks. Though some early workers (e.g. Lidz 1942) felt that the problem was more one of faulty retrieval than faulty storage, the traditional view of the amnesia consequent upon mesial temporal lobe excision held that there was a defect in the processes involved in the 'consolidation' of long-term memory (e.g. Milner 1966). In the past 10 years data have been accumulating which suggest that considerable information does enter some long-term memory system, and alternative views of the precise basis for the amnesic defect have arisen.

In principle, there are three types of models: (1) those which hold that there is some general defect in memory storage; (2) those which hold that there is a defect in retrieval processes; (3) those which hold that there is a selective defect in some aspects of storage, or in the storage of certain kinds of information. We hold the last of these positions; there is, according to our view, a selective loss of the ability to establish cognitive maps, and this leads to the loss of that form of long-term memory which utilizes spatio-temporal context for providing efficient retrieval of prior experience. Long-term storage in amnesics, on this view, depends completely upon taxon systems, which fail to provide this context and which work on principles leading to extensive interference between items which are similar in any way. This model predicts both faulty storage and faulty retrieval, characterized by abnormal interference, in much the same way that, in discussing the infra-human data, it predicts faulty place learning and abnormal persistence.

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* H.M. has not been tested formally on his mapping ability; his disorientation in space is virtually complete. However, he could draw an accurate floor plan of the house he was living in (Milner et al. 1968, Corkin, personal communication) after eight years, though he could not describe his place of work or the route to and from work. Further, H.M. has recently changed residence, and is unable to draw a floor plan of his new home after 18 months experience (Milner 1976, personal communication).

15.6.2. SHORT-TERM MEMORY

As we noted in the previous chapter, there is good evidence for the existence of short-term memory systems, based on some form of threshold reduction in neural representations, in both verbal and non-verbal modes. The information available in these systems is subject to decay, which can be prevented, as we have seen, by rehearsal. Thus, in order to arrive at a measure of pure short-term memory function and its decay properties rehearsal is often prevented by distracting tasks meant to absorb the subject's attention. Assuming that such distraction does prevent rehearsal, retention observed after a certain delay (c. 20-30 s) would reflect retrieval from a long-term memory system. Thus, in normal subjects rehearsal might not have a particularly dramatic effect upon retention; long-term memory can substitute for the decayed short-term trace. However, in amnesics, who lack long-term memory systems free from strong interference effects, such distraction could have much more serious effects.

Short-term memory, as it is traditionally defined, should be intact in amnesic patients. That is, that part of retention due to retrieval from transiently activated taxon stores in intact subjects should be evident in patients lacking a locale system. Further, given an opportunity to rehearse, amnesics should be capable of maintaining information in these stores for as long as necessary. This being said, the assessment of short-term retention in amnesics, when rehearsal is prevented and interfering material is interpolated between stimulus presentation and retention, could elicit deficits; this follows from what was said above concerning the contribution of long-term memory to short-term retention. This somewhat perplexing situation results from a confusion in the psychological literature between the *processes* being studied and the *means* of studying them. Short-term and long-term memory are terms we have applied to postulated neural processes; they are not necessarily parallel to those processes reflected in retention tests based solely on manipulating retention intervals. It should not be surprising, therefore, that controversy exists in the literature concerning the status of short-term memory in amnesic patients.

It has been suggested that short-term and long-term memory can be differentiated in terms of the nature of coding imposed on information; in the case of verbal information short-term memory is supposed to involve acoustic coding, while long-term memory involves semantic coding (e.g. Baddeley and Dale 1966). The usefulness of this separation seems to break down in the study of amnesics; though their short-term memory capacity seems intact and their long-term storage defective, there are indications that they are not incapable of coding verbal items on a semantic basis. Any discrepancy here could be resolved in either, or both, of two ways: (1) there might be some intact long-term storage in amnesics which utilizes semantic-category coding; (2) there might be semantic coding within short-term memory itself. We shall see that the former of these is almost certainly true, while the latter remains unclear.

There are two views of the status of short-term memory in amnesic patients, and considerable data which seem to support each position.* Warrington and her colleagues (e.g. Baddeley and Warrington 1970, Warrington and Baddeley 1974, Weiskrantz and Warrington 1975) suggest that amnesic patients have normal short-term memory. On the other side, Butters and his colleagues, in a series of reports (e.g. Samuels *et al.* 1971; Cermak, Butters, and Goodglass 1971, Cermak and Butters 1972, Butters and Cermak 1974, De Luca, Cermak, and Butters 1975) have indicated that there are deficits in short-term memory performance in Korsakoff patients.

For a time it seemed as though these conflicting reports might not be reconciled without assuming important differences in patient populations (cf. Butters and Cermak 1974), a most unfortunate possibility. However, a more parsimonious explanation might be possible, one which extends our understanding of the selectivity of the amnesic deficit and which makes sense of recent reports. A closer look at the results of some of these studies will bring this out. It is important at the outset to try and make some sense of the role played in short-term memory by rehearsal and distraction, as these can easily cloud the issue. With this in mind it is perhaps best to look first at the short-term memory for non-verbal items, which seems less subject to the facilitating effects of rehearsal.

15.6.2(a). Non-verbal short-term memory. Prisko (cited by Milner 1966) tested both unilateral temporal lobectomy patients and H.M. on a modified version of Konorski's delayed paired comparison test, in which two stimuli are presented successively with varying inter-stimulus intervals; the subject must state whether the stimuli were identical or not. Prisko used stimuli which were not easily amenable to verbal rehearsal, such as clicks, tones, and colours (shades), and an interpolated distraction was used on some of the trials. Patients with unilateral lesions performed normally on this task, even with delays of 60 s and interpolated distraction, except for a deficit on nonsense patterns in the right-temporal group. H.M., on the other hand, performed normally only at zero delay, and was severely impaired at the 60 s delay with or without distraction. This failure beyond 30 s is consistent with the results of Sidman *et al.* (1968), who tested H.M. on the delayed matching of ellipses and found that his behaviour was reliably under stimulus control only for 25 s or so. As Milner (1966) pointed out, it seems likely from these results that normal subjects perform well at long intervals through the mediation of some long term memory component; this is in accordance with our previous comments.

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* We shall not discuss 'immediate memory' as measured by such things as digit span. This appears intact in amnesics (e.g. Drachman and Arbit 1966, and many others).

The failure of distraction to affect the pattern of results suggests that at these longer intervals rehearsal is not a useful strategy for non-verbal materials.*

In agreement with these results, Wickelgren (1968) demonstrated good short-term memory on a tonal recognition task in H.M. Finally, Warrington and Baddeley (1974) have reported normal performance on a short-term memory task involving retention of the visual location of a dot, after a filled interval, in their mixed group of amnesics. This equivalence in performance between amnesics and controls was evident only at retention intervals up to 30 s, at which time the extent of control errors reached an asymptote, while that of amnesics continued to increase. This is consistent with the notion that, in controls, retention after 30 s was mediated at least partly by a long-term memory system unavailable to the amnesics.

In contrast to the uniform results provided by these studies, work from Butters' laboratory presents a more confusing picture (cf. Samuels *et al.* 1971, Samuels, Butters, and Fedio 1972, Butters *et al.* 1973, De Luca *et al.* 1975). The first two studies, using Korsakoff patients, demonstrated profound deficits in the short-term (9, 18 s) recall of non-verbal material. However, the two subsequent studies found normal short-term retention of non-verbal material, presented visually, auditorally, or tactually, in both unilateral temporal lobectomy patients and Korsakoff patients. The final study reported deficits in retention of nonsense shapes at 20 s in Korsakoff patients. This confusion has not been clarified to date, though Butters and Cermak (1975) now seem to feel that the short-term retention of non-verbal material, in the absence of potentially interfering distraction, is relatively normal in amnesic patients, at least up to 15 s or so.

All of the reports on non-verbal short-term memory are in agreement in finding that the results are not influenced by distraction during the retention interval, though it is important to note that this distraction has usually involved an interpolated verbal task, even if sometimes presented visually. This is pertinent because De Luca *et al.* (1975) have shown that distraction only prevents good short-term retention in verbal tasks in Korsakoff patients if it is verbal, and presumably thereby interferes directly with the test material. Butters and Cermak (1975) cite a study in progress which indicates that auditory distraction might be capable of interfering with the short-term retention of an auditory non-verbal task in amnesics; this is a possibility which only future research can clarify.

The following conclusions can be drawn from this survey: (1) non-verbal short-term memory seems to decay in about 15-30 s, depending on the nature of the material; (2) verbal distraction does not interfere with nonverbal short-term memory, in agreement with recent work in normal human subjects; (3) beyond 15-30 s performance is mediated by long-term memory systems; (4) amnesics perform normally during the time when short-term memory mediates performance, but are deficient beyond that.*

15.6.2(b). Verbal short-term memory. When we move to a consideration of short-term memory for verbal material, matters become even more confusing. H.M.'s verbal short-term memory is normal so *long as he is not distracted,* and he utilizes rehearsal to maintain his short-term memory for as long as is necessary. Once distracted, however, he loses the material completely, as well as the entire chain of rehearsal. Wickelgren (1968) tested H.M. in a short-term recognition paradigm and showed that a strength model of short-term memory (similar to the one we proposed above) fitted H.M.'s performance adequately; further, his decay functions were within the normal range. Though other temporal lobectomy patients have not been tested as exhaustively as H.M., it seems clear that much the same effects hold for them as well; when undistracted, short-term memory is normal. The fact that recall after distraction is so poor in amnesics, compared with normals, indicates again that retention in the latter can be mediated by a long-term memory component.

Baddeley and Warrington (1970) demonstrated normal short-term memory capability in a group of amnesics in two ways: (1) the amnesics showed normal recency effects in serial list learning, that is, they preferentially recalled the terminal items in the list; (2) the amnesics showed normal decay functions up to delays of 60 s in the Peterson forgetting paradigm, which involves presentation of three items and recall after varying delays. The normal forgetting curve seen on the Peterson task is surprising, as this task involves the interpolation of a distracting problem (typically backwards counting) between presentation and recall. As such, these data seem to be in disagreement with those obtained from temporal-lobe patients; as we have just seen, verbal short-term memory is seriously disrupted in these patients by distraction.** They are also in disagreement with a series of reports from Butter's laboratory (e.g. Samuels *et al.* 1971, Cermak *et al.* 1971, Goodglass and Peck 1972, Cermak and Butters 1972, Butters *et al.* 1973, De Luca *et al.* (1975) indicating that distraction during the retention interval can yield short-term memory defects in verbal tests.

The final study in this series of reports provides the most detailed information on the nature of the effect of distraction upon short-term verbal retention in amnesics. Both verbal and non-verbal distractors were used in conjunction with the verbal material, and it was seen that only, verbal distractors elicited a defect. In the absence of any distraction, amnesics retained verbal materials quite well. These data suggest that

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* This was confirmed, as we shall see, in a study by De Luca et al. (1975).

