Basic Neural Mechanisms in Behavior

K. S. Lashley (1930)
Behavior Research Fund, Chicago

First published in Psychological Review, 37, 1-24
(These articles are reproduced with permission from
Christopher D. Green's 'Classics and the History of Psychology')
doi : 10.5214/ans.0972.7531.2009.160409

Among the systems and points of view which comprise our efforts to formulate a science of psychology, the proposition upon which there seems to be most nearly a general agreement is that the final explanation of behavior or of mental processes is to be sought in the physiological activity of the body and, in particular, in the properties of the nervous system. The tendency to seek all causal relations of behavior in brain processes is characteristic of the recent development of psychology in America. Most of our text-books begin with an exposition of the structure of the brain and imply that this lays a foundation for a later understanding of behavior. It is rare that a discussion of any psychological problem avoids some reference to the neural substratum, and the development of elaborate neurological theories to 'explain' the phenomena in every field of psychology is becoming increasingly fashionable.

In reading this literature I have been impressed chiefly by its futility. The chapter on the nervous system seems to provide an excuse for pictures in an otherwise dry and monotonous text. That it has any other function is not clear; there may be cursory references to it in later chapters on instinct and habit, but where the problems of psychology become complex and interesting, the nervous system is dispensed with. In more technical treatises the neurological explanations are made up mostly of assumptions concerning the properties of the nerve cell which have no counterpart in physiological experiment. Thus we find the superiority of distributed over concentrated practice seriously 'explained' by the 'fact' that successive passage of neural impulses over a synapse reduces its resistance least when the impulses come in quick succession.

There is no direct evidence for any function of the anatomical synapse: there is no evidence that synapses vary in resistance, or that, if they do, the resistance is altered by the passage of the nerve impulse. If the explanation is to be given in terms of established facts, as it must be, then it is limited to the following form: the superiority of distributed practice is due to the discontinuity of the neurons, the polarity of conduction, the fact of learning, and the superiority of distributed practice.

This is a typical case of the neurological explanations to be found in our psychological literature. With such conditions prevailing, it seems time to examine critically the relations between psychology and neurology and to attempt an evaluation of current notions concerning the mechanisms of the brain.

INADEQUACY OF CURRENT THEORIES

The starting point for our attempts to account for behavior in terms of nervous processes has been either the cerebral localization of functions or the theory that all nervous integration is patterned after the spinal reflex. I need scarcely point out the difficulties encountered by the older doctrine of cerebral localization. It expresses the fact that destruction of definite areas results in definite symptoms and the probable inference that these different parts have diverse functions, but it has given us no insight into the manner in which the areas or centers exercise their functions or the way in which they influence one another. It is only, by applying psychological conceptions like that of association, or by turning to the theory of reflexes that the doctrine of localization is made to express the dynamic relations of behavior.

The extension of the theory of reflex conduction, first derived from studies of the spinal cord, to problems of cerebral function provided a welcome addition to the psychophysical doctrine of localization. It gave a clear interpretation of localized areas as relay points or centers along the course of the reflex arc and seemed to explain the functional relations of the areas. However, the theory has not worked well in application to the details of behavior. To understand the difficulties we should have clearly in mind the form and limitations of the theory. It states that the mechanism of cerebral function is essentially the same as that of the spinal reflexes, involving the conduction of nerve impulses from the sense organs over definite, restricted paths to the effectors. The performance of a habit, whether of speech or of manipulative movement, is determined by the existence of definite connections between a limited number of nerve cells, which are always functional in that habit. The model for the theory is a telephone system. Just as two instruments can be connected only by certain wires, so the sense organs and muscles concerned in any act are connected by nerve fibers specialized for that act.

Perhaps few neurologists would agree to such a bare statement. They point to the incalculable number of nerve cells, the interplay of inhibition and facilitation, and suggest that in so complex a system there are limitless possibilities. But the fact remains that the essential feature of the reflex theory is the assumption that individual neurons are specialized for particular functions. The explanatory value of the theory rests upon this point alone, and no amount of hypothetical elaboration of connections alters the basic assumption.

Both the doctrines of localization and of conditioned reflexes imply the correspondence of structural and functional units - the specialization of minute areas or of single cells for definite limited functions. Recent experimental and clinical evidence seems to show that there is no such correspondence, and thus to present fatal difficulties to both theories. I shall sketch the main lines of this evidence, then turn to a consideration of other possible mechanisms.

