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THE CELL ASSEMBLY: MARK II[1]

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The purpose of this paper, as its title implies, is to suggest a revision of the neural construct that Hebb (5) has called a "cell assembly." The cell assembly plays a fundamental role in Hebb's behavior system, especially in his admirable treatment of perceptual learning, but the proposed changes are not intended to affect these psychological implications of the construct; they are aimed, rather, at making the underlying neural processes more explicit. As Hebb has mentioned (8), he tried to limit himself to using only known, or highly probable, properties of neurons in setting up his model; but it is clear that these soon proved to be inadequate, and he then had to introduce *ad hoc* mechanisms to explain how such artificially simplified elements could combine to produce the observed molar behavior. We can now see that from many standpoints the theory would have been better if the additional postulates had been introduced at the neural level, where they could have been more easily evaluated and tested; but on the other hand, such a theory might never have been given a hearing.

There is little doubt that flaws exist in the arguments Hebb uses to derive the molar properties of his construct. As will be shown below, if we try to operate with only those properties and connections of individual neurons that Hebb considered respectable, the behavior of the resulting network is incompatible with that required for the cell assembly. Some of the inconsistencies can be eliminated simply by taking into account neurophysiological discoveries made during the last ten years, but in order to derive a system having anything like the characteristics required for the cell assembly it will be necessary also to invoke a number of purely hypothetical neural processes.

As far as can be ascertained from *The Organization of Behavior* (5), only four factors are considered to determine whether a cortical neuron will fire: (*a*) the number of impulses bombarding the neuron from all sources during the few milliseconds in which temporal summation is assumed to take place; (*b*) the "strength" of the synapses concerned (the strength of a synapse may increase beyond its native value in accordance with the provisions of a learning postulate); (*c*) whether the neuron is refractory; and (*d*) by implication at least, the effect of neural fatigue. In the network that Hebb postulated as a model of the cortex, neurons with the above-mentioned properties are randomly interconnected, each neuron having efferent and afferent connections with many others. Some of the neurons can be fired by sensory input.

Such a network is somewhat analogous to the nuclear fission reactor. In that machine a neutron may either induce a fission, in which case it releases several new neutrons, or it may be absorbed or lost without releasing any other neutrons. If the neutron flux in the machine is to remain constant, all the neutrons released at a fission must

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be lost except one. For stable operation, in other words, the multiplication factor (the average number of fissions produced by the neutrons released at a previous fission) must be one. Likewise, in the cortical network, a fired neuron may fire several others, or it may send impulses to refractory, fatigued, or inadequately facilitated neurons and produce no further firing. If the average neuron fires one other neuron, the total activity in the network will remain constant.

The analogy is not quite complete, however; in the fission reactor the multiplication factor is slightly reduced by an increase in the neutron flux, but in the cortical network just the opposite is true, the multiplication factor increasing with total neural activity. This results from the effect of summation at synapses. When a neuron is firing by itself, the probability of its firing another is small; but when many neurons are firing at the same time, they provide a facilitatory background for each other, and one of them may then be able to fire several neurons to which it sends efferents. Thus, the number of neurons fired by an average neuron will increase in proportion to the total number of neurons firing, a state of affairs which precludes stable operation. In the nuclear reactor, if the multiplication factor should fluctuate to a value of 1.001, a few more neutrons will be released than are lost, and the total flux will increase; at this higher flux the multiplication factor will be reduced again to one. If the same fluctuation should occur in the cortical network, a few more impulses will circulate, the general level of facilitation will rise, and the multiplication factor will increase still further. This increase will only be halted when all the neurons in the system are firing as fast as their refractory periods will allow. Unlike the nuclear pile, therefore, the cortical network has only two levels of stable operation, "full on" and "full off." Neither of these states fulfills the requirements of Hebb's cell assembly. It is hardly necessary to add that the phase sequence, which is essentially a chain of associated cell assemblies representing a "train of ideas," bears as tenuous a relationship to the basic neural axioms as does the cell assembly itself.