* The demonstration that H.M. estimates time accurately up to, but not beyond, about 20 s (Richards 1973) is probably a manifestation of this accurate short-term memory system.

** Corsi (1969) has extended this result to patients with unilateral left hippocampal damage.

distractors influence short-term memory performance by pre-empting the limited processing capacity of the verbal short-term memory system, thereby preventing rehearsal. The authors argue that the distraction task used by Baddeley and Warrington, backwards counting by 2's, was not sufficiently difficult to prevent rehearsal, a point also raised by Cermak et al. (1971) and Butters and Cermak (1974).

The weight of the available data support the conclusion that verbal short-term retention, when assessed in a paradigm employing verbal distraction, is defective in amnesics. Thus, we would accept that there is probably something about the methods used by Baddeley and Warrington which could account for the discrepant results; the possibility of rehearsal in the face of the distraction they used is a good candidate. This does not mean that amnesic patients lack a verbal short-term memory system. The results of the serial-list learning experiment (Baddeley and Warrington 1970) are quite clear on this point. It does mean, however, that information in this system decays within a short period of time unless rehearsed, and that in normal subjects a long-term system can mediate retention when the short-term system has been disrupted.

15.6.3. LONG-TERM RETENTION

Notwithstanding the considerable interest shown in short-term memory function in amnesics, it is clear that the primary focus of interest in this syndrome is in long-term memory. Most theoretical treatments have stressed this function, and we shall discuss the problem at some length. Three major theoretical positions have emerged in recent years, as we noted above. More specifically, these suggest that (1) there is a failure in the transfer of information from a short-term memory system to a long-term system (e.g. Milner 1966), (2) there is a failure in the selective forgetting of unwanted information, leading to abnormal interference during retrieval (e.g. Warrington and Weiskrantz 1971, Weiskrantz and Warrington 1975), and (3) there is a deficit in the spontaneous encoding of inputs along semantic, or other complex, lines, leading to poorer recall and increased interference from more simply coded memories (e.g. Butters and Cermak 1975).

At the present time there is sufficient evidence to disqualify all of these models, though the phenomena they purport to explain remain to be accounted for. In what follows we hope to show that a selective deficit in the storage of cognitive maps and the concomitant dependence upon interference-prone taxon memory systems can account for the known facts of the amnesic's long-term memory defect. We shall be concentrating here upon the long-term memory for verbal information, as the bulk of the data pertain to this. Our discussion of maze learning and spatial orientation has already demonstrated that the long-term storage of spatial maps is probably defective in amnesics; an analysis of verbal learning will raise the possibility of an analogous defect in the formation and storage of semantic maps.

In addition to reviewing data from studies of new learning, we shall look briefly at the way in which previously stored information is affected by the onset of amnesia, that is, the retrograde aspect of the disorder. Here, we shall show that old memories incorporated in semantic maps are also disrupted in amnesics, regardless of the time of storage. We shall be stressing the selectivity of the defect in terms of the form of memory affected, while at the same time attempting to show that this selective defect holds for both retrograde and anterograde deficits, and regardless of the form of retention test employed. Finally, we shall discuss the way in which abnormal interference effects result from the absence of a memory storage system designed to limit interference, in accordance with our previous discussion of the properties of imagery and cognitive mapping.

15.6.3(a). Basic effects. The long-term retention (beyond 60 s) of verbal material has been shown to be impaired in amnesics in a host of studies. In a way, the really interesting studies are those which define circumstances under which some long-term memory can be displayed by amnesics. The first indication that information was indeed getting into some kind of verbal long-term memory store came from several studies showing that the provision of information at the time of retrieval could improve the performance of amnesics (Warrington and Weiskrantz 1968). Such findings invalidated any theory of a general defect in the consolidation of information from short-term to long-term memory. It has now been shown, in a number of ways, that the provision of cues at the time of retention markedly improves the performance of amnesic patients.

The importance of these retrieval cues in the performance of subjects at retention is intuitively sensible; efficient recall of any specific bit of information from the welter of stored experiences requires a highly selective means of retrieving that information. Tulving and Pearlstone (1968) have emphasized this fact by drawing a distinction between the *availability* and *accessibility* of stored information, that which is available is not necessarily accessible. One of the functions of the cognitive-mapping system is to provide the basis for effective access to memory by imposing upon the latter a spatio-temporal framework which makes the identification of information simpler. Of course, certain forms of information ‘emerge’ from the cognitive map itself and are totally dependent upon it. Thus, knowledge of places requires the map, as does knowledge for the specific context within which events occurred. No manner of retrieval cue should enable the amnesic to improve the recall of such information; the facts are simply missing.

However, retrieval cues can aid the amnesic in attempts to get at information stored in the taxon systems, information which might be interfered with in the absence of such cues. Faulty retrieval derives from, in part, the category-based storage properties of the taxon systems. Retrieval cues act to circumvent these properties by specifying the information required. Thus, we are suggesting that the amnesic deficit includes both faulty storage (no maps) and faulty retrieval (no context).

15.6.3(b). Faulty retrieval. The poor retrieval of amnesic patients is generally characterized by abnormally large interference effects from information stored at other times.* This has been shown in a variety of situations in highly specific ways. Thus, repeated testing on word lists yields considerable intrusions from one list to another (cf. Weiskrantz and Warrington 1975 for a review of these interference effects), and interference itself can be shown to depend upon the similarity between items in different lists (cf. Butters and Cermak 1975 for a review of relevant studies). To put it in simple terms, the amnesic patient fails to provide outputs which are appropriate to the context required.

Retrieval cues, be they in the nature of partial information (Warrington and Weiskrantz 1968, 1970) or reminders of the categories to which words belonged, serve to reduce markedly the number of potential response alternatives. Thus, they reduce the possibility of responding with items which are contextually inappropriate. This effect holds for both recall and recognition testing. Recognition tests, as we noted earlier, are typically stronger tests of contextual memory than are recall tests. Proper recognition, in most experiments, is based not on some simple familiarity process, but rather on the knowledge that a certain (already familiar) item, or pattern, occurred in a particular experimental context. It has been known for some time that amnesic patients are particularly defective at recognition tests, and we would assert that this reflects the dependence of recognition on contextual memory. At the same time, however, it has been noted anecdotally that amnesic patients can often recognize faces as familiar or not, even though they cannot specify where or when the person in question was seen. This suggests that if recognition memory was assessed with materials which were truly unfamiliar, thereby eliminating any dependence upon contextual information, it might prove relatively normal in amnesic patients. Recent work confirms this prediction (Piercy and Huppert 1972, Huppert and Piercy 1976). When the recognition of familiar, high-frequency words was tested the standard deficit appeared. However, when the recognition of unfamiliar, low-frequency words was tested recognition memory was substantially improved, though still slightly inferior to normal performance.**

In free-recall tests a wide variety of retrieval cues have been shown to facilitate the performance of amnesics. That this effect works through improving retrieval of otherwise inaccessible material, rather than through improving storage, was shown by Cermak (1975). He found that cues available only at the time of stimulus presentation did not facilitate retrieval. Such cues must be present during the retention test in order to be useful. This result has been confirmed by Kinsbourne and Wood (1975). Much of the work on the use of retrieval cues has been aimed at uncovering a presumed defect in semantic encoding by amnesics, a proposal forwarded by Butters, Cermak, and their co-workers (cf. Butters and Cermak 1975).* While this work has suggested that amnesic patients encode less information about the semantic properties of words, it has not gone beyond that to an explanation of why this faulty semantic encoding should lead to the variety of defects seen in amnesic patients. This defect is limited to the *spontaneous* use of semantic encoding (Cermak, Butters, and Gerrein 1973); amnesics *can* encode semantic properties of words when directed to do so. Thus, the defect might be related to the short-term memory deficit reported by this group of investigators; in the above study, semantic information seemed to be spontaneously available to amnesics for at least a short period of time. Further, the demonstration that cues given at stimulus presentation do not, by themselves, facilitate recall indicates that the improvement seen with cues given at retrieval lies in their helping the subject gain access to stored material. Semantic retrieval cues could not be effective unless information was stored on the basis of semantic properties in the first instance.

The facilitating effects of cued recall can be mimicked by the use of memory tests which benefit subjects able to use any form of similarity between test items. Thus, lists in which words from the same category are clustered together elicit improvements in amnesics (Baddeley and Warrington 1973), while paired associates invoking phonetic or semantic relations between the stimulus and response items yield normal performance in amnesics (Winocur and Weiskrantz 1976).

The performance of amnesics during cued recall is consistent with the notion that excessive interference is at least partially responsible for the amnesic deficit, as Warrington and her co-workers have suggested. However, there is little evidence to support their contention that this interference owes to the mechanism they propose, a loss of selective forgetting, whatever that might actually mean. This position cannot predict the selective loss of any storage facility and, as we shall see, there is accumulating evidence that such a selective defect is central to the amnesic syndrome.

One form of retrieval aid which does not seem to facilitate amnesic

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* These interference effects show up in a variety of situations, including those involving hypothesis behaviour (e.g. Oscar-Berman 1973).

** These data indicate that, contrary to Gaffan's (1972, 1974) claim, familiarity detection is intact in amnesics, at least when spatio-temporal context is unimportant to the familiarity or unfamiliarity of the test material.

* These authors are talking about the semantic properties of single words, which is something quite distinct from what psycholinguists mean when they use the term or what Tulving (1972) meant by the same term.

performance concerns the use of imagery techniques (e.g. Baddeley and Warrington 1973). This suggests that there are forms of storage, facilitated in intact subjects by imagery instructions, which are unavailable to amnesic patients; we shall pursue this point shortly.

To sum up, the use of cued recall, or recognition tests with unfamiliar materials, indicates that certain forms of long-term verbal memory can be vastly improved in amnesic patients. This improvement seems to reside in the provision of a way to use available category memory without invoking the strong interference effects which typically plague memory retrieval from these systems. There is no indication in this work that amnesic patients can use anything other than material-specific, or category, cues, depending for their efficacy upon identity with, association with, or similarity to the to-be-recalled information. None the less, such cues can be quite effective in helping the amnesic in many clinical tests; that these tests have generally concentrated upon the retention of individual items, or clusters of categorially related items, is surely of some significance.

15.6.3(c). Faulty storage. As we have just seen, the use of cued recall tests indicates that amnesics can store information about the categorial properties of much verbal information. However, these studies do not directly test the ability of the subject to store contextual memory.

