Donald O. Hebb's book, *The Organization of Behavior*, is famous among neural modelers because it was the first explicit statement of the physiological learning rule for synaptic modification that has since become known as the Hebb synapse. However, the book covers a great deal more material than that, and is a thoughtful and thorough review of neuropsychology, as of 1949.

We have included Hebb's Introduction in this excerpt. It is a brief and lucid discussion of the connection between psychology and physiology, and has not dated one bit between 1949 and now.

The Introduction is also notable because in it is one of the first uses of the word "connectionism" in the context of a complex brain model. The final paragraph of the Introduction contains three lines: "The theory is evidently a form of connectionism, one of the switchboard variety, though it does not deal in direct connections between afferent and efferent pathways: not an S-R psychology if R means an muscular response. The connections serve rather to establish autonomous central activities, which then are the basis of further learning" (p. xix). Most modern day connectionists could find little to argue with in that summation.

A more detailed description of Hebb's physiological ideas is found in chapter 4. In this chapter, Hebb has a detailed discussion of neurophysiology and neuroanatomy as it relates to his ideas. It is worth emphasizing, if it is not obvious at this point, that the early modelers really knew their neuroscience. James, McCulloch, and Hebb were highly knowledgeable about the nervous system, and used their knowledge extensively in their models. Much modern work in neural networks has moved far away from its roots in the study of the brain and psychology. This is a cause for concern, both because the field is losing contact with its foundations and because it has lost a source of valuable ideas.

Hebb suggests several important ideas in chapter 4. First, and most famous, was the clear statement of what has become known as the "Hebb" synapse. To quote Hebb's description, for the nth time, "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" (p. 56). This, like the other ideas in Hebb's book, is not a mathematical statement, though it is close to one. For example, Hebb does not discuss the various possible ways inhibition might enter the picture, or the quantitative learning rule that is being followed. This has meant that a number of somewhat quite different learning rules can legitimately be called "Hebb synapses." (Paper 6, an early computer simulation of Hebb's ideas, discusses the modifications one must make to this bare outline to make the system work.)
Second, Hebb is keenly aware of the "distributed" nature of the representation he is assuming the nervous system uses. The idea is that to represent something, many cells must participate in the representation. Hebb was aware of the work of Lashley (paper 3), suggesting widely distributed representations, and made some use of his ideas, though not in the strongest form of complete "equipotentiality."

Third, Hebb postulated the formation of what he called "cell assemblies," which were really the heart of the entire book. The basic idea was that there were inter-connected, self-reinforcing subsets of neurons that formed the representations of information in the nervous system. Single cells might belong to more than one assembly, depending on the context. Multiple cell assemblies could be active at once, corresponding to complex perceptions or thoughts. There was a distributed representation at the functional level as well as at the anatomical level. Hebb devotes a good deal of attention to the details of the neuroanatomy and physiology that might underlie cell assemblies. The later chapters in the book contain many discussions of how cell assemblies can be used to help explain a number of psychological phenomena.

In retrospect, the idea that there exist temporarily stable, relatively long lasting neural activity patterns that are important in mental activity has reappeared in the various "attractor" models for brain activity (see, for example, Hopfield, paper 27; Grossberg, paper 24; or Anderson et al., paper 22). Details of the observed and predicted stability depend critically on learning assumptions that are nearly always based to some degree on Hebb synapses. Some of the ideas described in this book have become part of the accepted lore of the field.
Introduction

It might be argued that the task of the psychologist, the task of understanding behavior and reducing the vagaries of human thought to a mechanical process of cause and effect, is a more difficult one than that of any other scientist. Certainly the problem is enormously complex; and though it also could be argued that the process made by psychology in the century following the death of James Mill, with his crude theory of association, is by achievement scarcely less than that of the physical sciences in the same period, it is nevertheless true that psychological theory is still in its infancy. There is a long way to go before we can speak of understanding the principles of behavior to the degree that we understand the principles of chemical reaction. In an undertaking of such difficulty, the psychologist presumably must seek help wherever he can find it. There have been an increasing number of attempts to develop new mathematical methods of analysis. With some, in general, I do not attempt to deal. The method of factor analysis developed by Spearman (1927) and greatly elaborated by Thurstone (1935) is well established as a powerful tool for handing certain kinds of data, though the range of its use has been limited by dependence on norms that can be conveniently given to large groups of subjects. Another method is the application of matrix analysis more directly to the interaction of populations of neurons, by Yarrow, Pies, Householder, Lashly, McCulloch, and others.* Bishop (1946) has discussed the work from the point of view of neurophysiology, and his remarks are fully concurred with here. The preliminary studies made with this method so far have not only failed to simplify the physiological problem almost out of existence. This is not to criticize, since the attempt is to develop methods that can later be extended to deal with more complex data that as matters stand at present one must wait for further results before being sure that the attempt will succeed. Undoubtedly there is great potential value in such work, and if the right set of initial assumptions can be found it will presumably become, like factor analysis, a powerful ally of other methods of study.

However, psychology has an intimate relation with the other biological sciences, and may also seek for help there. There is a considerable overlap between the problems of psychology and those of neurophysiology, both the possibility (or necessity) of reciprocal assistance. The first object of this book is to present a theory of behavior for the consideration of psychologists; but another is to seek a common ground with the anatomist, physiologist, and neurologist, to show them how psychological theory relates to their problems and at the same time to make it more possible for them to contribute to that theory.