ANALYSIS OF THE ADEQUATE STIMULUS

The notion of the reflex arc was developed in studies of spinal preparations in which protopathic stimuli or muscle tensions are the chief sources of excitation. Under these simple conditions something like a point for point correspondence between receptor cells and muscle groups could be demonstrated, as in the case of the scratch reflex.

We first attempted the extension of this conception to instinctive behavior, on the assumption that the adequate stimulus to nursing, to the recognition of the mate or young, to the recognition of the nest site, to sexual excitement might be expressed in terms of the excitation of such and such receptor cells. This proved to be a vain hope. The adequate stimulus in such cases may be described in terms of a pattern having definite proportions but always, within wide limits, it is a matter of indifference to what receptor cells this pattern is applied.

A survey of various types of behavior shows that this is an almost universal attribute of the adequate stimuli.[2] It is most obvious in pattern vision and can be demonstrated in animals with a rather primitive cortex. I have recently improved the technique for study of vision in the rat so that habits of pattern vision may be established in 20 or 30 trials. It is thus easy to test the equivalence of stimuli under conditions where previous associations are ruled out. Not only do we find transposition as Köhler has described it for chimpanzees, but even more striking equivalencies. An animal trained to discriminate patterns of solid white on a black ground is undisturbed by reversal of the brightness relations, by substitution of outlines for the solid figures, or even by partial outlines which retain some of the proportions of the original figures.

In many cases it is clear that the equivalent stimuli involve none of the retinal elements which were activated during learning. Here we have a situation where a habit is formed by the activation of one set of receptors and executed immediately upon stimulation of an entirely different and unpracticed group. The equivalence of stimuli is not due to the excitation of common nervous elements. The equivalent patterns have in common only ratios of intensity or of proportion in the spacial distribution of excited points. I might multiply examples of this sort indefinitely, but the studies of the Gestalt psychologists leave little doubt that such a condition is the rule for all stimuli with which we deal in the study of behavior.

ANALYSIS OF REACTIONS

Turning to motor activity, we are confronted by an identical problem. If we train an animal in a maze and observe carefully his subsequent errorless running, we find little identity of movement in successive trials. He gallops through in one trial, in another shuffles along, sniffing at the cover of the box. If we injure his cerebellum, he may roll through the maze. He follows the correct path with every variety of twist and posture, so that we cannot identify a single movement as characteristic of the habit.[3]

I have earlier reported cases of the direct adaptive use in the performance of motor habits of limbs which were paralyzed throughout training and whose motor paths consequently could not have been exercised during training.[4] It is not helpful to say that previously formed general habits are utilized in such performances, for the preexisting habits have not been associated with the new situation and the problem of the spontaneous association of the new patterns remains unsolved.

The problem of equivalence of motor responses has been less studied than that of equivalence of stimuli, but the phenomenon seems to be equally common. Activities ranging from the building of characteristic nests by birds to the so-called purposive activities of man show the absence of stereotyped movements in the attainment of a predetermined goal. The most familiar and most striking example is that of grammatical form in speech. Once we learn a new word, we use it in correct grammatical relations in limitless combinations with other words, without having to form new associations for each new setting.

It is only in certain acts of skill that stereotyped movements are recognizable and the uniformity of these is a result of long practice. We seem forced to conclude that the same motor elements are not necessarily used in the learning and performance of motor habits and that motor elements can be utilized directly when no specific associations have been formed with them.

PLASTICITY IN CENTRAL ORGANIZATION

Studies of the central nervous system give a similar picture. The functions are relatively independent of the structural elements. I can only cite a few of the lines of evidence, but sufficient, I believe, to establish the point.

First with respect to the specificity of conduction paths. The final motor neurons have been studied by Weiss.[5] He grafted additional limbs on salamanders, cutting the nerve which supplied the original limb so that the regenerating fibers came to innervate both the original and the new limb. The two limbs innervated by the same nerve showed synchronization of movements in corresponding muscle groups. Histological examination showed that the axons of the original nerve had branched so that the muscles of the two limbs were supplied by fibers from the same axons. There is no selective outgrowth of regenerating fibers and the branches of the same axon do not necessarily go to corresponding muscles. It seems, then, that the coördination of the two limbs is not a function of the particular fibers which innervate each muscle, but is due to some property of the nerve impulse such that the same fiber can selectively elicit either of two antagonistic movements. These experiments are still the subject of controversy, but the objections raised against the results are not particularly impressive and, though they may raise some doubt on this conclusion, they certainly do not establish the specificity of the axon. The results of Weiss are in harmony with many facts revealed by the study of the central nervous system.