Some evidence pertinent to this question comes from studies exploring the effects of the use of imagery upon verbal learning in amnesics. With the exception of the study by Cermak (1975), the use of imagery totally fails to improve the performance of amnesic patients with bilateral damage (Jones 1974, Baddeley and Warrington 1973).* As expected, patients with unilateral damage in the left hemisphere, who show profound verbal memory defects (cf. Corsi 1972), are benefited by the use of the imagery techniques (Patten 1972a, Jones 1974). These data indicate that the hippocampus is crucial to the facilitating effects of imagery.**

Other evidence pertinent to the question of faulty storage comes from the study of the retrograde effects of hippocampal damage. Initial thinking on the nature of these effects suggested that retrograde loss was most marked in the period just prior to the operation, with a relative sparing of older memories (e.g. Milner 1966). The development of a more precise method for the assessment of 'old' memories, the long-term questionnaire, provided evidence against this assumption by showing that there was considerable memory loss at all times tested, including periods 30 and 40 years prior to the onset of hippocampal dysfunction (Sanders and Warrington 1971).

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* Cermak showed that amnesics were facilitated by instructions to tie two words together; Baddeley and Warrington showed that amnesics could not benefit from imaging four words together.

** Amnesic patients had no trouble forming an image, e.g. a picture, but they could not store information which would enable them to retrieve that image. This is further evidence that imagery works, not through the storage of a picture, but rather through the storage of some set of instructions (or deep structure) that enables the subject to recreate the image when needed.

More recent work (Marslen-Wilson and Teuber 1975, Sanders and Warrington 1975), which combined the long-term questionnaire technique with cued retention, showed that much of the old material thought to have been lost was actually available, though inaccessible without cueing. Though amnesics required more cues than did controls, they still showed a vast improvement in performance. These data, then, in common with the data from studies of cued recall in recent learning, indicate that amnesics have trouble getting at stored information. However, Kinsbourne and Wood (1975) have provided evidence that the deficit in retention of old memories goes beyond a loss of access. Amnesics were asked to produce a personal memory in response to high-imagery, high frequency words. Whereas control subjects produce highly specific memories in this situation, amnesics

> 'do not retrieve individual events at all. They characteristically, instead, and contrary to instruction, retrieve categorical information about the appropriate use or general availability of the object represented by the stimulus word' (p. 278).

The authors concluded that their amnesics could retrieve considerable general information from their early life, but could not retrieve specific event memories at all. They suggested that there is a selective failure in what Tulving (1972) referred to as episodic memory and what we have referred to as context-dependent locale memory. A similar suggestion was made by Gardner *et al.* (1973) and by Huppert and Piercy (1976).

Unfortunately, there is little direct evidence at present which would address the question of the role of the hippocampus in semantic maps, as we defined them above.* Lackner (1974) has shown that H.M. can detect certain linguistic ambiguities, but at a markedly reduced rate. We know from the work of Talland (1965) and others (e.g. Murray and Hitchcock 1973) that Korsakoff patients are quite poor at the recall of narrative, but this could be attributable to many things. Future research will have to elucidate the precise nature of any hippocampal role in context-specific memory. Present evidence suffices to assert that there is, in amnesics, a selective deficit in the establishment and long-term storage of event memory.

15.6.3(d). Conclusions. The study of the long-term aspects of memory loss in amnesic patients reveals several basic features to the syndrome.

(1) There appears to be a selective loss in the storage of event memory, or memory which is specified as to spatio-temporal context.
(2) There is relatively normal storage of category memory, up to and

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* Glowinski (1973) has shown that temporal-lobe epilepsy patients are markedly impaired at the recall of narrative texts, even when tested immediately. One can think of many tests that would get at the question of the role of the hippocampus in deep structure; the recent work of Bransford and McCarrell (1974) provides many examples of tests that should help to pinpoint this function.

including categories based on the semantic features of individual items.

(3) Retrieval from these category stores is considerably retarded in amnesics because of the powerful interference effects acting between items of a similar nature. Normal subjects can circumvent this difficulty through the use of context-specific memories, which are largely free of interference problems.

(4) The provision of retrieval aids, at the time of retention, helps amnesics to gain access to the information available in their taxon category stores. It cannot, however, substitute for the lost event-specific memory system. Such memories are neither accessible nor available to the amnesic.

(5) This loss of event-specific memory seems to include those memories formed before the onset of the disease or the surgical intervention.

15.6.4. OVERALL CONCLUSIONS

This review of the amnesic literature confirms that a theory postulating cognitive-mapping deficits after human hippocampal damage provides a decent fit to the available data. Clear-cut evidence for defects in exploration and spatial mapping is available, and there seems little doubt that the right hippocampus is crucial to these functions. Evidence for defects in semantic mapping and the use of imagery techniques is also available, while the assumption of a selective storage defect makes sense of the concomitant retrieval defect, expressed as abnormal interference, that is seen in amnesics. In virtually every way the effects of damage to the hippocampus in humans seem analogous to those seen in infra-humans.

Appendix

Appendix

TABLE A1 *(a)*

Anatomical studies of the septal afferents to the hippocampus

Daitz and Powell 1954
McLardy 19551a
Cragg and Hamlyn 1957[*]
Votaw 1960b
Powell 1963
Votaw and Lauer 1963a
Alksne and Blackstad 1965
Raisman *et al*. 1965
Anden *et al*. 1966
De Vito and White 1966
Petsche, Gogolak, and Stumpf 1966[*, **]
Raisman 1966
Lewis and Shute 1967
Lewis *et al*. 1967
Genton 1969
Storm-Mathisen 1970
Ibata *et al*. 1971
Siegel and Tassoni 1971b[**]
Storm-Mathisen and Fonnum 1972
Hjorth-Simonsen 1973
Mosko *et al*. 1973
Sethy *et al*. 1973
Srebro *et al*. 1973
Mellgren and Srebro 1973
Oderfeld-Nowak *et al*. 1974
Segal and Landis 1974
Srebro and Mellgren 1974
Storm-Mathisen and Guldberg 1974

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[*] Combined electrophysiological and histological study.

[**] Indicates disagreement with majority of studies that septo-hippocampal projection originates in the medial septum and diagonal band of Broca.

TABLE A1 (b)

Physiological studies of septal afferents to the hippocampus

Cragg and Hamlyn 1957
Andersen *el al*. 1961a,b
Brucke *et al*. 1963
Shaban 1970
Brust-Carmona *et al*. 1973 *
De France *et al*. 1973
Dudar 1975

∗ Indicates disagreement with majority of studies that the septo-hippocampal projection originates in the medial septum and diagonal band of Broca.

TABLE A2

Anatomical studies of the efferent connections of the hippocampus

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Allen 1944
Simpson 1952
Sprague and Meyer 1950
McLardy 1955b
Powell and Cowan 1955
Guillery 1956
Nauta 1956
Powell *et al.* 1957
Nauta 1958
Johnson 1959
Valenstein and Nauta 1959
Votaw 1960a
Powell 1963
Votaw and Lauer 1963a
Johnson 1965
Raisman *et al*. 1966
Raisman 1969
Van Buren 1970
De France *et al*. 1971
Hjorth-Simonsen 1971
Siegel and Tassoni 1971a
Pepeu *et al.* 1973
Segal and Landis 1974
Siegel *et al*. 1974
Meibach and Siegel 1975
Swanson and Cowan 1975a,b

TABLE A3

Physiological studies of the efferent connections of the hippocampus

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Kaada 1951
Kaada *et al*. 1953
Adey, Segundo, and Livingston 1957
Adey, Sunderland, and Dunlop 1957
Adey *et al*. 1958
Cazard and Buser 1958
Cragg and Hamlyn 1959*
Livingston 1959
Cragg and Hamlyn 1960*
Flynn and Wassman 1960
Akert and Gernandt 1962
Feldman 1962
Parmeggiani 1962
Cazard and Buser 1963
Fox *et al*. 1967
Gergen 1967
Parmeggiani 1967
Redding 1967
Yamaguchi *et al*. 1967
Psatta, Sirian, and Ungher 1968
Psatta, Ungher, and Sirian 1968
Sierra and Fuster 1968
Vanegas and Flynn 1968
Yokota and MacLean 1968
Parmeggiana and Rapisarda 1969
Redding 1969
Ungher *et al*. 1969
Kerr and Dennis 1970
Poletti *et al*. 1970
Golden and Lobar 1971
Grillo 1971
Kaada *et al*. 1971
Bland and Vanderwolf 1972b,
De France *et al*. 1972
Grantyn *et al*. 1972
McKenzie and Gilbert 1972
Andersen *et al*. 1973
Culberson and Bach 1973
De France, Kitai, Hatada, and Christensen 1973
De France, Kitai, and Shimono 1973a,b
De France, Kitai, McCrea, Kocsis, and Hatada 1973
Edinger *et al*. 1973
Gambarian *et al*. 1973
Poletti *et al*. 1973
Mok and Mogenson 1974
De France, McCrea, and Yoshihara 1975
De France and Yoshihara 1975
De France, Yoshihara, McCrea, and Kitai 1975

∗ Combined electrophysiological and histological study.

TABLE A4

Studies on the brainstem pathways involved in generating hippocampal theta and desynchronization

Kawamura *et al*. 1961
Torii 1961
Corazza and Parmeggiani 1963
Yokota and Fujimori 1964
Stumpf 1965a,b
Parmeggiani 1967
Kawamura and Domino 1968
Anchel and Lindsley 1972
Macadar *et al* 1974
Coleman and Lindsley 1975
Lindsley and Wilson 1975
Paiva *et al*. 1976

TABLE A5

Hippocampal EEG during exploration, orientation reflex, and searching behaviour

Theta	Theta frequency	LIA or SIA
Rat		
Brugge 1965	6-8	
Routtenberg 1968	6-8	
Gray and Ball 1970	7.5-8.5	
Irmis *et al*. 1970	7-8	
Komisaruk 1970[b]		
Pond *et al*. 1970[c]	8-9	
Pond and Schwartzbaum 1970[c]	8-10	
Routtenberg *et al*. 1970		
Albino and Caiger 1971	7.0-7.5	
Irmis *et al*. 1971[a]		
Morales *et al*. 1971	4-8	
Whishaw and Vanderwolf 1971[d]		
Gray 1972	7.5-8.5	
Gray *et al*. 1972	7.5-8.5	
Feder and Ranck 1973		
Kurtz and Adler 1973		
Ranck 1973		
Schwartzbaum and Kreinick 1973	8-9	
Whishaw and Vanderwolf 1973		
Kimsey *et al*. 1974	7-9	
Winson 1974		
Coenen 1975	8	
Kurtz 1975		
Schwartzbaum 1975	8	

TABLE *A5 - cont.*

Hippocampal EEG during exploration, orientation reflex, and searching behaviour

Theta	Theta frequency (Hz)	LIA or SIA
Gerbil		
Kramis and Routtenberg 1969	7-9	
Hamster		
Macrides 1975[b]	8.1-8.7	
Rabbit		
Torii and Sugi 1960	8	
Polc and Monnier 1970[c]	6-7	
Dog		
Yoshii *et al*. 1967	5-6	
Cat		
Grastyan *et al*. 1959	5	
Adey *et al*. 1960	4.0-7.5	
Lissak and Grastyan 1960		
Adey *et al*. 1962		
Brown and Shryne 1964[g]	3-6 early, 3.5-4.8 late	
Porter *et al*. 1964	4 -5	
Karmos *et al*. 1965		
Grastyan *et al*. 1966[h]		
Brown 1968	4.3-4.7	
Bennett 1970		
Anchel and Lindsley 1972[f]		
Bennett *et al*. 1973	4.7	
Grastyan and Vereczkei 1974	4-5	
Coleman and Lindsley 1975	4.5-5.5	
Kemp and Kaada 1975	up to 8.5	

a Good correlation between theta frequency during low-voltage fast sleep, amount of exploration in a novel environment, and amount of response to an auditory stimulus.
b Theta sometimes synchronizes to vibrissa twitch and sniff and (Komisaruk only) heart beat during exploratory sniffing.
c Elicited by stimulation of the lateral hypothalamus.
d No synchronization between theta and exploratory sniffing.
e Elicited by stimulation of the raphe nucleus and reticular formation.
f Elicited by stimulation of medial hypothalamus as well as occurring naturally.
g Early orienting involves whole-body exploration; late orienting involves primarily visual searching.
h Elicited by hypothalamic stimulation.