Psychology is no more static than any other science. Physiologists and clinicians who wish to get a theoretical conceptions cannot depend only on the writings of Pavlov or Freud. These were great men, and they have contributed greatly to psychological thought. But their contribution was rather in formulating and developing problems than in providing final answers. Pavlov himself seems to have thought of his theory of conditioned reflexes as something in constant need of revision, and experimental results have continued to make revisions necessary: the theory, let it is, is still developing. Again, if one were to regard Freud's theory as needing change only in its details, the main value of his work would be to some extent unnecessary: he was the one to see and state important facts of behavior, but they are also dangerously easy to treat in a misleadingly realistic or anthropomorphic way that was the demise of that, overwhelming one another by fact or guise, and being purged or being punished. Freud has left the task of developing these provisional formulations of his to the point where such a danger no longer exists. When theory becomes static it is apt to become dogmatic, and psychological theory has the further danger, as long as many of its problems are unresolved, of inspiring a refuge into the hedonism and overabundance of traditional thought.

*See papers by Cattell referred. Some, 1948, 13: 33–40 and 77: 103, and Bishop's review article, "of some of the more important of the whole in this book."
It is only too easy, no matter what formal theory of behavior one opposes, to entertain a concealed mysticism in one's thinking about that large segment of behavior which theory does not handle adequately.

To deal with behavior at present, one must oversimplify. The risk, as on the one hand, is of forgetting that one has oversimplified the problem; one may forget or even deny those inconvenient facts that one's theory does not accommodate. On the other hand is the risk of accepting the weak-kneed discouragement of the vitalist, of being content to show that existing theories are imperfect without seeking to improve them. We can take for granted that any theory of behavior at present must be inadequate and incomplete. But it is never enough to say, because we have not yet found out how to reduce behavior to the control of the brain, that no one in the future will be able to do so.

Modern psychology takes completely for granted that behavior and neural function are perfectly correlated, that one is completely caused by the other. There is no separate soul or life-force to sick a finger into the brain now and then and make neural cells do what they would not otherwise. Actually, of course, this is a working assumption only as long as there are unexplained aspects of behavior. It is quite conceivable that some day the assumption will have to be rejected. But it is important also to see that we have not reached that day yet: the working assumption is a necessary one, and there is no real evidence opposing it. Our failure to solve a problem so far does not make it insolvable. One cannot logically be a determinist in physics and chemistry and biology, and a mystic in psychology.

All one can know about another's feelings and awareness is an inference from what he does—from his muscular contractions and glandular secretions. These observable events are determined by electrical and chemical events in nerve cells. If one is to be consistent, there is no room here for a mysterious agent that is defined as not physical and yet has physical effects (especially since many of the entities of physics are known only through their effects). "Mind" can only be regarded, for scientific purposes, as the activity of the brain, and this should be mystery enough for any one: it doubles the appalling number of cells (some nine billion, according to Herrick) and even more appalling number of possible connections between them, the matter out of which cells are made is being itself renewed by the physical to something quite unlike the inert stick or stone with which mind is traditionally contrasted. After all, it is that contrast that at the bottom of the vitalist's objection to a mechanistic biology and the contrast has lost its force (Herrick, 1929).

The mystic might well concentrate on the electron and let behavior alone. A philosophical parallelism or idealism, whatever one may think of such conceptions on other grounds, is quite consistent with the scientific method, but interactionism seems not to be.

Psychologist and neurophysiologist thus chart the same b-y—working perhaps from opposite shores, sometimes overlapping and duplicating one another, but using some of the same fixed points and continually with the opportunity of contributing to each other's results. The problem of understanding behavior is the problem of understanding the total action of the nervous system, and vice versa. This has not always been a welcome proposition, either to psychologist or to physiologist.

A vigorous movement has appeared both in psychology and psychiatry to do rid of "physiologizing," that is, to stop using physiological hypotheses. This point of view has been clearly and effectively put by Skinner (1938), and it does not by any means represent a relapse into vitalism. The argument is related to modern positivism, emphasizes a method of correlating observable stimuli with observable response, and, recognizing that "explanation" is ultimately a statement of relationships between observed phenomena, proposes to go to the heart of the matter and have psychology confine itself to such statements now. This point of view has been criticized by Pratt (1939) and Kohler (1940). The present book is written in profound disagreement with such a program for psychology. Disagreement is on the grounds that this arises from a misconception of the scientific method as it operates in the earlier stages. Those apparently na"ive features of older scientific thought may have had more to do with hitting on sterile assumptions and hypotheses than seem necessary in retrospect. The anti-physiological position, thus, in urging that psychology proceed now as it may be able to proceed when it is more highly developed, seems to be in short a counsel of perfection, disregarding the limitations of the human intellect. However, it is logically defensible and may yet show by thoroughness of results that it is indeed the proper approach to achieving prediction and control of behavior.

If some psychologists jib at the physiologist for a bedfellow, many physiologists agree with them heartily. One must sympathize with those who want nothing of the psychologist's hair-splitting or the indefiniteness of psychological theory. There is much more certainty in the study of the electrical activity of a self-contained tract in the brain. The only question is whether a physiology of the human brain as a whole can be achieved by such studies alone. One can discover the properties of its various parts more or less in isolation; but it is
a truce by now that the part may have properties that are not evident in isolation, and these are to be discovered only by study of the whole brain. The method then calls for learning as much as one can about what the parts of the brain do (primarily the physiologist's field), and relating behavior as far as possible to this knowledge (primarily for the psychologist), then seeing what further information is to be had about how the total brain works, from the inter- 

craperacy between (1) actual behavior and (2) the 

behavior that would be predicted from adding up what is known about the action of the various parts.

This does not make the psychologist a physiologist, for precisely the same reason that the physiologist need not become a cytopathologist, though he is certainly concerned with the information that cytopathology and biochemistry provide. The difficulties of finding order in behavior are great enough to re- 

quire all one's attention, and the psychologist is inter- 

ested in physiology to the extent that it contributes to his own task.

The great argument of the positivists who object to "physiologyizing" is that physiology has not helped psychological theory. But, even if this is true (there is some basis for denying it), one has to add the words so far. There has been a great access of knowledge in neurophysiology since the twenties. The work of Berger, Dusser de Barenne, and Lecointe de Nol (as examples) has a profound effect on the physiological conceptions utilized by psychology, and physiology has not yet assimilated these results fully.