In work with injuries to the spinal cord Miss Ball and I[6] have found that orientation of the rat in the maze is undisturbed by interruption in the cervical cord of either the pyramidial, rubrospinal, or any other of the long descending tracts. The impulses controlling turning and threading the maze somehow get down the cord after the destruction of any half of the descending fibers. I have more recently been working with double hemisections of the cord. In these preparations one half of the cord is divided in the upper cervical region, the other half below the nucleus of the phrenic nerve, so that all the long fibers are interrupted above the motor centers for the limbs. After three months such preparations show coördinated movements in walking and are able to control the limbs for orientation in response to stimuli applied to the head. The control is established in spite of the permanent interruption of all the long spinal paths.

We have also been accumulating evidence upon the functions of the projection and association tracts of the cerebrum in the rat. The data are not yet complete, but it seems fairly certain that the interruption of the projection fibers to a part of a functional area produces far less pronounced symptoms than destruction of the cortical area supplied by those fibers. We have now a large number of cases in which linear lesions sever the connections between the different anatomical areas of the cortex or divide the association fibers within single areas. It is rare that any symptoms can be detected in such cases, unless there is involved a considerable destruction of cortical tissue. The most capable animal that I have studied was one in which the cortex and underlying association fibers had been divided throughout the length of each hemisphere. His I.Q., based on ten tests, was 309.

In higher forms there is evidence for a somewhat greater specificity of long tracts in the central nervous system, but even in man the evidence is unequivocal only for the pyramidal system, which we have reason to believe is a part of the postural system and not especially concerned in the higher integrative functions of the brain, and for sensory paths of the cord. Although I would not venture the opinion that the association tracts of the cerebrum are a skeletal structure, there is certainly no direct evidence for the existence in them of any sharply defined reflex paths whose interruption results in the loss of isolated elementary functions.

What is the evidence that the cortex itself contains the definite specialized synapses which are demanded by the reflex theory? The data from extirpation experiments are somewhat ambiguous, but taken as a whole, fairly conclusive. Small lesions either produce no symptoms or very transient ones, so that it is clear that the mechanisms for habits are not closely grouped within small areas. When larger areas are involved, there are usually amnesias for many activities. Some of our experiments show that the degree of amnesia is proportional to the extent of injury and, within wide limits, independent of the location of the injury. This may mean that the cells differentiated for the habits are widely and uniformly scattered, or that there are no especially differentiated cells. After injuries to the brain, the rate of formation of some habits is directly proportional to the extent of injury and independent of the position within any part of the cortex. This shows that the rate of learning is not dependent upon the properties of individual cells, but is somehow a function of the total mass of tissue. Rate of change in individual synapses does not express the facts of learning unless we postulate some means by which the capacity for change in any cell is modified by the activity of all the other cells of the cortex. Finally, when such habits have been formed after brain injury, their retention correlates with the amount of functional tissue. This can be interpreted only as evidence that memory is not a function of individual cells, but is a property of the total mass of tissue.[7]

The reflex theory is not helpful for an understanding of such facts, nor do they seem consistent with it. If we consider the whole reaction, from sense-organ to effector, the impossibility of a theory of specialized intercellular connections becomes apparent. Let us analyze a visual reaction, for here the anatomical localization of paths seems best established. The observations of Marie and Chatelain,[8] and of Holmes and Lister[9] suggest a detailed projection of the retina upon the cortex, the macula represented in the posterior calcarine region, and successive radial zones along the borders of the fissure. (I am not sure that this interpretation is correct. Poppelreuter[10] has pointed out that the forms of scotoma are not as varied as the manifold shapes of lesion should lead us to expect, and that all the forms of scotoma can be interpreted as radiating or converging disturbances of the functional balance within the entire area. I have observations of a migraine scotoma in which the blind area retained a characteristic shape but drifted from the macula to the periphery of the visual field in the course of half an hour). But granting a cortical retina, the problem of integration is only moved back a step. I have cited evidence to show that the retinal cells used in the formation of a habit need not be excited in order to reinstate the habitual response. This must be equally true, then, for the cortical retina. The same cells may not be twice called upon to perform the same function. They may be in a fixed anatomical relation to the retina, but the functional organization plays over them just as the pattern of letters plays over the bank of lamps in an electric sign.