TABLE A6(a)
Hippocampal EEG during motor behaviours: spatial displacement [a]

Theta	Behaviour[a] and theta frequency (Hz)	LIA or SIA
Rat		
Vanderwolf and Heron 1964		Routtenberg 1968
Vanderwolf 1969	w 8.0-8.3; rr 8.0-8.3; j 8-12	
Gray and Ball 1970	r 8-10	
Irmis *et al*. 1970	m 8-9	
Paxinos and Bindra 1970	8	
Albino and Caiger 1971	cl 7.5	
Teitelbaum and McFarland 1971	7	
Whishaw and Vanderwolf 1971	sw 7.5-8.3	
Bland and Vanderwolf 1972a[b]	r 8-9; j 9-10	
Gray 1972	8.5-10	
Gray *et al*. 1972	8.5-10	
Whishaw *et al*. 1972[e]		
Pond and Schwartzbaum 1972	w 8-10	
Feder and Ranck 1973		
Ranck 1973		
Vanderwolf *et al*. 1973		
Whishaw and Vanderwolf 1973	r 7.7; sw 7.6; j 9-11	
Kurtz and Adler 1973	w 8-10; m 7.9	
Fleming and Bigler 1974		
Kimsey *et al*. 1974	r 9.1	
Robinson and Whishaw 1974[c]	w 7.6	
Winson 1974		
Coenen 1975	8	
Kurtz 1975	r 8.5; m 8	
McFarland *et al*. 1975		
Vanderwolf 1975[d]	j 8	
Vanderwolf *et al*. 1975		
Myhrer 1975[d]		
Morris, Black, and O'Keefe 1976		
Gerbil		
Whishaw 1972	r 7.7	
Guinea pig		
Sainsbury 1970	w 8.3	

TABLE A6-*cont.*
Hippocampal EEG during motor behaviours: spatial displacement[a]

Theta	Behaviour[a] and theta frequency (Hz)	LIA or SIA
Rabbit		
Sadowski and Longo 1962[f]	8-9	
Klemm 1970		
Harper 1971	8	
Klemm 1971	5-9	
Kramis *et al*. 1975	hp 8; r up to 12	
Dog		
Lopes da Silva and Kamp 1969	5-6	Yoshii *et al*. 1967[h]
Yoshii *et al*. 1967	w 6-7	
Kamp *et al*. 1971	r 6-7; circle 4-5	
Arnolds *et al*. 1975[g]		
Cat		
Adey *et al*. 1960	r 5-6	Grastyan *et al*. 1959
Holmes and Adey 1960	w 5-6	Grastyan *et al*. 1966[j]
Adey *et al*. 1962	r 6	Bennett 1970
Porter *et al*. 1964	w 6	Bennett and Gottfried 1970
Radulovacki and Adey 1965	r 6	Bennett *et al*. 1973
Grastyan *et al*. 1966[j]	w	
Elazar and Adey 1967	r 5-6	
Whishaw and Vanderwolf 1973	w 5	
Grastyan and Vereczkei 1974[i]	6-7	

[a] Including walking (w), running (r), swimming (sw), jumping (j), rearing (rr), climbing (cl), male mounting during sex (m), and hopping (hp).
[b] Elicited by stimulation of the posterior hypothalamus.
[c] Theta frequency during walking is lowered to 6.6 Hz after posterior hypothalamic lesions.
[d] Two types of theta: non-movement theta is abolished by atropine and is elicited by eserine, ether, or mid-brain reticular stimulation; movement-related theta is not affected by atropine, and is stimulated by amphetamine and depressed by phenothiazines.
[e] Elicited by stimulation of the lateral hypothalamus.
[f] Theta during walking not affected by scopolamine but theta during alert immobility is (see Table A8(b)).
[g] Shifts in theta frequency are related to changes in mode of behaviour, e.g. from standing to walking.
[h] SIA recorded when animal abruptly changes direction of movement.
[i] Occasionally SIA during approach in early stages of conditioning.
[j] Stimulation through some hypothalamic placements causes running associated with hippocampal theta: at higher levels of stimulation very fast running was associated with SIA.

TABLE A6*(b)*

Hippocampal EEG during motor behaviours: non-spatial, non-consummatory[a]

Theta	Behaviour and theta frequency (Hz)	LIA or SIA
Rat		
Vanderwolf 1969[b]	hm 6.6-7.1; hfp 6.6-7.1	Vanderwolf 1969[b]
Whishaw and Vanderwolf 1971		Whishaw and Vanderwolf 1973[b]
Bland and Vanderwolf 1972*a*		
Whishaw *et al*. 1972		
Feder and Ranck 1973		
Ranck 1973		
Vanderwolf *et al*. 1973		
Whishaw and Vanderwolf 1973[b]		
Frederickson *et al*. 1974	bx	
Vanderwolf 1975	st 8.2	
Vanderwolf *et al*. 1975		
Gerbil		
Whishaw 1972		
Guinea pig		
Sainsbury 1970	dg 8.3; hm 7.3	
Dog		
Yoshii *et al*. 1967	hm 5-6	
Cat		
Sakai *et al*. 1973	em 3.5-4.3	Grastyan *et al*. 1959
Whishaw and Vanderwolf 1973		Kemp and Kaada 1975
Glotzbach 1975		

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[a] Including head movements (hm), eye movements (em), limb movements as when an animal boxes during fighting (bx), rotates a food pellet during eating (hfp), or digs (dg), postural adjustments (pa), and struggling while held (st).

[b] During small head movements and food pellet manipulation theta was recorded at some hippocampal placements and not others.

TABLE A6*(c)*

Hippocampal EEG during motor behaviours: lever pressing

Theta	Schedule and theta frequency (Hz)	LIA or SIA
Rat		
Bremner 1964	SA 5-7	Feder and Ranck 1973 (CRF & FR50)
Yoshii *et al*. 1967	CRF 7-8	
Vanderwolf 1969	CRF	Whishaw and Vanderwolf 1973 (CRF)
Pond and Schwartzbaum 1972[c]	FR	
McGowan-Sass 1973[b]	SA 6.6	
Whishaw and Vanderwolf 1973[d]	CRF	
Frederickson 1974*a*	FR	
Bennett 1975	CRF 6.6 c-CRF 6.6; DRL 6.8; c-DRL 6.2	
Gerbil		
Kramis and Routtenberg 1969		
Cat		
Bennett *et al*. 1973	CRF; c-DRL	Bennett 1970 (discriminated CRF)
Hatfield 1975	CRF 3.5-5.5	Bennett and Gottfried 1970 (CRF-DRL)
Dog		
Dalton and Black 1968		Yoshii *et al*. 1967 (CRF)
Black and Young 1972a	CRF 4.5-5.0	Lopes da Silva and Kamp 1969 (CRF)

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[a] Abbreviations: DRL (differential reinforcement of low rates of lever pressing); c-DRL (cued DRL); CRF (reinforcement for each lever press); FRx (reinforcement for each xth lever press); SA (Sidman avoidance schedule in which each lever press postpones a shock for a given period of time).

[b] Theta disappears over time from the ventral, but not the dorsal, hippocampus.

[c] More theta during lever presses 1-8 than during lever presses 9-16 of FR16.

[d] Theta during lever pressing from some hippocampal placements but not others.

TABLE A7

Hippocampal EEG during consummatory behaviours[a]

Theta	Behaviour[a] and theta frequency (Hz)	LIA or SIA
Rat		
Gray and Ball 1970[i]	d 6-7.5	Routtenberg 1968
Irmis *et al*. 1970	ej 8-9	Vanderwolf 1969
Pond *et al*. 1970[c]	e 6-8	Routtenberg *et al*. 1970
Gray 1972i	d 6-7.5	Pond *et al*. 1970[c]
Gray *et al*. 1972[i]	d 6-7.5	Albino and Caiger 1971
Kurtz and Adler 1973	in 8.2; ej 7.2	Whishaw and Vanderwolf 1971
Kimsey *et al*. 1974[i]	d 6.5	Black and Young 1972b[b]
Kurtz 1975	li 7; lej 6.5	Whishaw *et al*. 1972[d]
		Bland and Vanderwolf 1972a
		Pond and Schwartzbaum 1972
		Feder and Ranck 1973
		Ranck 1973
		Whishaw and Vanderwolf 1973
		Robinson and Whishaw 1974
		Coenen 1975
Gerbil		
		Kramis and Routtenberg 1968
		Whishaw 1972
Guinea pig		
		Sainsbury 1970
Cat		
		Whishaw and Vanderwolf 1973
Rabbit		
Torii and Sugi 1960	a 5-7; g 5-7	Kramis *et al*. 1975[c]
Sadowski and Longo 1962	e 4-6	
Huston and Brozek 1974	d 5.4; e 6.2-6.4	
Kramis *et al*. 1975	d 5.8[f]; e 6.5[g]; g 6.5	
Dog		
Yoshii *et al*. 1967[h]		Yoshii *et al*. 1967[h]
Konorski *et al*. 1968	e 4.3	Lopes da Silva and Kamp 1969

TABLE A8(a)

Hippocampal EEG during immobility prior to jumping

Theta	Theta frequency (Hz)	LIA or SIA
Rat		
Vanderwolf 1969	6.7	
Vanderwolf *et al*. 1973		
Whishaw and Vanderwolf 1973	7	
Vanderwolf and Cooley 1974		
Myhrer 1975[d]	8-10	
Vanderwolf 1975	6	

---

a Including eating (e), drinking (d), grooming (g), chewing (ch), male intromission (in) and ejaculation (ej) during sexual intercourse, female lordosis during male intromission (li) and ejaculation (lei), sniffing without movement, teeth chattering, vomiting, yawning, shivering, urination, defecation, vocalization, salivation, piloerection, and foot stomping in gerbils.

b Water licking used as operant to avoid shock.

c Normal eating in the unstimulated condition is associated with LIA, but agitated eating elicited by lateral hypothalamic stimulation is associated with theta but the frequency is 1-2 Hz below that elicited by the same stimulation in the absence of consummatory behaviour.

d Eating, drinking, and grooming elicited by lateral hypothalamic stimulation accompanied by LIA unless rat is `fidgety' when theta occurs (see note c).

e LIA during chewing without head movements.

f Theta during drinking in 7 of 11 rabbits.

g Theta during eating with head movements.

h Theta during mastication, LIA during eating, drinking, urination, and barking,

i Recording electrodes in the subiculum.