The central problem with which we must find a way to deal can be put in two different ways. Psychologically, it is the problem of thought some sort of process not is not fully controlled by environmental stimulation and yet cooperates closely with that stimulation. From another point of view, physiologically, the problem is that of the transmission of excitation from sensory to motor cortex. This statement may not be as much over- 

simplified as it seems, especially when one recognizes that "transmission" may be a very complex process indeed, with a considerable time lag between sensory stimulation and the final motor response. The failure of physiology to handle thought adequately (or the failure of neurophysiology to tell us how to conceive of cortical transmission) has been the essential weakness of modern psychological theory and the reason for persistent difficulties in dealing with a wide range of experimental and clinical data, as the following chap- 

ters will try to show, from the data of perception and learning to those of hunger, sleep, and sexuality.

In mammals even as low as the rat it has turned out to be impossible to describe behavior as an interaction directly between sensory and motor processes. Some-thing like thinking, that is, intervene. 'Thought' un-

doubtably has the connotation of a human degree of complexity as cerebral function and may mean too much to be applied to lower animals. But even in the rat there is evidence that behavior is not completely conditioned by sensory events, there are cen- 

tral processes operating also.

What is the nature of such relatively autonomous activities in the creature? Not even a tentative answer is available. We know a good deal about the different pathways to the cortex, about the various pathways from it, and about many at least linking the two. But the links are complex, and we know practically nothing about what goes on between the arrival of an excitation at a sensory projection area and its later departure from the motor area of the cortex. Psychology has had to 

find, in hypothesis, a way of bridging this gap in its physiological foundation. In general the bridge can be described as some comparatively simple formula of cortical transmission. The particular formula chosen mainly determines the nature of the psychological theory that results and the mood of choice is the major source of theoretical schism.

Two kinds of formula have been used, leading at two extremes to (1) switching theory, and sensori-motor 

connections; and (2) field theory. Either of these terms may be regarded as approximate; they are not so used here (1) In the first type of theory, at one extreme, cells in the sensory system acquire connections with cells in the motor system; the function of the cortex is that of a telegraphic exchange. Connections rigidly determine what animal or human being does, and their acquisi- 

tion constitutes learning. Current forms of the theory 

tend to be vaguer than formerly, because of effective criticism of the theory in its earlier and simpler forms, but the fundamental idea is still maintained. (2) Theory at the opposite extreme, there is a rigid dependence on 

connections at all, and attempts to utilize instead the field conception that physics has found so useful. The cortex is regarded as a medium of uniformity so that it can be treated as a statistically homogeneous medium. The sensori control of motor centers depends, accordingly, on the state of the network's excitation and on ratios of excitation, not on loci or the action of any specific cells.

Despite their differences, however, both theoretical approaches seek to imply a prompt transmission of sensory excitation to the motor side, if only by failing to specify that this is not so. No one, at any rate, 

* The original footnote appears to have been meant for another document and is not relevant here. It is not clear what the author meant to convey with this note.
has made any serious attempt to elaborate ideas of a central neural mechanism to account for the delay, between stimulation and response, that seems to be ac- cretive of thought. There have indeed been neural theories of "motion" thought, but they amount essen- tially to a continuous display of proprioception and minimal muscular action, and do not provide for any prolonged sequence of intracerebral events as such. But the recalcitrant data, of animal behavior have been drawn attention more and more insistently to the need of some better account of central processes. This is what Morgan (1943) has recognized in saying that "mental" variables, repeatedly thrown out because there was no place for them in a stimulus-response psychology, repeatedly find their way back in again in one form or another. The image has been a forbidden notion for twenty years, particularly in animal psy- chology; but the need was hardly exercised before "expectancy" had appeared instead. What is the neural basis of expectancy, or of attention, or interest? Other theory could use these words freely, for it made no serious attempt to avoid an interactionist philosophy. In modern psychology such terms are an embarrass- ment; they cannot be escaped if one is to give a full account of behavior, but they still have the smell of animism; and must have, until a theory of thought is developed to show how "expectancy" or the like can be a physiologically intelligent process.

In the chapters that follow this introduction I have tried to lay a foundation for such a theory. It is, on the one hand, and from the physiologist's point of view, quite speculative. On the other hand, it achieves some synthesis of psychological knowledge, and it attempts to hold as strictly as possible to the psychological evidence in those long stretchles where the guidance of anatomy and physiology is lacking. The desideratum is a conceptual tool for dealing with expectancy, atten- tion, and so on, and with a temporally organized intra- cerebral process. But this would lack little value if it did not also compute the main facts of perception, and of learning. To achieve something of the kind, the limitations of a schema are accepted with the purpose of developing certain conceptions of neural action. This is attempted in Chapters 4 and 5; Chapter 3 has been written to fill the ground for this undertaking. From Chapter 6 onward the conceptions derived from sche- matizing are applied to the problems of learning, vol- tion, emotion, hunger, and so on. In general, the reader may regard Chapters 1 to 3 as mainly preparatory, unless he is particularly interested in the neurological details, or in the treatment of perception, to get the gist of the theory that is presented here one should read the two following paragraphs, and turn directly to Chapter 6. In outline, the conceptual structure is as follows:

Any frequently repeated, particular stimulation will lead to the slow development of a "self-assembly," a diffuse structure comprising cells in the cortex and dienomphalon (and also, perhaps in the basal ganglia of the cerebrum), capable of acting briefly as a closed system, delivering facilitation to other such systems and usually having a specific motor facilitation. A series of such events constitutes a "phase sequence"—the thought process. Each assembly action may be aroused by a preceding assembly, by a sensory event, or—normally—by both. The central facilitation from one of these activities on the next is the prototype of "attention." The theory proposes that in this central facilitation, and its varied relationship to sensory pro- cesses, lies the answer to an issue that is made inescap- able by Pumphrey's (1940) penetrating review of the problem of the direction of thought.