We find then at the point of projection on the cortex a variable pattern shifting over a fixed anatomical substratum. How can this elicit a response from a definite set of motor cells? It can not do so by excitation over definite association paths, for there is evidence against the existence of such paths and, besides, there are no fixed points of origin for them. Nor is it certain that there are any fixed motor points. We have found in studies of the motor cortex that a point which will elicit a primary movement of the fingers on one day may, a week later, produce a movement of the shoulder and at another time even movements of the face.[11] And the motor cortex, with its somewhat definite localization, is probably not concerned in habitual activity, anyway.

There does not seem to be a possibility of a constant anatomical localization at any point from receptor surface to effectors. Somehow the motor system must be sensitized to respond to the sensory patterns, but the phenomena cannot be expressed in terms of definite anatomical connections. This is the fundamental problem of neural integration and must serve as the starting point for any adequate theory of cerebral function.

THE DOCTRINE OF CIRCULAR REFLEXES

An essential element to the reflex theory as applied to psychological problems is the doctrine that all the effects of stimulation are immediately observable in the motor systems. The James-Lange theory of emotion, the idea that mental attitudes are an expression of bodily postures or 'sets,' the theory that instincts and serial habits are chains of sensory-motor activity, the doctrine that implicit speech or gesture forms the basis of thinking: these are all expressions of the belief that the nervous system serves merely for the rapid switching and conduction of impulses from receptor to effector, without long-continued intraneural sequences of activity. This notion has been attractive, as offering a possibility of direct objective study of mental activity, but attempts to verify it experimentally have given disappointingly negative results.

The problem of emotion is still in such confusion that one can draw no conclusions with confidence, but the accumulation of evidence upon the variability of expressive reactions and the repeated failure to find any consistent correlations between bodily changes and either exciting situations or reported subjective states lends little support to the visceral theory.

On the question of maintained attitude or set we have some recent evidence which seems significant. Studying the influence of bodily posture upon the movements elicited by stimulation of the motor cortex Dr. Jacobsen and I mapped the motor area and selected for study a point giving extension of the fingers. We changed the posture of the limbs, head, and body of the preparation, stimulated muscles and nerve points electrically and in other ways sought to alter the conditions of peripheral stimulation. The excitability of the point was unaltered by this treatment and the same movement was elicited at five-minute intervals for two hours. We then altered the excitability of the point by stimulation of another distant point, changing the primary movement from extension to flexion. This new primary movement persisted for 55 minutes in spite of repeated changes in the posture of the animal, than reverted spontaneously to the original movement of extension. The experiment suggests that the pattern of organization of the motor cortex can be altered by central excitation and that the altered condition can be maintained for long periods without reinforcement from peripheral organs. It seems to fulfill the conditions for demonstration of a centrally maintained attitude.

Miss Ball and I have tested the effects on serial habits of sectioning the afferent paths of the cord, together with removal of all external directive clues after the animal is oriented in the starting box. Under these conditions the habits are run off without disturbance. With external and internal sensory cues eliminated it seems that the series of acts must be controlled by some wholly central mechanism.

The work of Thorson on tongue-movements[12] and unpublished observations on eye-movements during thinking, together with reports of the recovery of speech with use of an artificial larynx, oppose the doctrine of the completed reflex and point to some continued intraneural process as the basis of thinking. The weight of evidence, I believe, favors the view that in emotion, in all persistence of attitudes, in all serial activity there are continuously maintained central processes which, if they become intense, may irradiate to motor centers and produce expressive movements, implicit speech, and the like. The pattern of irradiation varies from subject to subject according to chance variation in the excitability of the motor or vegetative nervous systems, and the peripheral activities are not an essential condition for the maintenance of the central processes.

I have devoted so much time to criticism of the reflex theory of behavior because it seems to be deeply rooted in our thinking and to have had an important influence in the development of almost every phase of psychology. It has been valuable in counteracting certain trends toward vitalism and mysticism, but I believe that it is now becoming an obstacle rather than a help to progress. In the youth of a science there is virtue in simplifying the problems so that some sort of decisive experiments may be formulated, but there is a danger that oversimplification will later blind us to important problems. In the study of cerebral functions we seem to have reached a point where the reflex theory is no longer profitable either for the formulation of problems or for an understanding of the phenomena of integration. And if it is not serviceable here, it can scarcely be of greater value for an understanding of the phenomena of behavior.