The fact that Hebb's constructs are not rigorously derived is not as serious for the over-all theory as it might seem, because many of the more molar physiological principles that Hebb introduces are valid and important for the understanding of psychological phenomena, irrespective of the exact neural mechanisms involved. Nevertheless, the possibility of bridging the gap between the physiological and the psychological levels with a more substantial structure presents a fascinating challenge, especially in view of the improvements made to the supports on the physiological side since *The Organization of Behavior* was written.

A New Physiological Interpretation of the Cell Assembly

In what follows it will be recognized that we are adopting Hebb's descriptions of the cell assembly and the phase sequence as far as their psychological properties are concerned, but instead of trying to reconcile these to a minimum of oversimplified neural data, further postulates will be introduced in an attempt to provide an explicit relationship between the constructs and their elements.

The first additional postulate concerns inhibition. At the time when Hebb was developing his theory, many physiologists were strongly opposed to the idea of neural inhibition, largely because it was difficult to fit into the electrical theory of synaptic transmission. There was a feeling that all inhibitory phe-

nomena could be explained by lack of facilitation, or by invoking the principle of refractoriness, and Hebb seems to have accepted this view. Since then, the work of Eccles (3) and others has provided a better understanding of synaptic transmission, and inhibitory transmission is now generally accepted.

The processes underlying learning have still not been revealed by the neurophysiologists, and to fill this lacuna there seems to be nothing against retaining the postulate suggested by Hebb: "When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased" (5, p. 62).

Another assumption that we shall make here is that the cell assembly involves mainly cortical neurons. It is to be understood, however, that connections between cortical cells may often take place via cells in the thalamus, or other subcortical nuclei. In the model to be described, the fine-structure of the network has to be taken into account; Lorente de Nó's beautiful diagrams of the cortex (11) are invaluable for this purpose. They reveal a complex vertical organization amongst the different layers, including one conspicuous circuit to which Lorente himself draws attention. This is the return of collaterals from the long descending axons of some cells back to the region of the cell body, where they appear to end on Golgi type II, or similar short-axon cells. The Golgi cell axons proliferate in baskets around the bodies of the long-axon cells ". . . so that the discharge of the cell with a short axon results in powerful, practically simultaneous stimulation of a large number of other cells" (11, p. 303). It might appear from this description that the feedback circuit is admirably suited to produce uncontrolled

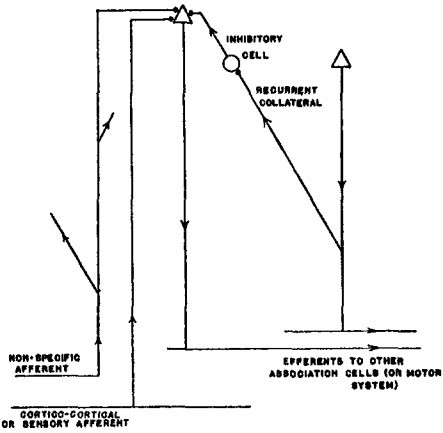

FIG. 1. Diagram of the postulated connections of cortical neurons.

epileptic discharge in the cortex, but recent microelectrode work by Eccles, Fatt, and Landgren (4) indicates that in the spinal cord, where a similar organization of long and short-axon cells is to be found, the short-axon cells inhibit the larger ones. It is likely that this is also true in the cortex.

The following highly simplified organization will therefore be assumed for the cortex (Fig. 1). Only two types of neuron are considered, those with long axons (having excitatory connections with other parts of the cortex or with the motor system), and those with short axons (which have local inhibitory connections). Those of the short-axon type receive impulses from the long-axon cells in their vicinity via recurrent collaterals, which leave the main shaft of the axon near the inner layer of the cortex and return toward the surface. Apart from those in the sensory cortex, which receive additional afferents from the sensory pathways, the long-axon cells receive their input from two major sources: (*a*) from other cortical cells, either directly via cortico-cortical association fibers, or indirectly by way of distributing cells in the association nuclei of the thalamus; and (*b*) from the nonspecific

projection system, which will here be regarded as a purely excitatory arousal system. This second source of afferent stimulation must play an important role in motivation, and it will be discussed in that connection later; for the moment, it need only be noted that we postulate that the facilitatory bombardment it provides is necessary for the conduction of impulses within the cortico-cortical network.