The kind of cortical organization discussed in the preceding paragraph is what is regarded as essential to adult waking behavior. It is proposed also that there is an alternate, "intrinsie" organization, occurring in sleep and in infancy, which consists of hypersynchrony in the firing of cortical cells. But besides these two forms of cortical organization there may be disorganiza- tion. It is assumed that the assembly depends com- pletely on a very delicate timing which might be disturbed by metabolic changes as well as by sensory events that do not accord with the pre-existent central process. When this is transient, it is called emotional disorientation, when chronic, emotional psychosis.

The theory is evidently a form of connectionism, one of the switchboard variety, though it does not deal in direct connections between different and efferent path- ways: not an "S-R" psychology, if R means a muscular response. The connections serve rather to establish autonomous centers, from which are the basis of further learning. In accordance with modern physio- logical ideas, the theory also utilizes local field pro- cesses and gradients, following the lead, in particular, of Marshall and Talbot (1946). It does not, further, make any single nerve cell or pathway essential to any habit or perception. Modern physiology has presented psychology with new opportunities for the synthesis of divergent theories and previously untested data, and it is in my intent to take advantage of these opportunities as I can.
4. The First Stage of Perception: Growth of the Assembly

This chapter and the next develop a schema of neural action to show how a rapprochement can be made between (1) perceptual generalization, (2) the permanence of learning, and (3) attention, determining tendency or the like. It is proposed that a repeated stimulation of specific receptors will lead slowly to the formation of an "assembly" of association-area cells which can act briefly as a closed system after stimulation has ceased; this prolongs the time during which the structural changes of learning can occur and constitutes the simplest instance of a representative process (image or idea). The way in which this cell assembly might be established, and its characteristics, are the subject matter of the present chapter. In the following chapter the interrelationships between cell assemblies are dealt with; these are the basis of temporal organisation in central processes (attention, attitude, thought, and so on). The two chapters (4 and 5) construct the conceptual tools with which, in the following chapters, the problems of behavior are to be attacked.

The first step in this neural schematizing is the bald assumption about the structural changes that make lasting memory possible. The assumption has repeatedly been made before, in one way or another, and repeatedly found unsatisfactory by the critics of learning theory. I believe it is still necessary. As a result, I must show that in another context, of added anatomical and physiological knowledge, it becomes more defensible and more fruitful than in the past.

The assumption, in brief, is that a growth process accompanying synaptic activity makes the synapse more readily reversed. This hypothesis of synaptic resistances, however, is different from earlier ones in the following respects: (1) structural connections are postulated between single cells, but single cells are not effective units of transmission and such connections would be only one factor determining the direction of transmission; (2) no direct sensory-motor connections are supposed to be established in this way, in the adult animal; and (3) an innate relationship is postulated between reverberatory action and structural changes at the synapse, implying a dual trace mechanism.

The Possibility of a Dual Trace Mechanism

Hilgard and Marquis (1948) have shown how a reverberatory, transient trace mechanism might be proposed on the basis of Lorente de Nó's conclusions, that a cell is fired only by the simultaneous activity of two or more afferent fibers, and that interneuronal fibers are arranged in closed (potentially self-excitatory) circuits. Their diagram is arranged to show how a reverberatory circuit might establish a sensori-motor connection between receptor cells and the effector which carry out a conditioned response. There is of course a good deal of psychological evidence which is opposed to such an oversimplified hypothesis, and Hilgard and Marquis do not put weight on it. At the same time, it is important to see that something of the kind is not merely a possible but a necessary inference from certain neurological ideas. To the extent that anatomical and physiological observations establish the possibility of reverberatory after-effects of a sensory event, it is established that such a process would be the physiological basis of a transient "memory" of the stimulus. There may, then, be a memory trace that is wholly a function of a pattern of neural activity, independent of any structural change.

Hilgard and Marquis go on to point out that such a trace would be quite unstable. A reverberatory activity would be subject to the development of refractory states in the cells of the circuit in which it occurs, and external events could readily interrupt it. We have already seen (in Chapter 1) that an "activity" trace can hardly account for the permanence of early learning, but at the same time none may regard reverberatory activity as the explanation of other phenomena.

There are memories which are instantaneously established, and as evanescent as they are immediate. In the repetition of digits, for example, an interval of a few seconds is enough to prevent any interference from one series on the next. Also, some memories are both instantaneously established and permanent. To account for the permanence, some structural change seems necessary, but a structural growth presumably would require an appreciable time. If some way can be found of supposing that a reverberatory trace might cooperate with the structural change, and carry the memory until the growth change is made, we should be able to account for the temporary nature of the trace which is an activity only, without having to ascribe all memory to it. The conception of a transient, unalterable reverberatory trace is therefore useful, if it is possible to suppose also that some more permanent structural change reinforces it. There is no reason to think that a choice must be made between the two conceptions; there may be traces of both kinds, and memories which are dependent on both.

A neurophysiological Postulate

Let us assume then that the persistence or repetition of a reverberatory activity (or "trace") tends to induce
lasting cellular changes that add to its stability. The assumption can be precisely stated as follows: 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.

The most obvious and I believe much the most probable suggestion concerning the way in which one cell could become more capable of firing another is that synaptic knobs develop and increase the area of contact between the afferent axon and efferent soma. ("Soma" refers to dendrites and body, or all of the cell except its axon.) There is certainly no direct evidence that this is so, and the postulated change if it exists may be metabolic, affecting cellular rhythmicity and lumen; or there might be both metabolic and structural changes, including a limited neurobiotaxis. There are several considerations, however, that make the growth of synaptic knobs a plausible conception. The assumption stated above can be put more definitely, as follows:

When one cell repeatedly fires in firing another, the axon of the first cell develops synaptic knobs (or enlarges them if they already exist) in contact with the soma of the second cell. This seems to me the most likely mechanism of a lasting effect of nevrobiotaxic action, but I wish to make it clear that the subsequent discussion depends only on the more generally stated proposition italicized above.