THE ALTERNATIVE TO THE REFLEX THEORY

What is the alternative to the doctrine of the specialization of nervous elements for definite reactions? It is possible that the modes of organization in the brain are not less numerous and diverse than the types of behavior to which they give rise. We have little direct evidence as to the nature of these central processes, but can deduce some laws from the effects of cerebral injury which may point the way to the significant investigations of the future.

DYNAMIC ASPECTS OF LOCALIZATION

Specialization of functions in the cerebral cortex is an indisputable fact, but we have yet to find an adequate interpretation of it. We have asked, Where are psychological functions localized in the brain? and have gained a meaningless answer. We should ask, How do specialized areas produce the details of behavior with which they are associated: what are the functional relationships between the different parts and how are they maintained?

VARIABLE DEGREES OF LOCALIZATION

If we survey the disturbances produced by brain injuries in a wide range of activities we are forced to the conclusion that the accuracy of localization or the degree of specialization varies greatly. Definitely limited defects appear in the visual and tactile and to a lesser extent in the motor fields after limited lesions to the calcarine, postcentral and precentral gyri. In other sensory spheres and in all the more elaborate organizations of behavior, there is little evidence for an equal fineness of differentiation. The visual cortex probably represents the maximum of specialization of small units. In the somesthetic field there is also a cortical projection, but less finely differentiated. In other functions we find every degree of specialization up to the limit where all parts of the cortex participate equally in the same function. The latter is apparently the condition for the maze habit in the rat. Destruction of any part of the cortex produces a partial loss of the habit and equal amounts of destruction produce equal amounts of loss, regardless of locus within the cortex.

An area which is highly specialized for one function may play a more generalized rôle in another. The habit of brightness discrimination in the rat is abolished by injury to the area striata, and by injury to no other part of the cortex. Here is a clear case of specialization. But the maze habit is abolished by destruction of this same area or of any other of equal size. Is it because the maze habit is dependent on vision? No, for blinding trained animals does not affect the habit, whereas destruction of the area striata abolishes the habit in animals which were blind during training. The deterioration does not differ in any observable way from that following lesions to other parts of the brain.

Except in projection areas there is no evidence for ana- tomical specialization within the general areas of specialization. Thus in the aphasias showing predominantly a loss of naming ability or of memory for words there is not a selective effect upon memories for specific words, but a general difficulty of recall which embraces all words of a functional group.

The evidence on localization suggests that where the relations of stimuli in space are of importance for behavior, there exists in the cortex a spacial distribution of points corresponding to the sensory surfaces, but that for all other functions a similar spacial arrangement is lacking. In terms of the reflex theory such a spacial arrangement has little meaning, but in terms of the hypothesis to which I am leading it is of prime importance.

Functional Levels of Organizations. - Turning to the dynamics of localization, we find that loss or partial loss of functions may find expression in various ways. In some cases it seems that the fundamental organization for a function has been very little disturbed but that the ease of arousal is markedly altered. Thus in monkeys and probably in man, the severity of cerebral paralysis varies somewhat with the current emotional state, and during great excitement the power of voluntary movement may be temporarily restored. The paralysis seems to consist of a greater or lesser difficulty in initiating movements, whose organization is undisturbed[13] The emotional facilitation can restore the capacity for movement. It clearly does not supply the specific integrations but only makes the final common paths more excitable or increases the intensity of activity in the integrating mechanisms. Here we have the energy for activity supplied, as it were, from an outside source. Some of the symptoms of cerebellar ataxia and the conditions described as pure motor aphasia present the same sort of picture. I have used the term energy here with reluctance, for the notion of nervous energy has led to many extravagant speculations, yet it seems impossible to deal with such phenomena except in terms of some general factor which may influence the ease of functioning of a system of activities without changing the specific integrations.

In another type of quantitative reduction in efficiency, the integrative mechanism itself seems affected, but without disintegration into elementary functions. In the rat, destruction of the occipital cortex abolishes the habit of brightness discrimination in the Yerkes box. Brightness vision is actually undisturbed, as can be demonstrated by other methods, but the association with the specific activities of the training box is disturbed. The amount of practice necessary to reestablish the association is closely proportional to the extent of lesion. Here we are dealing with some function akin to the memory trace of Ebbinghaus. Just as the memory trace grows weaker with the passage of time, so it is weakened by cerebral injury. Recall may be impossible, yet a persisting trace of the former training may be demonstrated by the "savings method." The strength of the trace is determined by the quantity of tissue. The efficiency of performance is determined by the summated action of all parts of the area.

