(1949)
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The Organization of Behavior, New York: Wiley, Introduction and Chapter 4, "The first stage of perception: growth of the assembly," pp. xi–xix, 60–78

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 could also be argued that the progress made by psychology in the century following the death of James Mill, with his crude theory of association, is an 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 these, 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 handling certain kinds of data, though the range of its use has been limited by dependence on tests that can be conveniently given to large groups of subjects. Another method is the application of mathematics more directly to the interaction of populations of neurons, by Rashevsky, Pitts, Householder, Landahl, 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 been obliged to simplify the psychological problem almost out of existence. This is not a criticism, since the attempt is to develop methods that can later be extended to deal with more complex data; but 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 look for help there. There is a considerable overlap between the problems of psychology and those of neurophysiology, hence 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 orientation 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 continual need of revision, and experimental results have continued to make revisions necessary: the theory, that 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 stultified. Theorizing at this stage is like skating on thin ice—keep moving, or drown. Ego, Id, and Superego are conceptions that help one to see and state important facts of behavior, but they are also dangerously easy to treat as ghostly realities: as anthropomorphic agents that *want* this or *disapprove* of that, *overcoming* one another by force or guile, and *punishing* or *being punished*. Freud has left us 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 dogma; and psychological theory has the further danger, as long as so many of its problems are unresolved, of inviting a relapse into the vitalism and indeterminism of traditional thought.

*Two papers by Culbertson (*Bull. Math. Biophys.*, 1948, *10*, 31–40 and 97–102), and Bishop's review article, list some of the more important of the actual titles in this field.

It is only too easy, no matter what formal theory of behavior one espouses, 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, 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 subsume. 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 stick a finger into he 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 opposed to it. Our failure to solve a problem so far does not make it insoluble. 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 awarenesses 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 anyone: besides 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 reduced by the physicist to something quite unlike the inert stick or stone with which mind is traditionally contrasted. After all, it is that contrast that is 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 bay—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 be 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 Köhler (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 naïve features of older scientific thought may have had more to do with hitting on fertile assumptions and hypotheses than seems 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 its fertility 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 well-defined 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 truism 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 intact 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 discrepancy 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 cytologist or biochemist, though he is intimately concerned with the information that cytology and biochemistry provide. The difficulties of finding order in behavior are great enough to require all one's attention, and the psychologist is interested in physiology to the extent that it contributes to his own task.

The great argument of the positivists who object to "physiologizing" 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*. These has been a great access of knowledge in neurophysiology since the twenties. The work of Berger, Dusser de Barenne, and Lorente de Nó (as examples) has a profound effect on the physiological conceptions utilized by psychology, and psychology 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 that 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 oversimplified as it seems, especially when one recognizes that the "transmission" may be a very complex process indeed, with a considerable time lag between sensory stimulation and the final motor response. The failure of psychology 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 chapters will try to show, from the data of perception and learning to those of hunger, sleep, and neurosis.

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. Something like *thinking*, that is, intervenes. "Thought" undoubtedly has the connotation of a human degree of complexity in 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 controlled by immediate sensory events: there are central processes operating also.

What is the nature of such relatively autonomous activities in the cerebrum? Not even a tentative answer is available. We know a good deal about the afferent pathways to the cortex, about the efferent pathways from it, and about many structures 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 need of choosing is the major source of theoretical schism.

Two kinds of formula have been used, leading at two extremes to (1) switchboard theory, and sensori-motor connections; and (2) field theory. (Either of these terms may be regarded as opprobrium; 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 telephone exchange. Connections rigidly determine what animal or human being does, and their acquisition 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 denies that learning depends on connections at all, and attempts to utilize instead the field conception that physics has found so useful. The cortex is regarded as made up of so many cells that it can be treated as a statistically homogeneous medium. The sensory control of motor centers depends, accordingly, on the distribution of the sensory excitation and on ratios of excitation, not on locus or the action of any specific cells.

Despite their differences, however, both theoretical approaches seem 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 simplicity possibly accounts for the opinion expressed by an anatomist who claimed that psychologists think of the brain as having all the finer structure of a bowlful of porridge.

has made any serious attempt to elaborate ideas of a central neural mechanism to account for the delay, between stimulation and response, that seems so characteristic of thought. There have indeed been neural theories of "motor" thought, but they amount essentially to a continual interplay 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 drawing 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 psychology; but the fiend was hardly exorcised before "expectancy" had appeared instead. What is the neural basis of expectancy, or of attention, or interest? Older theory could use these words freely, for it made no serious attempt to avoid an interactionist philosophy. In modern psychology such terms are an embarrassment; 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 intelligible 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 stretches where the guidance of anatomy and physiology is lacking. The desideratum is a conceptual tool for dealing with expectancy, attention, and so on, and with a temporally organized intracerebral process. But this would have little value if it did not also comprise 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; Chapters 1 to 3 try to clear the ground for this undertaking. From Chapter 6 onward the conceptions derived from schematizing are applied to the problems of learning, volition, emotion, hunger, and so on. (In general, the reader may regard Chapters 1 to 5 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 "cell-assembly," a diffuse structure comprising cells in the cortex and diencephalon (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 processes, lies the answer to an issue that is made inescapable by Humphrey'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, "intrinsic" 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 disorganization. It is assumed that the assembly depends completely 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 disturbance; when chronic, neurosis or psychosis.