It is wise to be explicit on another point also. The proposition does not require action at any great distance, and certainly is not the same as Kappers' (Kappers, Huber, and Crosby, 1936) conception of the way in which neurobiotaxis controls axonal and dendritic outgrowth. But my assumption is evidently related to Kappers' ideas, and not inconsistent with them. The theory of neurobiotaxis has been severely criticized, and clearly it does not do all it was once thought to do. On the other hand, neurobiotaxis may still be one factor determining the connections made by neural cells. If so, it would cooperate very neatly with the knob formation postulated above. Criticism has been directed at the idea that neurobiotaxis directs axonal growth throughout its whole course, and that the process sufficiently accounts for all neural connections. The idea is not tenable, particularly in view of such work as that of Weiss (1941) and Sperry (1945).

But none of this has shown that neurobiotaxis has any influence in neural growth, its operation, within ranges of a centimeter or so, is still plausible. Thus, in figure 6 (Lorenne de No, 1938a), the multiple synaptic knobs of fiber 2 on cell C might be outgrowths from a fiber passing the cell at a distance, and determined by the fact of repeated simultaneous excitations in the two. Again, the course followed by fiber 7 in the neighborhood of cell D may include deflections from the original course of the fiber, determined in the same way.

The details of these histological speculations are not important except to show what some of the possibilities of change at the synapse might be and to show that the mechanism of learning discussed in this chapter is not wholly out of touch with what is known about the neural cell. The changed facilitation that constitutes learning might occur in other ways without affecting the rest of the theory. To make it more specific, I have chosen to assume that the growth of synaptic knobs, with or without neurobiotaxis, is the basis of the change of facilitation from one cell to another, and this is not altogether implausible. It has been demonstrated, by Arvanitaki (1942) that a contiguity alone will permit

*See p. 219 for a further discussion of this point and an elaboration of the assumptions made concerning the nature of memory.

Figure 6 Relationships between synaptic knobs and the cell body. From Lorenne de No, 1938a. Courtesy of Charles C. Thomas and the author.
the excitation aroused in one cell to be transmitted to another. There are also earlier experiments, reviewed by Arvanitaki, with the same implication. Even more important, perhaps, is Erlanger's (1939) demonstration of impulse transmission across an artificial "synapse," a blocked segment of nerve more than a millimeter in extent. Consequently, in the intact nervous system, an axon that passes close to the dendrites or body of a second cell would be capable of helping to fire it, when the second cell is also exposed to other stimulation at the same point. The probability that such closely timed coincidental excitations would occur is not considered for the moment but will be returned to. When the coincidence does occur, and the active fiber, which is merely close to the soma of another cell, adds to a local excitation in it, it is assumed that the joint action tends to produce a thickening of the fiber—forming a synaptic knob—or adds to a thickening already present.

Lorren de Né (1938a) has shown that the synaptic knob is usually not a terminal structure (thus the term "end foot" at "end button" is misleading) nor always separated by a stalk from the axon or axon collateral. If it were, of course, some action at a distance would be inevitably suggested, if such conceptions are formed in learning. The knob instead is often a rather irregular thickening in the unmyelinated part of an axon near its ending, where it is threading its way through a thicket of dendrites and cell bodies. The point in the axon where the thickening occurs does not appear to be determined by the structure of the cell of which it is a part. It is not connected to the cell and related to the presence of a second cell. The number and size of the knobs formed by one cell in contact with a second cell vary also. In the light of these facts it is not implausible to suppose that the extent of the contact established is a function of joint cellular activity, given propriety of the two cells.

Also, if a synapse is crossed only by the action of two or more afferent cells, the implication is that the greater the area of contact the greater the likelihood that action in one cell will be decisive in firing another.

*One point should perhaps be made explicit. Following Lorren de Né, two afferent cells are considered to be efficacious at the synapse, when one is not, only because their contacts with the effenter cell are closer together so their action summates. When both are active, they create a larger region of local disturbance in the effenter soma. The larger the knobs in a given cluster, therefore, the smaller the number that might activate the cell on which they are located. On occasion, a single afferent cell must be effective in transmissio. It is worth pointing this out, also, because it might appear to the reader otherwise that there is something mysterious about emphasis on the multiplicity of afferents to a mere cell to activate the synapse. All that has really been shown is that in some circumstances two or more effenter cells are necessary. However, this inevitably implies that an increase in the number of afferent cells simultaneously active must increase the reliability with which the synapse is traversed.*

Thus all afferent fibers with extensive knob contact could fire a cell that otherwise might be fired only by four or more fibers; or fired sooner with knobs than without.

In short, it is feasible to assume that synaptic knobs develop with neural activity and represent a lower form of synaptic resistance. It is implied that the knobs appear in the course of learning, but this does not give us a means of testing the assumption. There is apparently no good evidence concerning the relative frequency of knobs in infant and adult brains, and the assumption does not imply that there should be none in the newborn infant. The learning referred to is learning in a very general sense, which might certainly have begun long before birth (see e.g., the footnote on pp. 121-2).

Conduction from Area 17

In order to apply this idea (of a structural reinforcement of synaptic transisition) to visual perception, it is necessary first to examine the known properties of conduction from the visual cortex, area 17, to areas 18, 19, and 20. (In view of the criticisms of architectonic theory by Laschley and Clark [1946], it may be said that Brodmann's areas are referred to here as a convenient designation of relative cortical position, without supposing that the areas are necessarily functional entities or always histologically distinctive.)