We cannot here use the accepted theories of summation or reinforcement, for these theories are based upon the phase relations of nerve impulses and we seem to be dealing with a continuous summation. It seems impossible to express the facts in other terms than simple variation in energy.

The Relative Fragility of Functions. - I have pointed out that the same area may be involved in quite diverse functions. These may be differently affected by lesions. Thus the habit of threading a complex maze is seriously disturbed by destruction of any part of the cortex, provided the lesion involves more than 15 per cent. The habit of a simpler maze is unaffected by lesions involving as much as 50 per cent of the cortex. We do not have an extensive series of tests with different mazes, but a comparison of Cameron's cases[14] with my own indicates that there is a definite relationship between the complexity of the maze habit and the minimal lesion which will produce a measurable disturbance of it.

Dr. Jacobsen has similar evidence from experiments with monkeys[15] Animals were trained to open a series of simple puzzle boxes and also a box in which the latches of the simple boxes were combined. After destruction of the frontal or parietal lobes, the ability to open the simple boxes was retained, but the same latches in combination could not be opened.

We have similar results on the limits of training for both the rat and monkey. Simple problems may be learned at almost normal rate after brain injuries; complex problems are learned slowly, if at all. Further, the greater the brain injury, the greater is the disproportion between the learning of simple and complex habits. In such cases the brain injuries seem to limit the complexity of organization which may be acquired, without disturbing the capacity for the simple acts which are mediated by the same areas.

The clinical literature presents many comparable cases. The aphasic patient may be able to understand and execute simple commands and yet be unable to grasp the same instructions when several are given at the same time. Head cites numerous instances of this limitation in complexity of organization[16] In pattern vision, the stages through which the patient passes during recovery from cortical blindness form a series with respect to complexity. It seems probable that the great fragility of color vision and of the perception of depth is due to the high degree of organization required for these functions, rather than to their separate localization in the cortex.

Both the animal experiments and the clinical material point to the conclusion that a given area may function at different levels of complexity, and lesions may limit the complex functions without disturbing the simpler ones. Further, we cannot ascribe this limitation to the loss of some necessary elementary functions or to disturbances of nutrition or to shock, for it has been shown in some cases to be solely a function of the quantity of tissue. In this respect the limitation of complexity seems to accord with Spearman's view[17] that intelligence is a function of some undifferentiated nervous energy.

.........To be continued in next issue

References

1. Address of the President of the American Psychological Association before the Ninth International Congress of Psychology at New Haven, September 4, 1929.
2. Goldstein K, Die Topik der Grosshirnrinde in ihrer klinischen Bedeutung. Dtsch Zsch f Nervenheilk 1923; 77: 7-124.
3. Lashley KS and McCarthy DA. The survival of the maze habit after cerebellar injuries, J Comp Psychol 1926; 6: 423-433.
4. Lashley KS. The theory that synaptic resistance is reduced by the passage of the nerve impulse, Psychol Rev 1924; 31: 369-375.
5. Weiss P. Die Funktion transplantierter Amphibienextremitäten. Arch f mik Anat 1924; 52: 645-672.
6. Lashley KS and Ball J. Spinal conduction and kinæsthetic sensitivity in the maze habit. J Comp Psychol 1929; 9: 70-106.
7. Lashley KS. Brain mechanisms and intelligence. Chicago Univ Press 1929, pp. 186.
8. Marie P. C. Chatelin, Les troubles visuels dus aux lésions des voies optiques intracérébrales et de la sphère visuelle corticale dans les blessures du crâne par coup de feu. Rev neurol 1914-15; 28: 882-925.
9. Holmes G and Lister WT. Disturbances of vision from cerebral lesions with special reference to the macula. Brain 1916; 39: 34-73.
10. Poppelreuter W. Die psychischen Schädungen durch Lopfschuss. Leipzig 1917.
11. Lashley KS. Temporal variation in the function of the gyrus precentralis in primates. Amer J Physiol 1923; 65: 585-602.
12. Thorson AM. The relation of tongue movements to internal speech. J Exper Psychol 1925; 8: 1-32.
13. Minkowski M. Etude physiologique des circonvolutions rolandique et pariétal. Arch Suisse de neurol et psychiat 1917; 1: 389-459.
14. Cameron N. Cerebral destruction in its relations to maze learning. Psychol. Monog 1928; 39, (No. 1): 1-68.
15. To be reported soon in *J Comp Neur*.
16. Head H. Aphasia and kindred disorders of speech. New York 1926.
17. Spearman C. The abilities of man. New York 1927.