TABLE A8(b)

Hippocampal EEG: animal immobile, alert, and attentive

Theta	Theta frequency (Hz)	LIA or SIA
Rat		
Robinson and Whishaw 1974[a]	4-6	Brugge 1965[b]
Black 1975	< 7	Vanderwolf 1969
		Paxinos and Bindra 1970
		Albino and Caiger 1971
		Ranck 1973
		Vanderwolf *et al*. 1973
		Whishaw and Vanderwolf 1973
		Frederickson *et al*. 1974[c]
		Robinson and Whishaw1974[a]
		Coenen 1975
		Kurtz 1975
		Vanderwolf 1975
Gerbil		
		Whishaw 1972
Rabbit		
Green and Arduini 1954	5-7	Torii and Sugi 1960
Torii and Sugi 1960	5-7	Kramis *et al*. 1975
Sadowski and Longo 1962	4-6	
Klemm 1970[d]		
Harper 1971	7	
Klemm 1971[d]		
Huston and Brozek 1974	7.1	
Cat		
Porter *et al*. 1964	4	Anchel and Lindsley 1971[e]
Radulovacki and Adey 1965	3-7	Grastyan and Vereczkei 1974
Brown 1968	4.4	Hatfield 1975
Bennett 1969		
Bennett 1970		
Whishaw and Vanderwolf 1973		
Coleman and Lindsley 1975		
Kemp and Kaada 1975		
Dog		
Urban *et al*. 1974[d]	5	Dalton and Black 1968
		Black and Young 1972a

[a] Following large posterior hypothalamic lesions, rats sometimes show 4-6 Hz theta during alert immobility.

[b] Occasionally theta when animal fails to orient to a stimulus.

[c] LIA when immobile rat is bitten during fighting.

[d] Theta during immobility associated with phasic increases in EMG and brainstem multi-unit activity.

[e] Associated with cold block of the medial hypothalamic pathway or stimulation of the lateral hypothalamic pathway.

[f] $ACTH_{4-10}$ reduces theta frequency while dog waits for cue to press lever from above 5 Hz to below 5 Hz.

TABLE A9

Hippocampal EEG during sensory stimulation

Theta (sensory modality[a])	Theta frequency (Hz)	LIA or SIA
Rat		
Bremner 1968 (v)[b]	5	Whishaw 1972(a)[c]
Bremner and Ford 1968 (v)	5-6	Ranck 1973
Irmis *et al*. 1970 (a)[d]	6.7-8.5	
Whishaw 1972 (a)[c]		
Schwartzbaum and Kreinick 1973 (v)	5	
Rabbit		
Jung and Kornmuller 1938 (a, n)	5-6	Jung and Kornmuller 1938 (v, l, t)
Green and Arduini 1954 (o, v, a, ta, n)	3-6	
Eidelberg *et al*. 1959 (a)		
Costin *et al*. 1967 (1)		
Harper 1971		
Kramis *et al*. 1975	7	
Martin *et al*. 1975 (a)[e]		
Cats		
Green and Arduini 1954		Grastyan *et al*. 1959 (a)[f]
Grastyan *et al*. 1959 (a)[f]	5	Karmos *et al*. 1965[f]
Karmos *et al*. 1965[f]		
Kemp and Kaada 1975		
Kramis *et al*. 1975		
Dogs		
		Lucas *et al*. 1974[g]

[a] Abbreviations: v (visual), a (auditory), n (noxious), 1 (labyrinthine), t (thermal), o (olfactory), ta (tactile)

[b] No change in theta with habituation to the stimulus.

[c] Theta if rat moves in response to tone; LIA if no movement.

[d] Decrease in theta with habituation to the stimulus. Animals explore stimulus.

[e] No change in theta elicited by auditory stimulus after injection of glucocorticoids.

[f] Theta during orienting response to stimulus; no theta if no orienting.

[g] Hippocampal EEG desynchronized by unfamiliar but not by familiar stimulus.

TABLE A10

Hippocampal EEG in response to cues used in appetitive and aversive tasks[a]

Theta (task)	Theta frequency (Hz)	LIA or SIA
Rat		
Pickenhain and Klingberg 1967 (jump avoid)		
Bremner 1968 (cued ESB)	6	
Albino and Caiger 1971 (ap)	6.5	
Whishaw 1972 (av)	5.9	
McGowan-Sass 1973 (SA lever press)	6.6 dorsal; 6.2 ventral	
Bennett 1975 (lever press)	6.6 CRF; 6.2 DRL	
Schwartzbaum 1975 (CER)	6	
Rabbit		
Sadowski and Longo 1962 (cued ring pull)		
Powell and Joseph 1974 (conditioned eye blink)		
Cat		
Grastyan *et al.* 1959 (ap)[b]	4-7	Grastyan *et al.* 1959 (ap)[b]
Radulovacki and Adey 1965 (cued start in Y maze)	4-5	Lissak and Grastyan 1960 (ap & av)[b]
Elazar and Adey 1967*b* (cued start in T maze)	5	Holmes and Beckman 1969 (go–no-go runway)[c]
Holmes and Beckman 1969 (go–no-go runway)[c]		Bennett 1970[b]
Bennett 1970[b]		Bennett and Gottfried 1970 (DRL lever press)
Bennett *et al.* 1973 (CRF & DRL lever press)	5.5	Grastyan and Vereczkei 1974 (ap)[b]
Grastyan and Vereczkei 1974 (ap)[b]	4-5	Hatfield 1975 (conditioned immobility)
Hatfield 1975 (CRF lever press)		
Dog		
Dalton and Black 1968 (CRF lever press and hold still)		Dalton and Black 1968 (CRF lever press and hold still)[e]
Ellison *et al.* (delayed lever press)		Lopes da Silva and Kamp 1969 (CRF)
Konorski *et al*. 1968 (ap & lever press)		
Dalton 1969 (op. reinforcement of theta)	4-6	Black *et al*. 1970 (operant reinforcement of theta and LIA)[d]
Black *et al*. 1970 (operant reinforcement of theta and LIA)[d]		Black and Young 1972a (conditioned immobility)
Black and Young 1972a (CRF lever press)	4.5-5	
Preobrazenskaya 1974 (av)	5-5.5	

a Abbreviations: ap (appetitive), av (aversive), ESB (electrical stimulation of the brain), SA (Sidman avoidance in which the animal must do something, such as press a lever, to delay the onset of shock), CER (conditioned emotional response), CRF (continuous reinforcement), DRL (differential reinforcement of low rates of lever pressing).

b LIA elicited by the cue in approach or avoidance tasks when no investigative orientation response occurs; theta when an orientation response does occur.

c One stimulus signals the onset of a go trial, another a no-go trial; theta during the first but not the second.

d Curarized dogs were operantly reinforced for producing theta in the presence of one stimulus and LIA in the presence of a second; in the unparalysed state the animals moved more during the first stimulus than during the second.

e Dogs were reinforced for lever pressing to one stimulus and for holding still to a second stimulus; the first stimulus elicited theta and the second LIA.

TABLE A11(a)

Hippocampal EEG during slow wave sleep

Theta	Behaviour and theta frequency	LIA or SIA
Rat		
		Morales *et al*. 1971 Olmstead *et al*. 1973 Winson 1974 Coenen 1975
Rabbit		
		Torii and Sugi 1960 Harper 1971
Cat		
		Brown and Shryne 1964 Karmos *et al*. 1965 Brown 1968 Glotzbach, 1975

TABLE A11 (b)

Hippocampal EEG during low-voltage fast sleep

Theta	Behaviour and theta frequency (Hz)	LIA or SIA
Rat		
Irmis *et al*. 1971	6-8	
Morales *et al*. 1971	5-8	
Kurtz and Adler 1973	6.5-9.5	
Sano *et al*. 1973	6.8-8.5	
Whishaw and Vanderwolf 1973	6.7 (7.6 during twitches)	
Winson 1974		
Coenen 1975	8	
Cat		
Jouvet and Mounier 1962	5	
Brown and Shryne 1964[a]	4.5-5.5	
Karmos *et al*. 1965		
Brown 1968	5	
Sakai *et al*. 1973	4.2–6.2	
Glotzbach 1975		
Kemp and Kaada 1975	7.5	
Rabbit		
Harper 1971	8	
Winson 1976 a, b		

[a] SIA, not theta, is seen for the first few episodes of lvf sleep in timid fearful cats in a new environment

TABLE A 12

Hippocampal EEG during electric stimulation of the brain[a]

Theta	Reinforcement	Brain area	LIA or SIA	Reinforcement	Brain area
Rat					
Ito 1966[b]	o	mrf, mh	Ito 1966[b]	+	lpo, lh, s
Yoshii *et al.* 1967	-	lh	Gray and Ball 1970	+	s
Routtenberg and Kramis 1968	-	dmt	Paxinos and Bindra 1970[d]	+	lh
Paxinos and Bindra 1970[d]	+	lh	Pond and Schwartzbaum 1970	+	s
Pond *et al.* 1970	+	lh			
Pond and Schwartzbaum 1970	+, -	lh, dmt	Gray *et al.*	-	s
Routtenberg 1970	+, -, *o*	mbs	Frederickson 1974b[c]	+	
Gray 1972		s			
Sinnamon and Schwartzbaum, 1973	+, -	lh, dmt			
Gerbil					
Kramis and Routtenberg 1969	+	ph			
Car					
Grastyan *et al.* 1966	+	h	Grastyan *et al.* 1966	-	h
Anchel and Lindsley 1972[e]		mh	Grastyan *et al.* 1968	+	h
Coleman and Lindsley 1975[e]		mh	Anchel and Lindsley 1972[f]		lh
			Coleman and Lindsley 1975[f]		lh
Rabbit					
Polc and Monnier 1970[b]		r, mrf			

[a] Abbreviations: + (positively reinforcing), - (negatively reinforcing), o (non-reinforcing), lh (lateral hypothalamus), mh (medial hypothalamus), ph (posterior hypothalamus), dmt (dorsal mid-brain tegmentum), mbs (mesencephalic brain stem), mrf (mesencephalic reticular formation), s (septum), h (hypothalamus), r(raphe).