It has already been seen that there is a topological reproduction of retinal activities in area 17, but that conduction from 17 to 18 is diffuse. Von Bonin, Gaüzé, and McCulloch (1942) have found that a localized excitation in 17 is conducted to a large part of 18, a band lying along the margins of 17. There is no point-to-point correspondence of 17 and 18. Excitation from 18 is conducted back to the nearest border region of 17, to all parts of area 18 itself; and to all parts of the contralateral 18, of area 19 (lying anterior to 18), and of area 20 (in the lower part of the temporal lobe).

The diffusity of conduction from area 17 is illustrated by the diagram of figure 7. Cells lying in the same part of 17 may conduct to different points in 18. The cells in 18, thus stimulated, also lead to points in 18 itself which are widely separated to any part of the ipsilateral areas 19 and 20; and, though one synapse, to any part of the contralateral 19 and 20. Conversely, cells lying in different parts of 17 or 18 may have connections with the same point in 18 or 20.

Thus there is convergence as well as spread of excitation. The second point illustrated by figure 7 is a selective action in 18, depending on the convergence of fibers from 17. In the figure, F and G are two cells in area 18 connecting the same mono-striate areas, F,
however, is that one happens to be exposed to excita-
tions from both A and B (two different regions in
area 17). When an area-17 excitation includes both A
and B, P is much more likely to be fired than G. The
figure does not show the short, closed, multiple chains
which are found in all parts of the cortex and whose
facilitating activity would often make it possible for
a single fiber from B to fire G. But the same sort of
local bombardment would also aid in firing F; and the
cell which receives excitations from two area-17 fibers
simultaneously would be more likely to fire than that
which receives excitation from only one.

On the other hand, when B and C instead of A and
B are excited simultaneously, G would be much more
likely to fire than F. Any specific region of activity in area 17
would tend to excite specific cells in area 18 which
would tend not to be fired by the excitation of another
region in 17. These specific cells in 18 would be diffusely
arranged, as far as we know at random. They would be
usually at some distance from one another and would
always be intermingled with others which are not
fired by the same afferent stimulation, but because of
their lasting structural connections would tend always
to be selectively excited, in the same combina-
tion, whenever the same excitation recurs in area 17.
This of course would apply also in areas 19 and 20.
Since a single point in 18 fires to many points through-
out 19 and 20, excitation of any large number of area-
18 cells means that convergence in 19 and 20 must be
expected. How often it would happen is a statistical
question, which will be deferred to a later section.

The tissues made active beyond area 17, by two dif-
ferent visual stimuli, would thus be (1) grossly the same.

(2) histologically distinct. A difference of stimulating
pattern would not mean any gross difference in the par-

The in the last chapter it was shown that there are impor-
tant properties of perception which cannot be ascribed
to events in area 17, and that these are properties
which seem particularly dependent on learning. That
"identity" is not due to what happens in 17 is strongly
implied by the distortions that occur in the projection
of a retinal excitation to the cortex. When the facts of
hemianopic completion are also considered, the con-
clusion appears inescapable. Perception must depend
on other structures besides area 17.

But we now find, at the level of area 18 and beyond,
that all topographical organization in the visual pro-
cess seems to have disappeared. All that is left is activity
in an irregular arrangement of cells, which are inter-
tangled with others that have nothing to do with the
perception of the moment. We know of course that
perception of simple objects is unified and determinate,
a well-organized process. What basis can be found for
an integration of action, in cells that are anatomically
so disorganised?

An answer to this question is provided by the struc-
tural change at the synapse which has been assumed
to take place in learning. The answer is not simple;
perceptual integration would not be accomplished
directly, but only as a slow development, and, for
the purposes of exposition, at least, would involve several
distinct stages, with the first of which we shall now be
concerned.

The general idea is an old one, that any two cells or
systems of cells that are repeatedly active at the same
time will tend to become "associated," so that activity
in one facilitates activity in the other. The details of
speculation that follow are intended to show how this
old idea might be put to work again, with the equally
old idea of a lowered synaptic "resistance," under the
eye of a different neurophysiology from that which
engendered them. (It is perhaps worth while to note
that the two ideas have most often been combined only
in the special case in which one cell is associated with
another, or a higher level or order in transmission, which it fits; what I am proposing is a possible basis of 
interaction of twoafferent fibers of the same cell — in principle, a sensori-sensory association, in addition to the linear 
association of conditioning theory.)

The proposal is most simply illustrated by cells A, B, and C in figure 8. A and B, visual-sensory cells, are 
simultaneously active. The cell A synapses, of course, with a large number of cells in 18, and C is supposed 
to be one that happens to load back into 17. Cells such as C would be those that produce the local wedge-
shaped area of firing in 17 when a point in 18 is strychnized (von Bonin, Glade, and McCulloch, 1942). The 
cells in the region of 17 to which C leads are being fired by 
the same massive sensory excitation that fires A, and 
C would almost necessarily make contact with some cell B that also fires into 18, or communicate with B at 
one step removed, through a short-saxon circuit. 
With repetition of the same massive excitation in 17 the 
same-firing relations would recur and, according to the 
assumption made, growth changes would take place at 
synapses AC and CB. This means that A and B, both 
afferent neurons of the same order, would no longer act 
independently of each other.

At the same time, in the conditions of stimulation that are diagrammed in figure 8, A would also be likely 
to synapse (directly, or via a short closed link) with a cell D which leads back into an unexcited part of 17, 
and there synapses with still another cell E of the same 
order as A and B. The synapse DE, however, would be 
easily to be traversed, since it is not like CB expected to 
be potentiated affrent bombardment. Upon frequent 
repetition of the particular excitation in area 17, a 
functional relationship of activity in A and B would 
increase much more than a relationship of A to E.

It should be observed, however, that some theories have continued to maintain that "S-S" (sensori-sensory) excitations are formed in the learning process, and have provided experimental evidence that seems to establish the fact. See, e.g., Brogden, J. Exp. Psychol., 1941, 
17, 127-139; and other papers cited therein.