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 a *muscular* response. The connections serve rather to establish autonomous central activities, which then are the basis of further learning. In accordance with modern physiological ideas, the theory also utilizes local field processes and gradients, following the lead particularly of Marshall and Talbot (1942). 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 unrelated data, and it is my intent to take such 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 first 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 organization 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 a 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 fertile than in the past.

The assumption, in brief, is that a growth process accompanying synaptic activity makes the synapse more readily traversed. 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 sensori-motor connections are supposed to be established in this way, in the adult animal; and (3) an intimate 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 (1940) 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 internuncial fibers are arranged in closed (potentially self-exciting) circuits. Their diagram is arranged to show how a reverberatory circuit might establish a sensori-motor connection between receptor cells and the effectors 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 one 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 recognize the theoretical value of the trace which is an activity only, without having to ascribe all memory to it. The conception of a transient, unstable 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 axion 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 limen; 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 assists 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 reverberatory 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*b*) and Sperry (1943).

But none of this has shown that neurobiotaxis has *no* influence in neural growth; its operation, within ranges of a centimeter or so, is still plausible. Thus, in figure 6 (Lorente de Nó, 1938*a*), 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.

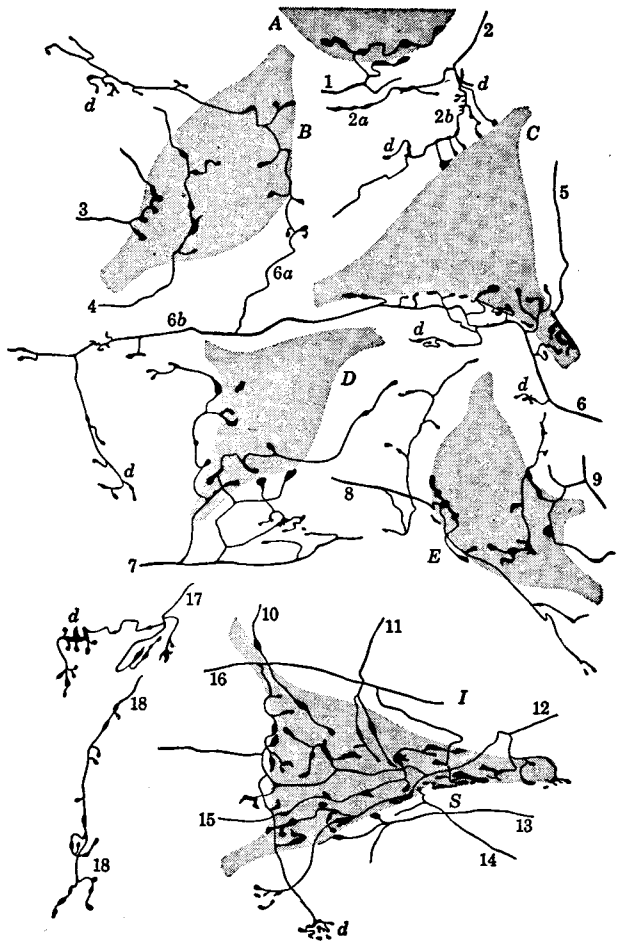

Figure 6 Relationships between synaptic knobs and the cell body. From Lorente de Nó, 1938*a*. Courtesy of Charles C. Thomas and of the author.

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 on another, and this is not altogether implausible. It has been demonstrated by Arvanitaki (1942) that a contiguity alone will permit

* See p. 229 for a further discussion of this point and an elaboration of the assumption made concerning the nature of memory.

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 dendrities 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, I assume that the joint action tends to produce a thickening of the fiber—forming a synaptic knob—or adds to a thickening already present.

Lorente de Nó (1938*a*) has shown that the synaptic knob is usually not a terminal structure (thus the term "end foot" or "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 connections 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 but by something external 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 propinquity 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 Lorente de Nó, two afferent cells are considered to be effective at the synapse, when one is not, only because their contacts with the efferent cell are close together so their action summates. When both are active, they create a larger region of *local* disturbance in the efferent 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 transmission. It is worth pointing this out, also, because it might appear to the reader otherwise that there is something mysterious about emphasis on the necessity of activity in two or more cells to activate the synapse. All that has really been shown is that in some circumstances two or more afferent 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 three 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 lowered 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 must 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 transmission) 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 Lashley 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, Garol, 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 macroscopic areas. *F*,