[b] ESB elicits attentive sniffing and searching; ESB in lateral hypothalamus and lateral preoptic region elicits self-stimulation in 7 of 16 rats; ESB in septum elicits self-stimulation in 2 of 20 rats.

[c] ESB reinforced rat for holding still.

[d] Theta if ESB reinforces movement; LIA if ESB reinforces immobility.

[e] ESB elicited head turning; also produced by cold block of lateral hypothalamus (in Anchel and Lindsley 1971 only).

[f] ESB elicited arrest of ongoing activity attentive fixation of gaze; also produced by cold block of medial hypothalamic pathway (in Anchel and Lindsley 1972 only).

TABLE A13

Reactions to novel items after hippocampal lesions

	Normal	Decreased	Increased
NONCOMPETITIVE			
Rat	Kaplan 1968		Coover and Levine 1972
	Crowne and Riddell 1969		
	Hendrickson *et al.* 1969		
	Sanwald *et al.* 1970[a]		
	Kemble and Ison 1971		
	Dawson *et al.* 1973		
	Olton and Gage 1974		
	Groves *et al.* 1974[b]		
Cat	Rogozea and Ungher 1968	Rogozea and Ungher 1968	
	Ungher *et al.* 1971	Ungher *et al.* 1971	
	Nonneman and Kolb 1974		
Guinea pig	Ireland and Isaacson 1968		Ireland and Isaacson 1968
Gerbil		Glickman *et al.* 1970	
Monkey	Bagshaw *et al.* 1965		Hara and Myers 1973[c]
COMPETITIVE			
Rat		Wickelgren and Isaacson 1963	
		Raphelson *et al.* 1965[d]	
		Kaplan 1968	
		Hendrickson *et al.* 1969	
		Crowne and Riddell 1969	
		Cohen 1970	
		Cohen and Swenson 1970	
		Gustafson 1975	

a Normal cardiac deceleration and habituation of deceleration, but absence of late acceleration.
b Normal intrasession, but deficient intersession, habituation of acoustic startle response.
c Only a slight increase in some reactions.
d No group responded to the introduction of sandpaper floors.

TABLE A 14

Reactions to novel places after hippocampal lesions

	Normal	Increased		Decreased
Rat	Douglas and Isaacson 1964[a]	Kim 1960a	Moorcroft 1971	Jarrard 1968
	Spiegel *et al*. 1966	Niki 1962	Means *et al*. 1971	Jarrard and Bunnell 1968
	Hostetter and Thomas 1967	Roberts *et al*. 1962	Campbell *et al*. 1971	
	Haddad and Rabe 1967	Kimble 1963	Capobianco and Hamilton 1973	
	Boitano and Isaacson 1967	Teitelbaum and Milner 1963	Alvarez-Pelaez 1973	
	Jackson 1967	Douglas and Isaacson 1964	Dawson *et al*. 1973	
	Bender et al. 1968	Leaton 1965	Bayer *et al.* 1973	
	Kaplan 1968	Stein and Kimble 1966	Alexander *et al*. 1974	
	Boitano et al. 1968	Leaton 1967	Kearley *et al*. 1974	
	Rabe and Haddad 1969a	Jackson 1967	Lanier *et al*. 1974	
	Jarrard and Korn 1969	Kirkby *et al*. 1967	Lanier and Isaacson 1975	
	Strong and Jackson 1970[b]	Jarrard 1968	Murphy *et al*. 1975	
	Campbell *et al*. 1971	Jarrard and Bunnell 1968	Jacobs *et al.* 1975	
	Moorcroft 1971	Bermant *et al*. 1968	Myhrer and Kaada 1975	
	Alvarez-Pelaez 1973	Sengstake 1968	Myhrer 1975b	
	Greene and Stauff 1974	Kaplan 1968	Myhrer 1975a	
	Kearley *et al*. 1974	Nadel 1968	Peters and Brunner 1976	
	Lanier and Isaacson 1975	Gotsick 1969	Wallace *et al.* 1976	
	Lanier *et al*. 1975	Strong and Jackson 1970	Ely *et al.* 1976	
	Murphy *et al*. 1975	Clark 1970	Iuvone and Van Hartesveldt 1976	
	Myhrer and Kaada 1975	Kim *et al*. 1970		
	Myhrer 1975a	Winocur and Mills 1970b		
	Myhrer 1975b	Eichelman 1971		
Gerbil		Glickman *et al.* 1970		
Cat		Kim *et al*. 1971		
Hamster	Jarrard and Bunnell 1968			

a Increase with electrolytic, but not aspiration, lesions.

b Decrease in one study; no effect in another.

TABLE A 15

Spontaneous alternation after hippocampal lesions

Alternation			Random			Repetition		
	ITI	*confine*[a]		*ITI*	*confine*[a]		*ITI*	*confine*[a]
Kirkby *et al*. 1967	50 min	50 min	Roberts *et al*. 1962	10-480 s	0 s	Lash 1964[b]	5 min	0 s
Gross *et al*. 1968	0 s	0 s	Lash 1964[b]	5 min	0 s	Kirkby *et al*. 1967	50 s	50 s
Stevens 1973a	51 min	50 min	Douglas and Isaacson 1964			Dalland 1970	1 min	0 s
Stevens and Cowey 1973[c]	51 min	50 min	Gross *et al*. 1968	5-15 min	0 s	Greene 1971		
Myhrer 1975a[f]	1 min	0 s	Ellen and De Loache 1968[d]	1 min	15 s	Stevens 1973a	50 s	50 s
			Means *et al*. 1971	20 s	10 s	Stevens and Cowey 1973[c]	50 s	50 s
			Means *et al*. 1972	20 s	20 s	Stevens and Cowey 1973[e]	51 min	50 min
			Stevens 1973a	51 min	50 s	Dawson *et al*. 1973[g]	30 s	0 s
			Stevens and Cowey 1973	50 s	50 s	Dalland 1976		
			Bayer *et al*. 1973					
			Myhrer and Kaada 1975[h]	1 min	0 s			
			Myhrer 1975a[i]	1 min	0 s			
			Douglas 1975[j]					

a Inter-trial interval (ITI) and confinement time in arm chosen on first trial (confine). Time in seconds (s) or minutes (min). ITI includes confinement time.
b Repetition in two different shaped mazes; random choice in a third.
c Ventral hippocampal lesions.
d Random choice to spatial cues but alternation to brightness cues.
e Dorsal hippocampal lesions.
f CA1 or medial fimbrial lesions,
g 1 stage or seriatim lesions.
h Dorsal fornix lesions.
i Lateral or total fimbrial lesions.
j Random choice after bilateral lesions, one stage or two stage, adult or neonatal; some alternation with small electrolyte lesions.

TABLE A 16

Spatial discrimination after hippocampal lesions

Normal	Deficient
Kimble and Kimble 1965	Greene 1971
Niki 1966	Samuels 1972
Uretsky and McCleary 1969	Means *et al.* 1972
Brown *et al.* 1969	Jones and Mishkin 1972
Means and Douglas 1970[a]	Mikulka and Freeman 1975[b]
Mahut 1971	
Cohen *et al.* 1971	
Hirsch and Segal 1972	
Mahut 1972	
Cohen and LaRoche 1972	
Cohen and LaRoche 1973	
Mahut and Zola 1973	
Riddell *et al.* 1973	
Greene and Stauff 1974	
Thomas and McCleary 1974	

[a] A subgroup trained against preference had a deficit.

[b] Deficit with 10 s delay between response and reinforcement; normal without delay.

TABLE A 17

Non-spatial discrimination after hippocampal lesions

Normal	Deficient
Kimble 1963	Niki 1962
Kimble and Pribram 1963	Pribram *et al*. 1962
Teitelbaum 1964	Andy *et al*. 1967[a]
Webster and Voneida 1964	Douglas *et al*. 1969[b]
Douglas 1966	Duncan and Duncan 1971
Stein and Kimble 1966	Woodruff and Isaacson 1972
Kimble and Zack 1967	Woodruff *et al.* 1972[c]
Silveira and Kimble 1968	Olton 1972b[d]
Winocur and Salzen 1968	Bauer 1974[f]
Isaacson *et al.* 1968	
Truax and Thompson 1969	
Kimble and Kimble 1970	
Winocur and Mills 1970a	
Mahut 1971	
Jones and Mishkin 1972	
Samuels 1972	
Mahut 1972	
Mahut and Zola 1973	
Harley 1972	
Zola and Mahut 1973[e]	
Nonneman and Isaacson 1973	
Riddell *et al.* 1973	
Alexander *et al.* 1974	
Anton and Bennett 1974	
Stevens and Cowey 1974[g]	

a Normals also did not learn; hippocampals had position habit.
b Lesioned animals normal with one negative cue, but defective with two or four negative cues.
c Animals were pretrained on CRF; hippocampals had higher inter-trial response rate.
d Hippocampals had pronounced position habit.
e Hippocampals were facilitated.
f See p. 279 of text for discussion of this study.
g Slight deficit in compound cue discrimination.

TABLE A l8

Spatial discrimination reversals after hippocampal lesions

Normal	Deficient
Cohen *et al*. 1971	Thompson and Langer 1963
Stevens 1971[b]	Kimble and Kimble 1965
Cohen and LaRoche 1972	Thompson *et al*. 1964[a]
Cohen and LaRoche 1973	Niki 1966
Stevens 1973b[d]	Samuels and Valian 1968[c]
	Uretsky and McCleary 1969
	Brown *et al*. 1969
	Greene 1971
	Mahut 1971
	Cohen *et al*. 1971
	Hirsch and Segal 1972
	Samuels 1972
	Cohen and LaRoche 1972
	Gaffan 1972
	Mahut 1972
	Jones and Mishkin 1972
	Cohen and LaRoche 1973
	Mahut and Zola 1973
	Greene and Stauff 1974
	Nonneman *et al*. 1974
	Thomas and McCleary 1974

a 30 s inter-trial interval ameliorates deficit.
b Deficit with 4 min inter-trial interval and reversal to non-preferred side.
c Deficit is ameliorated by added visual cues.
d Massed trials improve reversal.