The same considerations can be applied to the activity of the enormous number of individual cells in 18, 19, 
and 20 that are simultaneously aroused by an extensive 
activity in 17. Here it should be observed, the evidence of 
neurophysiology implies that there are anatomical 
connections of every point with every other point, 
within a few milliseconds, and that there is no orderly 
arrangement of the cells concerned.

Figure 9 diagrams three cells, A, B, and C, that are 
effectively fired in 18 by a particular visual stimulation, 
repeatedly repeated (by fixation, for example, on some 
point in a constant distant environment). D, E, and X 
represent possible connections which might be found 
between such cells, directly or with intervening links. 
Supposing that time relations in the firing of these 
cells make it possible, activity in 4 would contribute to 
the firing of E, and that in B to firing C and D. Growth 
changes at the synapses AE, BC, BD, and so on, 
would be a broadening of integration and would 
increase the probability of coordinated activity in each 
pair of neurons.

The fundamental meaning of the assumption of 
growth at the synapse is that this effect would have on 
the timing of action by the effcrcnt cell. The increased 
area of contact means that firing by the effector cell is 
more likely to follow the lead of the afferent cell. A fiber 
of order n thus gains increased control over a fiber 
n+1, making the firing of n+1 more predictable or 
determinate. The control cannot be absolute, but 
"optimal" (Lorente de Nó, 1939), and depends also on 
other events in the system. In the present case, 
however, the massive excitation in 17 would tend to 
establish constant conditions throughout the system 
during the brief period of a single visual fixation; and 
the postulated synaptic changes would also increase 
the degree of this constancy. A would acquire an 
increasing control of E, and E, with each repetition of the 
visual stimulus, would fire more consistently at the
same time that $B$ is firing (8, it will be recalled, is directly controlled by the area-17 action). Synaptic changes $EH$ would therefore result. Similarly, $B$ acquires an increasing control of $D$; and whenever a cell such as $D$ happens to be one that connects again with $B$, through $X$, a closed cycle $(BDXB)$ is set up.

It is, however, misleading to put emphasis on the coincidences necessary for the occurrence of such a simple closed circuit. Instead of a ring or hoop, the best analogy to the sort of structure which would be set up or "assembled" is a closed solid cage-work, or three-dimensional lattice, with no regular structure, and with connections possible from any one intersection to any other. Let me say explicitly, again, that the specificity of such an assembly of cells in 18 or 20, to a particular excitation in 17, depends on coverage. Whenever two cells, directly or indirectly controlled by that excitation, converge on another cell (as $F$ and $X$ converge on $B$ in figure 9) the essential condition of the present schematizing is fulfilled: the two converging cells need not have any simple anatomical or physiological relation to one another, and physiological integration would not be supposed to consist of independent closed chains.

This has an important consequence. Lorente de Nó (1938) has put stress on the fact that activity in a short closed circuit must be rapidly extinguished, and could hardly persist as long as a hundredth of a second. It is hard, on the other hand, to see how a long, many-linked chain, capable of longer reverberation, would get established as a functional unit. But look now at figure 10, which diagrams a different sort of possibility. Arrows represent not neurons, but multiple pathways, of whatever complexity is necessary so that each arrow starts for a functional unit. These units fire in the order 1, 2, 3, ... 15. The pathway labeled (1, 4) is the first to fire, and also the fourth; (2, 14) fires second and fourteenth; and so on. The activity 1-2-3-4 is in a relatively simple closed circuit. At this point the next unit (2, 14) may be refractory, which would effectively extinguish reverberation in that simple circuit. But at this point, also, another pathway (5, 9) may be excitable and permit activity in the larger system to continue in some way as that suggested by the numbers in the figure. The sort of irregular three-dimensional net which might be the anatomical basis of perceptual integration in the association areas would be infinitely more complex than anything one could show with a diagram and would provide a large number of the multiple parallel (or alternate) units which are suggested by figure 16. If so, an indefinite reverberation in the structure might be possible, so long as the background activity in other cells in the same gross region remained the same. It would not of course remain the same for long, specially with changes of visual fixation; but such considerations make it possible to conceive of "alternating" reverberation — which might frequently last for periods of time as great as half a second or a second.

(What I have in mind, in emphasizing half a second or so as the duration of a reverberatory activity, is the observed duration of a single content in perception (Pillsbury, 1913, Boring, 1933). Attention wanders, and the best estimate one can make of the duration of a single "conscious content" is of this time-order.)

This then is the cell-assembly. Some of its characteristics have been defined only by implication, and these are to be developed elsewhere, particularly in the remainder of this chapter, in the following chapter, and in Chapter 8 (see pp. 195-7). The assembly is thought of as a system inherently involving some equipotentiality, in the presence of alternate pathways each having the same function, so that brain damage might remove some pathways without preventing the system from functioning, particularly if the system has been long established, with well-developed synaptic knobs which decrease the number of fibers that must be active at once to traverse a synapse.

**Figure 10** Arrows represent a simple "assembly" of neural pathways or open multiple chains firing according to the numbers on each (the pathways 1, 4 fire first and fourth, and so on), illustrating the possibility of an "alternating" reverberation which would not extinguish as readily as that in a simple closed circuit.

**Statistical Considerations**

It must have appeared to the reader who examined figures 8 and 9 carefully that there was something unlikely about its being arranged at the outset to have such neat connections exactly where they were most needed for my hypothesis of perceptual integra-

The answer of course is statistical: the neurons diagrammed were those which happen to have such connections, and, given a large enough population of connecting fibers distributed at random, the improbable
connection must become quite frequent, in absolute numbers. The next task is to assess the statistical element in these calculations, and show that probability is not stretched too far.

The diagrams and discussion of the preceding section require the frequent existence of two kinds of coincidence: (1) synchronisation of firing in two or more converging axons, and (2) the anatomical fact of convergence in fibers which are, so far as we know, arranged at random. The necessity of these coincidences sets a limit to postulating functional connections ad lib as the basis of integration. But this is not really a difficulty, since the psychological evidence as we shall see also implies that there are limits to perceptual integration.