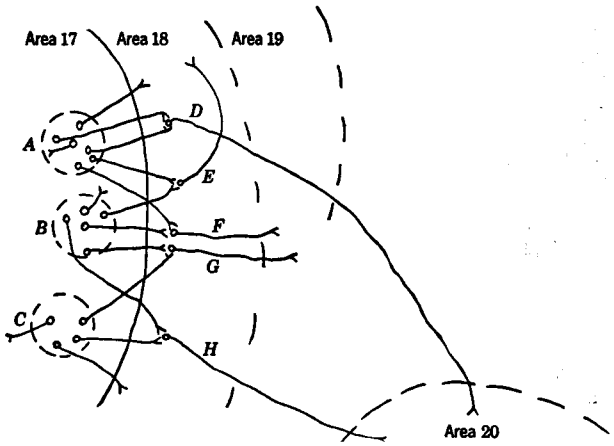

Figure 7 Illustrating convergence of cells in Brodmann's area 17 upon cells in area 18, these cells in turn leading to other areas. *A*, *B*, *C*, three grossly distinct regions in area 17; *D*, *E*, *F*, *G*, *H*, cells in area 18. See text.

however, is one that happens to be exposed to excitations from both *A* and *B* (two different regions in area 17). When an area-17 excitation includes both *A* and *B*, *F* 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 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 combination, 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 throughout 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 different 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 part of the brain which mediates perception (except in the afferent structures up to and including area 17, the visual cortex). Even a completely unilateral activity, it should be noted, would have diffuse effects throughout areas 18, 19, and 20 not only on one side of the brain but on both. At the same time, a difference of locus or pattern of stimulation would mean a difference in the particular cells in these areas that are consistently or maximally fired.

Mode of Perceptual Integration: The Cell-Assembly

In the last chapter it was shown that there are important 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 conclusion 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 process seems to have disappeared. All that is left is activity in an irregular arrangement of cells, which are intertangled 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 disorganized?

An answer to this question is provided by the structural 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

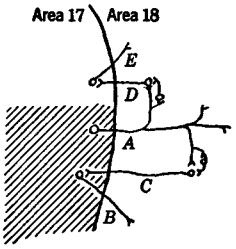

Figure 8 Cells *A* and *B* lie in a region of area 17 (shown by hatching) which is massively excited by an afferent stimulation. *C* is a cell in area 18 which leads back into 17. *E* is in area 17 but lies outside the region of activity. See text.

another, or a higher level or order in transmission, which it fires; what I am proposing is a possible basis of association of two afferent fibers of the same order—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-area 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 lead 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 strychninized (von Bonin, Garol, 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-axon 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 unlikely to be traversed, since it is not like *CB* exposed to concentrated afferent 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 theorists have continued to maintain that "S-S" (sensori-sensory) associations are formed in the learning process, and have provided experimental evidence that seems to establish the fact. See, *e.g.*, Brogden, *J. Exp. Psychol.*, 1947, 37. 527–539, and earlier papers cited therein.

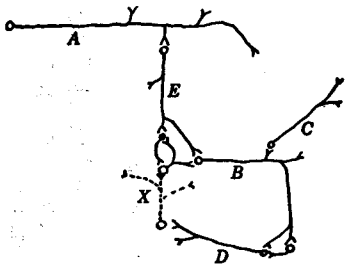

Figure 9 *A*, *B*, and *C* are cells in area 18 which are excited by converging fibers (not shown) leading from a specific pattern of activity in area 17. *D*, *E*, and *X* are, *among the many cells with which* A, B, *and* C *have connections*, ones which would contribute to an integration of their activity. See text.

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 neuronography implies that there are anatomical connections of every point with every other point, within a few millimeters, 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, frequently 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 *A* 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 beginning 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 in the effect this would have on the timing of action by the efferent cell. The increased area of contact means that firing by the efferent 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 "optional" (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 (*B*, it will be recalled, is directly controlled by the area-17 action). Synaptic changes *EB* 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 covergences.* Whenever two cells, directly or indirectly controlled by that excitation, converge on another cell (as *E* 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*b*) 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 stands 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 10. 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, especially with changes of visual fixaton; 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.

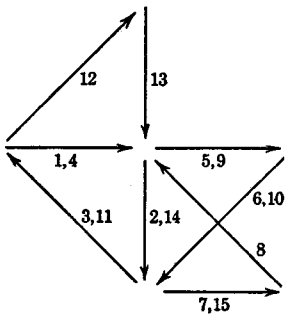

Figure 10 Arrows represent a simple "assembly" of neural pathways or open multiple chains firing according to the numbers on each (the pathway "1, 4" fires first and fourth, and so on), illustrating the possibility of an "alternating" reverberation which would not extinguish as readily as that in a simple 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 Creation to have such neat connections exactly where they were most needed for my hypothesis of perceptual integration. 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) synchronization 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 or half 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 for example an estimate of 1300 synaptic knobs on a single anterior horn cell. Lorente de Nó's drawings (1943, figures 71–73, 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 collaterals 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 strychnine 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*b*.) 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 retinal activity. This is to be kept in mind as one approaches the physiological question of synchronization 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 10 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 pathway (5, 9)–(6, 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 oversimplification of such diagrams is highly misleading. At each synapse there must be a considerable dispersion in the time of arrival of impulses, and in each individual fiber a constant variation 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 synchronization, and this has another aspect. In the integration which has been hypothesized, depending on the development of synaptic knobs and an increasing probability of control by afferent over efferent fibers, there would necessarily be a gradual change of 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 thus be a slow growth in the assembly*, understanding 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 gradiation 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 properties 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 periods as great as a year following operation (Senden, 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.