TABLE A19

Non-spatial discrimination reversals after hippocampal lesions

Normal	Deficient
Isaacson *et al*. 1968	Teitelbaum 1964
Mahut 1971	Webster and Voneida 1964
Mahut 1972	Douglas and Pribram 1966[a]
Jones and Mishkin 1972	Silveira and Kimble 1968
Mahut and Zola 1973	Isaacson *et al*. 1968
Zola and Mahut 1973[c]	Hsiao and Isaacson 1971
	Samuels 1972[b]
	Nonneman and Isaacson 1973[d]

a Mild deficit on 100 per cent vs. o per cent task; large deficit on 70 per cent vs. 30 per cent probability task.
b Small lesions yielded no deficit; larger lesions yielded deficit.
c Lesioned monkeys were better than normal at reversal.
d Neonatal lesions did not retard reversal.

TABLE A20

Complex maze learning after hippocampal lesions

Normal	Deficient
Gross *et al*. 1965	Thomas and Otis 1958b
Ellen and Bate 1970[b]	Kaada *et al*. 1961
	Kimble 1963
	Kveim *et al*. 1964
	Madsen and Kimble 1965
	Hughes 1965
	Stein and Kimble 1966
	Spiegel *et al*. 1966
	Niki 1966
	Jarrard and Lewis 1967
	Hostetter and Thomas 1967
	Bender *et al*. 1968
	Zack 1968
	Jackson and Strong 1969[a]
	Winocur and Breckenridge 1973[c]
	Myhrer and Kaada 1975
	Myhrer 1975a
	Myhrer 1975c

a See text, p. 289.
b Very small lesions.
c No deficit if cues added at choice points; see text p. 290.

TABLE A21

One-may active avoidance after hippocampal lesions

Normal	Deficient	Mixed
Niki 1962	McNew and Thompson 1966	Liss 1968[a]
Kirkby and Kimble 1968	Olton and Isaacson 1968a	Nadel 1968[b]
Winocur and Mills 1970a	Coscina and Lash 1969[c]	Olton and Isaacson 1969[d]
Thomas and McCleary 1974	De Castro and Hall 1974	
Haggbloom *et al*. 1974		
Klein *et al*. 1975		
Ross *et al*. 1975		
Myhrer 1975b[e]		

a Normal with 5 s inter-trial interval; deficit with to min inter-trial interval.
b Dorsal lesions yield deficit; ventral lesions do not.
c Aspiration lesions yield deficit; ventral electrolytic lesions do not.
d Lesioned rats affected by prior pairing of CS and US or by pseudo-pairing.
e Jump-avoidance task.

TABLE A22

Two-may active avoidance after hippocampal lesions

Normal	Facilitated	Deficient
Andy *et al*. 1967	Isaacson *et al*. 1961	Thomas and Otis 1958a
Schmaltz 1971	Green *et al*. 1967	Andy *et al*. 1968
Molino 1975[c]	Liss 1968	Eckersdorf *et al*. 1973
	Olton and Isaacson 1968b	Myhrer 1975a[b]
	Rabe and Haddad 1969a	Myhrer 1975b
	Ackil *et al*. 1969	
	Van Hoesen *et al*. 1969	
	Ireland *et al*. 1969	
	Papsdorf and Woodruff 1970	
	Lovely *et al*. 1971	
	Antelman and Brown 1972	
	Woodruff and Isaacson 1972	
	Van Hoesen *et al*. 1972	
	Alvarez-Pelaez 1973	
	Bayer *et al*. 1973	
	Ross *et al*. 1975	
	Myhrer 1975a[b]	
	Lovely 1975a	
	Ross and Grossman 1975	
	Wallace *et al*. 1976	

a Facilitation is removed by hypophysectomy.
b Deficit with medial fimbrial lesions; facilitation with lateral or total fimbrial lesions.
c Both neonatal and adult dorsal lesions had no effect; neonatal ventral lesions had no effect but adult ventral lesions yielded facilitation.

TABLE A23

Passive avoidance after hippocampal lesions

	Normal	Deficient	Mixed[e]
Step-down or step-through	Kimble *et al*. 1966 Nadel 1968 Riddell 1968[a] Winocur and Mills 1969 Blanchard, Blanchard and Fial 1970 Brunner *et al*. 1970 Riddell 1972 Dawson *et al*. 1973[h]	Glick *et al*. 1974	Boast *et al*. 1975f
Drinking	Kaada *et al*. 1962 Kveim *et al*. 1964 Boitano and Isaacson 1967 Boitano *et al*. 1968 Brunner and Rossi 1969 De Castro and Hall 1975 Ross *et al*. 1975 Myhrer 1975a Myhrer 1975b Myhrer and Kaada, 1975		
Running and drinking	Boitano and Isaacson 1967 Hostetter 1968 Van Hoesen *et al*. 1972	Riddell 1968 Wishart and Mogenson 1970 Best and Orr 1973 Bayer *et al*. 1973 Lanier *et al*. 1974	Kimura 1958[b] Isaacson and Wickelgren 1962 Snyder and Isaacson 1965 Kimble *et al*. 1966 Andy *et al*. 1967 Stein and Kirkby 1967[c] Van Hoesen *et al*. 1969 Fried 1970 Fried 1971 Fried 1972 Fried 1973[d] Nonneman and Isaacson 1973 Greene and Stauff 1974
Escape then step down		Teitelbaum and Milner 1963 Blanchard and Fial 1968 Blanchard, Blanchard, and Fial 1970	
Punished 1-way active or escape	Nadel 1968 Liss 1968 Coscina and Lash 1969 Thomas and McCleary 1974	Isaacson, Olton, Bauer and Swart 1966 Liss 1968 Coscina and Lash 1969 De Castro and Hall 1975	McNew and Thompson 1966
Taste aversion	McGowan *et al*. 1972 Murphy and Brown 1974 Nachman and Ashe 1974 De Castro and Balagura 1975 Miller *et al*. 1975 Thomka and Brown 1975	Miller *et al*. 1971	Best and Orr 1973[g]

a Two separate studies.
b Deficit with posterior lesions; normal with anterior lesions.
c Normal with 5 days training on running task; deficit with 10 days training.
d Deficit only with combined dorsal and ventral lesions.
e Deficit with one measure, typically latency; no deficit with another, typically shocks taken.
f Deficit at immediate retest only with fascia dentata lesions, at 24 h retest with all lesions.
h One subgroup had deficit.

TABLE A24

Effect of hippocampal lesions on lever-press rates in operant tasks

	Normal rates	Increased rates	Decreased rates
CRF	Schmaltz and Isaacson 1966a Swanson and Isaacson 1967 Schmaltz and Isaacson 1967 Means *et al*. 1970 Henke and Bunnell 1971 Van Hartesveldt 1973	Rabe and Haddad 1968[a] Haddad and Rabe 1969[a] Winocur and Mills 1970a	Clark and Isaacson 1965 Jackson and Gergen 1970
FR	Carey 1969 Van Hartesveldt 1973 Schmaltz *et al*. 1973[b]	Rabe and Haddad 1968	
FI, VI	Ellen and Powell 1962	Jarrard 1965 Beatty and Schwartzbaum 1968 Haddad and Rabe 1969a, b Jackson and Gergen 1970 Ross and Grossman 1975	
DRL	Gol *et al*. 1963 Ellen *et al*. 1964 Ellen and Aitken 1970 Pellegrino and Clapp 1971[e] Rickert *et al*. 1973[e] Ellen *et al*. 1973[g]	Clark and Isaacson 1965 Schmaltz and Isaacson 1966a Schmaltz and Isaacson 1966b Haddad and Rabe 1967 Schmaltz and Isaacson 1968 Isaacson and Schmaltz 1968 MacDougall *et al*. 1969[d] Carey 1969 Nonneman and Isaacson 1973 Schmaltz *et al*. 1973[f] Riddell, Malinchoc, and Reimers 1973 Nonneman *et al*. 1974 Kearley *et al*. 1974 Ross and Grossman 1975	

a Increases with total lesions; normal with anterior lesions only.
b Some increases on FR80, FR160.
c Increased rate on the DRL task indicates a deficit.
d Total fornix lesion yields deficit; medial fornix lesion alone does not.
e Normal with cue; increased rate and deficit without cue.
f Deficit unassociated with increased rate.
g Deficit only with total dorsal plus ventral lesion and extensive pretraining on CRF.

TABLE A25

Delayed response, alternation, and go–no-go after hippocampal lesions

	Normal	Deficit	Facilitated
Delayed response	Mahut 1971	Mishkin 1954 Mishkin and Pribram 1954 Orbach *et al.* 1960 Niki 1962 Karmos and Grastyan 1962 Ungher and Sirian 1970	
Spatial alternation-maze		Racine and Kimble 1965 Means *et al.* 1971 Greene 1971 Greene *et al.* 1972 Greene and Stauff 1974	
Spatial alternation-operant chamber	Brown *et al.* 1969 Waxler and Rosvold 1970[a] Mahut 1971 Stevens and Cowey 1972 Stevens and Cowey 1973[b]	Pribram *et al.* 1962 Rosvold *et al.* 1964 Niki 1966 Correll and Scoville 1967 Riddell, Malinchoc, and Reimers 1973	
Go–no-go alley	Mering and Mukhin 1973[c] Brunner *et al.* 1974	Franchina and Brown 1970	
Go–no-go operant chamber	Gaffan 1973 Freeman *et al.* 1973[d] Freeman and Kramarcy 1974	Niki 1965 Swanson and Isaacson 1967 Schmaltz *et al.* 1973 Woodruff *et al.* 1973[e] Buerger 1970	
Go–no-go alternation-operant chamber		Warburton 1969 Walker *et al.* 1972[f] Walker and Means 1973 White 1974	Means *et al.* 1970 Walker, Means, and Isaacson 1970

a Some hippocampals normal; some deficient.
b Without cue: dorsals facilitated, ventrals normal. With cue: dorsals facilitated, ventrals dificient.
c Deficit with temporal cue only.
d Normal when tone is S+; deficit when tone is S-.
e No deficit relative to cortical controls.
f Facilitated with 10s ITI; normal with 20s ITI; deficient with 40, 80 s ITI.