Consider first the enormous frequency and complexity of the actual neural connections that have been demonstrated histologically and physiologically. One is apt to think of the neural cell as having perhaps two or three out of a dozen connections with other cells, and as leading from one minute point in the central nervous system to one other minute point. This impression is far from the truth and no doubt is due to the difficulty of representing the true state of affairs in a printed drawing.

Forbes (1939) mentions an example of 300 synaptic knobs on a single anterior horn cell. Lorente de Nó’s drawings (1943, figures 71–75) show a complexity, in the ramification of axon and dendrite, that simply has no relation whatever to diagrams (such as mine) showing a cell with one or two connections. The gross extent of the volume of cortex infiltrated by the collateral of the axon of a single neuron is measured in millimeters, not in microns; it certainly is not a single point, microscopic in size. In area 18, the striychnine method demonstrates that each tiny area of cortex has connections with the whole region. (These areas are about as small as 1 sq. mm., according to McCulloch, 1944-45.) It puts no great strain on probabilities to suppose that there would be, in area 18, some anatomical connection of any one cell, excited by a particular visual stimulation, with a number of others excited in the same way.

There is, therefore, the anatomical basis of a great number of convergences among the multitude of cortical cells directly or indirectly excited by any massive mental activity. This is to be kept in mind as one approaches the physiological question of synchronisation in the converging fibers. In the tridimensional, lattice-like assembly of cells that I have supposed to be the basis of perceptual integration, those interconnecting neurons which synapse with the same cell would be functionally in parallel. Figure 1 illustrates this. The pathways labeled (1), (4), (8), and (13), converging on one synapse, must have the same function in the system; or the two-link pathways (5), (6), (8), (10) the same function as the single link (2), (14). When impulses in one such path are not effective, those in another, arriving at a different time, could be.

Once more, the over-simplification of such diagrams is highly misleading. At each synapse these must be a considerable delay in the time of arrival of impulses, and in each individual fiber a constant useon of responsiveness and one could never predicate a determinate pattern of action in any small segment of the system. In the larger system, however, a statistical constancy might be quite predictable.

It is not necessary, and not possible, to define the cell assembly underlying a perception as being made up of neurons all of which are active when the proper visual stimulation occurs. One can suppose that there would always be activity in some of the group of elements which are in functional parallel (they are not of course geometrically parallel). When for example excitation can be conducted to a particular point in the system from five different directions, the activity characteristic of the system as a whole might be maintained by excitation in any three of the five pathways, and no one fiber would have to be synchronized with any other one fiber.

There would still be some necessity of synchronisation, and this has another aspect. In the integration which has been hypothesized, depending on the development of sympathies and an increasing probability of control by afferent over efferent fibers, there would necessarily be a gradual change in the frequency characteristics of the system. The consequence would be a sort of fractionation and recruitment, and some change in the neurons making up the system. That is, some units, capable at first of synchronizing with others in the system, would no longer be able to do so and would drop out. "Fractionation." Others, at first incompatible, would be recruited. With perceptual development there would be a slow growth in the assembly, under-stating by "growth" not necessarily an increase in the number of constituent cells, but a change. How great the change would be there is no way of telling, but it is a change that may have importance for psychological problems when some of the phenomena of association are considered.

This then is the statistical approach to the problem. It is directly implied that an "association" of two cells in the same region, or of two systems of cells, would vary, in the probability of its occurrence, over a wide range. If one chose such pairs at random one would find some between which no association was possible,
some in which association was promptly and easily established when the two were simultaneously active, and a large proportion, making up a gradation from one of these extremes to the other. The larger the system with a determinate general pattern of action, the more readily an association could be formed with another system. On a statistical basis, the more points at which a chance anatomical convergence could occur, the greater the frequency of effective interfacilitation between the two assemblies.

Psychologically, these ideas mean (1) that there is a prolonged period of integration of the individual perception, apart from associating the perception with anything else; (2) that an association between two perceptions is likely to be possible only after each one has independently been organized, or integrated; (3) that, even between two integrated perceptions, there may be a considerable variation in the ease with which association can occur. Finally, (4) the apparent necessity of supposing that there would be a "growth," or fractionation and recruitment, in the cell-assembly underlying perception means that there might be significant differences in the proportion of perception at different stages of integration. One cannot guess how great the changes of growth would be, but it is conceivable, even probable, that if one knew where to look for the evidence one would find marked differences of identity in the perceptions of child and adult.

The psychological implications of my schematizing, as far as it has gone, have been made explicit in order to show briefly that they are not contrary to fact. We are not used to thinking of a simple perception as slowly and painfully learned, as the present chapter would suggest, but it has already been seen, in the discussion of the vision of the congenitally blind after operation, that it actually is. The slowness of learning, and the frequent instances of total failure to learn at all in privates as great as a year following operation (Seguin, 1932), are extraordinary and incredible (if it were not for the full confirmation by Riesen, 1947). The principles of learning to be found in psychological textbooks are derived from the behavior of the half-grown or adult animal. Our ideas as to the readiness with which association is set up apply to the behavior of the developed organism, as Boring (1946) has noted: there is no evidence whatever to show that a similarly prompt association of separate perceptions can occur at birth—that it is independent of a slow process in which the perceptions to be associated must first be integrated.

As to the wide range in difficulty of associating two ideas or perceptions, even for the adult, this is psychologically a matter of common experience. Who has not had trouble remembering, in spite of repeated efforts, the spelling or pronunciation of some word, or the name of some acquaintance? The fact of the unequal difficulty of associations is not stressed in the literature, probably because it does not fit into conditioned-reflex theory, but it is a fact. My speculations concerning the nature of the trace and the aboriginal development of perception thus are not obviously opposed to the psychological evidence. Further evaluation can be postponed until the speculations have been fully developed.