It appears from Lorente's diagrams (11, pp. 296, 298) that the long axons from the association cells branch and travel through the white matter to a number of different parts of the cortex. Each of these branches divides again as it ascends through the layers of the cortex to synapse with other long-axon cells. With this in mind, we can now proceed to analyze the activity in this network in the way that we previously analyzed that of Hebb's model. We shall start by assuming that no cortical cells are firing, but that there is a sufficient facilitation from the nonspecific projection system to make transmission between cortical cells possible. Suppose now that one cortical cell is somehow fired, and that it has effective connections to several other long-axon cortical cells (ten, let us say). These ten long-axon cells will in turn fire about a hundred others, and so on. At this rate the activity would soon snowball to an astronomical value, so that it clearly cannot continue to multiply for very long; in fact, the spread will quickly be checked by the firing of inhibitory cells. As the density of firing cells increases, impulses from many sources will arrive almost simultaneously in any small region of the cortex. When a long-axon cell is fired by one of the impulses, it will fire the short-axon cells in its vicinity and so prevent many subsequently arriving impulses from firing other neighboring cells. Thus, many of the impulses circulating in the cortex will be lost because they arrive in the regions of strong inhibition surrounding recently fired cells. Finally, a level of activity will be reached at which only ten per cent of the impulses are able to fire cells, so that in the example given (in which one cell could fire ten others in the absence of inhibitory influences) a state of equilibrium will be reached. If more long-axon cells start to fire, the regions of inhibition will be crowded together and less than one tenth of the circulating impulses will fire new cells; the activity must therefore drop back to the equilibrium value. If, on the other hand, too few cells are fired, the multiplication factor will increase above unity and the total activity will increase again. It is true that many factors have been neglected in this analysis; some of them are important and will be considered in a moment; other difficulties (such as the fact that when the level of neural activity rises, the increase in inhibition will be offset to an indeterminate extent by increased temporal and spatial summation) cannot be dealt with until better quantitative data are at hand. In the meantime we can only postulate that the parameters involved have values within the limits necessary to allow the network to behave in the manner described.

For reasons that will be clear later, it is desirable to construct the model in such a way that the equilibrium activity continues to circulate within a single group of cortical cells, instead of progressing continuously to new cells at each step in the firing. To this end, the following postulate will be made regarding the anatomical organization: the short-axon inhibitory cells which receive recurrent collaterals from a long-axon cell have fewer inhibitory connections to that particular long-axon cell than they do to other long-axon cells in the region. This is illustrated in an exaggerated way in Fig. 2, where it is seen

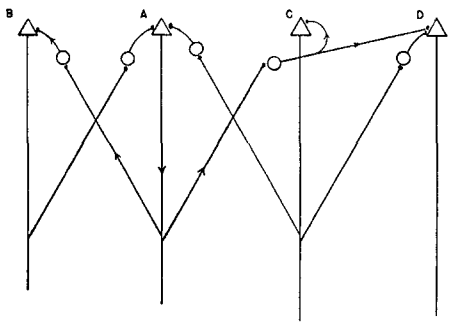

FIG. 2. Types of recurrent inhibitory connections.

that when Cell A is firing it causes the inhibition of its neighbors, B, C, and D, but is not itself inhibited. In fact, because the surrounding long-axon cells cannot now be fired, there is no way in which the short-axon cells discharging on to A can be fired. Therefore, as long as A continues to fire, it protects itself from being inhibited. This is not the only mechanism—perhaps not even the most likely mechanism—by which the desired result could be achieved (repetitive firing during the hypersensitive phase of the recovery cycle would also tend to confine the activity to the group of neurons that were already firing), but it is a useful hypothesis on which to base subsequent discussion.

Let us now see how this postulate will affect the equilibrium activity that can be sustained in our model of the cortex. There is a good chance that the cell which originated the activity will receive an afferent impulse from some other cell which is fired later in the avalanche. If it does, there will be nothing to prevent its firing again in response to the returning impulse, because its original discharge will have inhibited neighboring cells, thus greatly reducing the probability that it will be inhibited itself. When it fires again it will also be able to re-excite the ten cells that it fired previously, because they will have likewise remained free from inhibition. Thus the whole group will tend to re-excite itself, confining the activity to those neurons in which it first spread. If by some chance the original cell does not receive connections from any of the cells subsequently fired, some of the next ten cells, or those further along in the chain of activity, will do so, and the final result will be much the same.