TABLE A26

Extinction after hippocampal lesions

Normal	Deficient
Niki 1962 (one-way active avoidance)	Isaacson *et al.* 1961 (two-way active avoidance)
Schmaltz and Isaacson 1967b (CRF-lever press)	Webster and Voneida 1964 (non-spatial discrimination)
Nadel 1968 (CER)	Jarrard, Isaacson, and Wickelgren 1964[a] (runway)
Kaplan 1968 (CER)	Jarrard and Isaacson 1965 (runway)
Ackil *et al.* 1969[c] (two-way active avoidance)	Niki 1965 (CRF-lever press)
Gaffan 1972[e] (nose poke)	Peretz 1965 (WGTA)
Schmaltz and Theios 1972[f] (classical conditioning)	Douglas and Pribram 1966 (discrimination)
Nonneman *et al.* 1974 (DRL-lever press)	Raphelson *et al.* 1966 (runway)
Thomas and McCleary 1974 (one-way active avoidance)	Jarrard and Lewis 1967 (maze)
Murphy and Brown 1974 (taste aversion)	Isaacson *et al.* 1968 (runway)
Kearley *et al.* 1974 (DRL-lever press)	Rabe and Haddad 1968 (FR-lever press)
Ross *et al.* 1975 (runway-lever press)	Brown *et al.* 1969 (modified WGTA)
	Winocur and Mills 1969 (runway)
	Kimble 1969 (Y-maze non-spatial discrimination)
	Cohen 1970 (runway)
	Franchina and Brown 1970 (runway)
	Kimble and Kimble 1970 (Y-maze non-spatial discrimination)
	Greene 1971 (T-maze discrimination)
	Coover *et al.* 1971b (CRF-lever press)
	Lovely *et al.* 1971[d] (two-way active avoidance)
	Henke and Bunnell 1971 (CRF-lever press)
	Gaffan 1972[e] (runway)
	Fried 1972 (runway)
	Warburton 1972 (lever press)
	Amsel *et al.* 1973 (FR-lever press)
	Brunner *et al.* 1974 (runway)

a Deficit with spaced trials; normal with massed trials.
b Deficit on first day of first extinction; normal for remainder of this and subsequent extinctions.
c Deficit if there is adaptation to CS prior to training.
d No deficit in terms of responses made after first non-response on two-way active avoidance task.
e Deficit on alley; normal on operant task.
f Normal on first extinction; deficit on subsequent extinctions.

TABLE A27

Species-typical behaviour after hippocampal lesions

	Normal	Increased	Decreased
Eat or drink	Kim 1960a Niki 1962 Jarrard 1965 Haddad and Rabe 1967 Boitano *et al.* 1968 Beatty and Schwartzbaum 1968 Gotsick 1969 Haddad and Rabe 1969 Murphy and Brown 1970[a] Glickman *et al.* 1970[b] Holdstock 1972[c] Boitano *et al.* 1973 Donovick and Burright 1973 Brown and Murphy 1973 Ross *et al.* 1975 Murphy *et al.* 1975[d] Thomka *et al.* 1975	Kimble and Coover 1966	
Maternal, sexual, social	Kling 1964 Kimble *et al.* 1967[e] Bermant *et al.* 1968[f] Glickman *et al.* 1970[h] Capobianco and Hamilton 1973	Kim 1960b	Kim 1960a Peretz 1967 Kimble *et al.* 1967[e] Dewsbury *et al.* 1968[g] Michal 1973 Kolb and Nonneman 1974 Nonneman and Kolb 1974 Ely *et al.* 1976

a Increased intake of glucose + saccharine, salt water after formalin pretreat.
b Some increase after ventral lesions in long sessions.
c Hippocampal females drink more often when food is available.
d Normal on post-operative days 6-10 ; increase on days 18-22.
e Normal sexual; decreased maternal.
f Only dorsal lesions, not ventral nor dorsal + ventral, increase sex.
g Only dorsal + ventral lesions, not dorsal, increase latency to first copulation.
h Some decrease in shredding

TABLE A28

List of hippocampal steroid studies

I. Uptake and unit studies

McEwen *et al*. 1969
McEwen *et al*. 1970
McEwen and Pfaff 1970
McEwen *et al*. 1970a
McEwen *et al*. 1970b
Kawakami and Kubo 1971
Pfaff *et al*. 1971
Ford *et al*. 1971
Stumpf and Sar 1971
Gerlach and McEwen 1972
Knizley 1972
Dafny *et al.* 1973
Stevens *et al*. 1973
Luttge *et al*. 1973
McEwen and Wallach 1973
Luttge *et al*. 1974
McEwen *et al.* 1974
Michal 1974
Warembourg 1975

II. Resting levels

Mason 1958
Mason 1959
Endroczi and Lissak 1960
Knigge 1961
Slusher and Hyde 1961
Endroczi and Lissak 1962
Mandell *et al.* 1963
Nakadate and de Groot 1963
Galicich *et al*. 1965
Slusher 1966
Rubin *et al*. 1966
Fendler *et al*. 1961
Bohus *et al*. 1968
Kawakami *et al*. 1968
Kawakami, Seto and Yoshida 1968
Van Wimersma Griedanus and de Wied 1969
Endroczi and Nyakas 1971
Moberg *et al*. 1971
Coover *et al*. 1971b
Endroczi 1972
Kawakami *et al*. 1973
Lengvari and Halasz 1973
Wilson and Critchlow 1973/1974
Kearley *et al*. 1974
Jackson and Regenstein 1974
Lanier *et al.* 1975

III. Behavioural studies

Porter 1954
Endroczi *et al*. 1959
Knigge 1961
Kim and Kim 1961
Endroczi and Lissak 1962
Knigge and Hays 1963
Davidson and Feldman 1967
Coover *et al*. 19716
Bohus 1971
Dupont *et al*. 1972
Endroczi 1972
Wilson and Critchlow 1973/1974
Kearley *et al*. 1974
Lovely 1975
Iuvone and Van Hartesveldt 1976

TABLE A29

List of hippocampal stimulation studies

I. General effects

Kaada 1951
Kaada *et al*. 1953
MacLean 1957a
MacLean 1957b
Fisher and Coury 1962
Andy *et al*. 1962
Votaw and Lauer 1963b
Ursin *et al*. 1966
Feldman *et al*. 1967
Coury 1967
Musty *et al*. 1967
Grant and Jarrard 1968
Siegel and Flynn 1968
Rogozea *et al*. 1969
Mountford 1969
Milgram 1969a
Milgram 1969b
Gumulka *et al*. 1970
Kaada *et al*. 1971
Rogozea *et al*. 1971
Bland and Vanderwolf 1972b
Levitt and O'Hearn 1972
Brown and Winocur 1973
Oliver *et al*. 1974
Jackson and Gardner 1974
Nagy and Decsi 1974
Lico *et al*. 1974
Huston *et al*. 1974
Siegfried *et al*. 1975
Milgram *et al*. 1975

II. Effects on performance

Bures *et al*. 1960
Weiskrantz *et al*. 1962
Stein 1965
Erickson and Chalmers 1966
Andy *et al*. 1968
Szekely *et al*. 1968a
Szekely *et al*. 1968b
Margules and Stein 1968
Leaton 1968
Vanegas and Flynn 1968
Siegel and Flynn 1968
Vergnes and Karli 1969
Van Abeelen *et al*. 1972
Oliver *et al*. 1973
Overstreet 1974
Ross and Grossman 1974

III. Effects on learning

Correll 1957
Olds and Olds 1961
Weiskrantz *et al*. 1962
Rabe 1963
Grossman and Mountford 1964
Hirano 1966
Kesner and Doty 1968
Avis and Carlton 1968
Wyers *et al*. 1968
Stein and Chorover 1968
Grossman 1969
Brunner and Rossi 1969
Erickson and Patel 1969
Hughes 1969
Psatta *et al*. 1970
Greene and Lomax 1970
Greene 1971
Nakajima 1972
Wilson and Vardaris 1972
Kesner and Conner 1972
Shinkman and Kaufman 1972a
Shinkman and Kaufman 19726
Bresnahan and Routtenberg 1972
Livesey and Wearne 1973
Weiss and Hertzler 1973
Henderson *et al*. 1973
Ott and Matthies 1973
Zornetzer and Chronister 1973
Zornetzer *et al.* 1973
Landfield *et al.* 1973
Haycock *et al*. 1973
Wiener and Messer 1973

TABLE A29-*cont.*

List of hippocampal stimulation studies

III. Effects on learning-cont.

Brunner *et al*. 1970
Lidsky and Slotnick 1970
Barcik 1970
Nyakas and Endroczi 1970
Vardaris and Schwartz 1971
Kapp and Schneider 1971
Shinkman and Kaufman 1970
McDonough and Kesner 1971
Singh *et al*. 1974
George and Mellanby 1974
Kesner and Conner 1974
Destrade and Cardo 1974
Sideroff *et al*. 1974
Kapp *et al*. 1974
Leith and Barrett 1975
Livesey and Meyer 1975
Soumireu-Mourat *et al*. 1975
Gustafson *et al*. 1975
Kesner *et al*. 1975
Livesey and Bayliss 1975
Daniels 1971
Schmaltz 1971
Serota 1971
Whishaw and Deatherage 1971

TABLE A30

Retention after hippocampal lesions

Normal	Deficient
Mishkin 1954[f] (delayed response, colour and brightness)	Kimura 1958[a] (passive avoidance)
Orbach *et al*. 1960[f] (visual discrimination)	Niki 1962 (maze, brightness discrimination)
Niki 1962 (one-way active avoidance)	Pribram *et al*. 1962 (delayed alternation)
Kimble and Pribram 1963[g] (pattern discrimination)	Raphelson *et al*. 1965 (runway)
Schwartzbaum *et al*. 1964 (go-no-go)	Isaacson *et al*. 1966 (successive discrimination)
Correll and Scoville 1965 (matching to sample)	Olton and Isaacson 1968a (one-way active avoidance)
Breen and Thompson 1966 (two-way approach task)	Thomas 1971 (maze)
Raphelson *et al*. 1966 (runway)	Wild and Blampied 1972 (go–no-go)
Olton and Isaacson 1968a (two-way active avoidance)	Riddell *et al*. 1973 (position reversal learning set)
Winocur and Salzen 1968 (visual size discrimination)	Eckersdorf *et al*. 1973[e] (two-way avoidance)
Truax and Thompson 1969 (brightness discrimination)	Thompson 1974 (maze)
Uretsky and McCleary 1969[d] (one-way active avoidance)	Jarrard 1975 (Y-maze spatial alternation)
Winocur and Mills 1970a (brightness discrimination)	
Buerger 1970 (go–no-go)	
Schmaltz 1971 (two-way active avoidance)	
Schmaltz and Giulian 1972 (lever-press avoidance)	
Fried 1973[d] (passive avoidance)	
Glick and Greenstein 1973[c] (passive avoidance)	
De Castro and Marrone 1974 (shock-induced fighting)	

a Deficit with posterior lesions; normal with anterior lesions.
b Normal with massed trials; deficit with spaced trials, except with ventral lesions.
c Deficit when lesion made just after training, but not if made 1 h later.
d Deficit if lesion made 8 h after training, but not at 3 days.
e Deficit if trained to low criterion only.
f Mild deficit only on retention of pattern discrimination.
g Mild deficit on self-ordered sequential task only.

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Subject index

The letters f, n, and t carry the meanings figure, footnote, and table, respectively.

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