It now remains to see what prevents the activity from persisting in this group of cells indefinitely. A property of most cells in the nervous system is that if they are subjected to a constant source of excitation their frequency of discharge steadily falls. We may therefore expect a decline in the intensity of firing in the group. As each cell adapts, it will deliver fewer impulses to other cells in the group, and reduced bombardment will combine with the adaptation to slow down the activity. On the other hand, there will be a concomitant decrease of inhibitory firing, so that the over-all activity will not decline as rapidly as it otherwise would. However, there is a limit to the compensation that can be provided in this way, because when the inhibition becomes too small some of the cells outside the group, which were previously prevented from firing, will be released. The firing of these cells will then bring in fresh short-axon cells which will immediately inhibit any fatigued cells in the vicinity. Thereupon, the balance of excitation and inhibition will be disturbed in regions to which the affected long-axon cells send impulses. If most of the cells in the group were already becoming adapted, or fatigued, the disturbance will snowball in reverse; each cell that stops firing causing connected cells to stop also. In this way the firing of one group might come to an abrupt end, and another group of cells take over. Note that although the activity will swing from one group of cells to another, it

can never die out; long before that happened the level of inhibition would become so low that all the unfatigued cells would be able to fire. The self-maintained cortical activity described above is, as the reader has no doubt realized, intended to form the basis of the revised version of the cell assembly.

One behavioral phenomenon which Hebb explains in terms of the cell assembly is "set." In order to do the same with the present model we shall have to add a further neural postulate. Perhaps the simplest type of behavior exemplifying "set" is that in which a stimulus, A, followed by another stimulus, B, gives rise to the response, C, although neither A nor B alone will produce the response. Presentation of A induces a set to respond to B. In neural terms, it seems that after a cell assembly has ceased to exert a direct control on an animal's behavior, it continues to influence the way in which that behavior develops, perhaps for a period of minutes. Only two categories of neural process seem able to account for this long-lasting aftereffect of a cell assembly; either some neurons of the group representing Stimulus A, for example, continue to fire, though the main body of the activity is now taking place in cells of the group representing Stimulus B (this is the "active" trace hypothesis), or the activity A leaves behind it physical or chemical changes which last long enough to influence subsequent firing patterns (the "latent" trace hypothesis). Hebb attempts to explain "set" in terms of the first of these alternatives—the active trace hypothesis—and the same process might be applicable to the present model. However, the second mechanism seems to offer much more interesting possibilities, which we shall now proceed to explore. The hypothesis might be formally stated as follows: If afferent impulses fall on a cortical cell but do not fire it (because of inhibition or lack of adequate background facilitation), they nevertheless leave the cell with a lowered threshold, the effect dying away with a time constant of many seconds. This influence of one cortical neuron on another will be called "priming." This postulate complicates the original model slightly because now, at the moments of transition from one pattern of cortical activity to another, we have to take account of the residual priming left by earlier activities, as well as of the momentary excitations. For example, a neuron that receives synaptic connections from neurons of several previously active groups will have a better chance of firing than a neighboring neuron that has been stimulated only by neurons of the most recently active group.

We must now examine the effect of sensory afferents on this postulated cortical system. The sensory projection areas of the cortex are regarded for this purpose as the final distribution centers for sensory impulses. The long-axon cells with specific-sensory afferent connections are assumed to have widespread efferent connections to the rest of the cortex, so that a change in the pattern of sensory input will result in multiple changes of the local fields of excitation there. If the cortex is already intensely active, and if the sensory change is a slight one, the added excitation will be unable to affect the ongoing pattern of cortical firing. The cells which are not already firing will be too strongly inhibited by those that are firing to be influenced by the input. Behaviorally, this means that the animal will ignore the stimulus and pursue its former course. On the other hand, if the stimulus is a strong one, or if the cortex is relatively inactive when it is presented, the sensory impulses will determine, or significantly influence, the pattern of the next activity in the cortex.

The learning mechanism can now be

made explicit. It has been postulated that if a neuron helps to fire another neuron on several occasions, it becomes a more potent agent for firing that neuron subsequently. This clearly implies that the neurons of a group that fire one another, in the way that has been described, will tend to bind themselves together more closely. Let us assume that one group has been activated on several occasions, and that consequently each neuron in the group has strengthened its synaptic connections with one or more other cells in the group, but not with any of the nonfiring cells to which its connections were initially equally strong. At this stage, if a neuron in the group is fired, the likelihood of its firing other cells of the group will have been increased; and the more cells of the group that are so aroused, the more the remaining members will be subjected to preferential excitation. This selective effect will often cause the total group activity to be established, starting from the firing of only a fraction of the constituent neurons. Moreover, each time this concerted firing takes place, the connections will be further strengthened, and the probability of the group's firing as a unit will become still greater. This process is equivalent to Hebb's mechanism for building up a cell assembly by repeated presentation of a "perceptual element"; its significance for the explanation of perceptual learning and concept formation has been discussed in some detail by Hebb (5, 6) and need not be repeated here. It must not be supposed, however, that after a stimulus has been presented often enough it will become able to determine the total pattern of the assembly; there will always be an inconstant "fringe" of cells that fire because of some previous cortical activity, metabolic conditions, or other factors independent of the stimulus. This fringe plays an important role in the association of one cell assembly with another, as we shall now see.

Associative learning was mentioned earlier as being the linking together of cell assemblies into phase sequences. This will usually occur because the assemblies in question have been aroused contiguously. It has been assumed up to now that the neurons that initiate an assembly activity are themselves fired by a sensory event, but clearly once a group of cells has strengthened its interconnections sufficiently, it does not matter which of its elements are fired first; the activity will in any case spread to the rest. If enough cells of one assembly are left in a primed or excited state by firing in other cell assemblies, that assembly may burst into activity when a previous one fades out. The question we have to answer is how such an interrelationship between two assemblies could be established by experience.

Let us suppose that two stimuli, giving rise to Cell Assemblies A and B, respectively, are presented in succession a number of times. Under these conditions some of the fringe neurons of Assembly B will be determined by A, and so will not be random any more. In other words, some of the cells primed by A will be active with Assembly B on every occasion that the stimulus pair is presented, and these cells will start to acquire stronger connections with the cells of the B assembly. Before long, in fact, they will become members of the assembly. When this has happened it will be possible for these cells, which on the first presentation of the stimulus pair were merely a part of the random fringe of B, to arouse the whole B cell assembly in the same way that any other fractional firing of the assembly would arouse it. It remains only to point out that these particular cells are the ones that are so strongly primed by the activity of the A assembly that they have a high probability of firing when

it stops (which is how they became part of the firing of B in the first place), and the method by which two assemblies become associated will be evident. Of the cells released from inhibition when Assembly A dies away, those that have on previous occasions fired with (and thus acquired strong connections with) cells of the B assembly, will be able to recruit the rest of the cells of that assembly. On the other hand, the successors of Assembly A which are incompatible with firing in the B assembly will have no strengthened connections, and will be promptly suppressed as the rest of the B assembly begins to fire.

It may be observed that the amount of perceptual "overlearning" that has gone into the development of the assembly, before its association with another, will make a great deal of difference to the ease with which the linking is achieved. If the total assembly action can spring from the firing of, say, five per cent of its constituent cells, links will have to be strengthened to a smaller number of cells than would be necessary if twenty per cent of the cells had to be fired to insure assembly action. That is, if the intragroup connections are weak to start with, the new cells will have to acquire stronger and more numerous connections to the rest of the group in order to compensate for the deficiency. Another point to be remembered is that only rarely will a single cell assembly be able to fire another one unaided, as in the example given. In most cases, the assembly aroused by association will have been primed by many previously active assemblies before it builds up enough sensitized cells to enable it to fire without its proper sensory input. This is an explanation of the importance of context, and set, for association; the arousal of a particular association may depend on the surroundings being similar to those in which the original learning took place, because the firing of a number of cell assemblies by the environmental stimuli is necessary to build up priming in the required group.

The model described above represents a brain mechanism capable of associating contiguously presented stimuli. This is only part of the apparatus needed to explain an animal's behavior in a learning situation, but it is an essential part. The most important other mechanism has to do with motivation. In this paper motivation will be considered to influence learned behavior in two ways: (*a*) by affecting the rate of learning, and (*b*) by helping to evoke responses.

In order to discuss motivation from a physiological standpoint, we must return to the system which was mentioned briefly before, the nonspecific projection system. It will be remembered that our model of the functional organization of the cortex was developed on the assumption of a fixed level of facilitatory bombardment from this system, such that one long-axon cortical cell was able to fire ten others in the absence of inhibition. If in the illustration we had assumed a lower level of nonspecific activity, the total cortical firing would have been less, though not proportionally so because of the accompanying decrease in inhibitory firing. It might be remarked, in passing, that a number of loose ends may be rather neatly tied together if it is assumed that impulses reaching a long-axon cell in the cortex from other cortical cells do not actually fire that cell, but induce a long-lasting depolarization that sensitizes it to bombarding impulses from the arousal system. Thus the cell would be *primed,* but not fired, by other cortical firing, and then fired by the next few impulses that it receives from the nonspecific arousal system. Such a mechanism would be consistent with the observation of Li and Jasper (9) that the rate of cortical firing increases with the level

of arousal; it would also be in agreement with the data of Clare and Bishop (2) on the long-lasting dendritic depolarization set up by association afferents. Moreover, the work of Bradley (1) might be interpreted as indicating that the arousal system afferents are cholinergic, and as acetylcholine is destroyed instantly on being released it is unlikely that the arousal system impulses would give rise to any long-lasting effect in the cortex.

Lindsley (10) has indicated that there is a rough correlation between the motivational and emotional states of an animal and the level of activity of its arousal system; and Sharpless (12) has shown that novel stimuli are more potent stimulators of the arousal system than are familiar ones. It is also generally true that a hungry or otherwise uncomfortable animal has an increased level of arousal activity, especially in the presence of a satisfier. It thus seems that conditions which increase firing in the nonspecific system are those which tend to promote learning. There is still dispute as to how essential reinforcement might be for learning, but there is little doubt that some sort of "emphasis" on the relevant stimuli accelerates the learning process. In terms of our model, this means that cortical cells are linked together more rapidly when the rate of cortical activity is higher—a most reasonable conclusion. It might also be deduced from the model that *some* association should be possible as long as *any* cortical cells are firing (during dreams, for example). The reinforcement controversy thus reduces to the question of *how much* firing is going on in the cell assemblies concerned, reinforcing agents serving to increase that firing through their effects on the nonspecific projection system. According to this theory, therefore, the arousing rather than the need-reducing properties of the reinforcement are those important for learning. Evolution would account for the fact that, for most present-day animals, need reducers and dangerous situations tend to be particularly arousing.

It should be noted that, in the examples given, it has not been a response but rather a relationship between stimuli that has been learned. It is not too difficult to extend the model to account also for the association between stimuli and responses, but we then have to face the problem of why the stimulus is sometimes followed by the response and at other times not. No doubt this is sometimes due to the failure of effective association between the cell assemblies concerned, but we have introspective evidence which suggests that we may know what to do to achieve a goal, but not be sufficiently interested to perform the actions. This brings us to the problem of the effect of motivation on performance. Unfortunately, we cannot do justice to this topic in the space of this paper, but a brief indication will be given as to how the model might be able to handle it. We must assume that many motor neurons will be fired along with the neurons of the cell assemblies we have described, but because they must compete with postural and other reflexes for control of the musculature, they can only produce a response if they fire at a high enough frequency. Below some threshold value, cortical firing has no effect on overt behavior. It is suggested that the arousal system is responsible for raising cortical activity above this threshold when the stimulus conditions are sufficiently exciting. (However, as Hebb has suggested [7], if the firing becomes too vigorous, some other factor may intervene to prevent further responses.) In the case of motivation set up by expectancy of reward or punishment, it must be assumed that the arousal system has been excited, not by the stimulus actually present, but by a cell assembly representing the moti-

vating stimulus which, through previous experience in the situation, had become associated with the cell assembly for the actual stimulus. The cell assembly for the motivating stimulus will be able to fire the arousal system because of an association built up through innumerable presentations of the motivating stimulus, under conditions where it fired both the cortical and the subcortical systems simultaneously. Thus the environmental stimuli fire their cell assemblies, these arouse the assembly of the motivating stimulus by association, and this in turn fires the arousal system, also by association.

An example might make the proposition clearer. When a rat is first placed in a maze, the arousal value of the novel surroundings will be sufficient to raise the level of cortical firing so that more or less randomly associated "motor" cell assemblies will influence overt behavior; the rat will explore. If no incentive is introduced, the effect of novelty will wear off and the rat will lie down and doze. However, if the rat is food-deprived, and food is placed in the maze, the interaction of the stimuli from the food with the cells in the rat's nervous system that are sensitized by deprivation will produce persistent excitation of the arousal system. Moreover, cell assemblies fired by the food stimuli (if the food is familiar) will have strong connections with the cells in the arousal system, so that even if the deprivation is not severe the presence of food will be likely to produce arousal. These cell assemblies for food stimuli will, of course, become associated with any other cell assemblies active at about the same time (those representing the sight of the food dish, or the feel of the maze floor, for example). Therefore, when on a subsequent occasion the hungry rat is returned to the maze, one of the assemblies fired by the stimuli in the starting box may be able to excite, by association, the assembly which represents the food, and thus, indirectly, the arousal system. The firing in the latter will not die out as soon as the cell assembly which aroused it ceases to fire; activity in the arousal system does not stop abruptly (possibly because its activity releases adrenalin into the blood stream). It will therefore continue to bombard the cortex at an increased rate for perhaps several minutes, and the motor component of any cell assemblies occurring during that time will fire intensely enough to produce a response. If, for example, a running response has been associated with the starting-box stimuli, the rat will actually run; if the same cell assembly (for running) had become active before the arousal system had been fired by the association between the starting-box stimuli and food, then the cortical firing would probably not have been intense enough to elicit overt movements. The same sluggishness would be expected if the animal was sated, so that the cells of the arousal system were not sensitive to food stimuli or their associated cell assemblies.

The above example is admittedly superficial. More complicated situations, such as avoidance learning, or extinction, cannot be explained without introducing still more postulates, in particular a system for inhibiting movements. The value of such a model would be primarily to demonstrate that it is possible to design a machine that would not only "want" or "avoid" arbitrarily designated stimuli, but also learn what to do about them under all environmental conditions to which it was sensitive. Whether the design would bear any relationship to the actual mechanism of animals which can also do these things could be known only after further research.

SUMMARY

A neural model has been presented, based on an "association-of-ideas" para-

digm of learning, similar to that used by Hebb. In it groups of neurons (cell assemblies) become representors of stimuli, and can then be linked together by being fired contiguously. The model differs from Hebb's in that an inhibitory regulatory system is postulated which limits (to a minute fraction of the total) the number of cortical neurons that can fire simultaneously, and insures that those firing are dispersed as widely as possible. A further change is introduced to meet the paradox that cell assemblies can be associated with one another without losing their individuality and being submerged in a composite new cell assembly. In association, it is not the cells of one assembly that acquire connections with the cells of another; instead, cells primed, or sensitized, by the first assembly become incorporated into the second. Thus, one principle of learning—the binding of cells into a group by repeated simultaneous firing—fulfills a double role; when the newly added cells are predominantly primed by sensory input perceptual learning results; and when the new cells are primed by the firing of another cell assembly, associative learning results.

Because the effect of priming lasts for many seconds, it is possible for a cell assembly to accumulate the sensitizations induced by the activities of a number of associated assemblies, and so increase the probability that it will itself fire.

The dual role of motivation—the facilitation of learning and the elicitation of responses—has been discussed in terms of the arousal effects of the nonspecific projection system on the postulated cortical network.

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