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MASS ACTION IN CEREBRAL FUNCTION[1]

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INTRODUCTION

IN the field of neurophysiology no fact is more firmly established than the functional differentiation of various parts of the cerebral cortex. We are removed from Flourens by nearly 75 years of intensive anatomical research which has settled beyond question the histological diversity of the cortical fields and of their connections with subcortical nuclei. A wealth of physiological and clinical evidence accords with the anatomical findings and proves the association of at least the majority of the cortical fields with special functions. No one to-day can seriously believe that the different parts of the cerebral cortex all have the same functions or can entertain for a moment the proposition of Hermann that because the mind is a unit the brain must also act as a unit.

Yet the problems of localization and of cerebral physiology are far from solved by the demonstration of the anatomical diversity of the cortical fields and of consistent symptoms following the destruction of each. From the practical view of diagnosis there are still problems of the fineness of localization, of the types of functions which are localizable, of the significance of individual variations, and the so-called negative cases.

Symptoms involving purely sensory or motor defects have the most certain diagnostic value: a monoplegia or a limited zone of cutaneous anesthesia are surely indicative of a focal lesion whose position may sometimes be predicted within a few centimeters. But a defect of color vision, a disability in reading, an

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[1] Lecture delivered before the Harvey Society, New York, November 20, 1930.

apraxia, or a syntactical aphasia are indicative only of disturbance somewhere within a rather widely extended field, while a simple defect of judgment or change in personality, even when of unquestionable organic origin, can not now be associated with any cerebral locus.

Except in a few instances, it is impossible to predict with any certainty from the locus or severity of a lesion what will be the course of recovery or of deterioration. Until we know more of the nature of depression, diaschisis and the factors of spontaneous recovery, until we know the limitations of reeducation and of vicarious functioning, therapeutic methods must be somewhat uncertain.

From the standpoint of an adequate cerebral physiology also, the classical concept of cerebral localization is of limited value, because of its static character and its failure to provide any answer to the question of how the specialized parts of the cortex interact to produce the integration evident in thought and behavior. The problem here is one of the dynamic relations of the diverse parts of the cortex, whether they be cells or cortical fields. The diversification of parts is a fact of fundamental importance, but it is only one of many which must be discovered before we can form any adequate conception of cerebral organization.

There is a very close relation between the problems of cerebral physiology and of psychology and each science must be broad enough to accommodate itself to the facts revealed by the other. We must agree with Henschen that *a priori* psychological analysis has contributed little to our understanding of cerebral function and has often confused the issues, especially in the study of the aphasias. On the other hand, simplified physiological theories of neural integration have hampered the development of psychology and have contributed to a futile sort of psychological atomism. Cerebral physiology and to a large extent the concepts of psychology must be built up from the empirical basis of anatomical and clinical facts, but we can not ignore the problems of integration which are presented by normal behavior. We must not forget, as many of the diagrammatists seem to have done, that cerebral areas somehow do perform the functions which are lost when they are destroyed, and that we must account not only for defects, but for normal activity as well.

DIVERSE FUNCTIONS OF THE VISUAL AREA

To-night I wish to report the results of our attempts to analyze the function of a single field of the cerebral cortex, the visual area. This, of all cortical fields, presents the most definite evidence for fine structural and functional differentiation and at the same time reveals activities which are among the most difficult to fit into any schema of localization. Our approach has been essentially that of Goltz, Munk, Luciani and Hitzig, but with the use of quantitative methods of studying behavior and of analyzing the anatomical findings which were not available to earlier workers.

The experimental work is largely limited to the rat. The advantages of this material are the simplicity of the animal's behavior, its steadiness in activity under the motivation of hunger and its availability in large numbers. The last has made possible the use of experimental and statistical controls which would have been impossible with any larger form.

The danger of generalization from the rat to man is obvious. Our program includes the use of this material only as a means of outlining problems and gaining clues which must in every case be retested by experiments with primates and by comparison with clinical evidence. So far as we have been able to carry out such controls, there has been a clear agreement between the results with the rat and with primates. Actually these lower animals seem to show the beginnings of every human mental trait and I have come to doubt that the evolution of mammals has introduced any change in the fundamental organization or mechanism of cerebral activity. The enormous differences are in degree rather than in kind.

Our present knowledge of the structure of the visual system in the rat is still imperfect. There is a definite visual area of the unistriate type occupying a position on the dorsal convexity of the occipital pole.[2] Volkmann[3] also distinguishes a smaller medial area as homologous with the visual association area of higher forms. From the retina there is a large crossed tract which terminates in the lateral geniculate body and in the superior colliculus. The small uncrossed bundle in Marchi preparations seems to terminate in the lateral geniculate body. From the thalamus a large tract ascends to the striate area. Its exact origin and termination have not been worked out. From the cortical area a large tract descends to the internal capsule, a second smaller tract passes to the callosum. Except for this commissural tract there are no long transcortical fibers from the area.

A word as to methods: for testing vision in animals we have used two types of apparatus. The more familiar type is the discrimination box in which two different visual stimuli are displayed at the ends of

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[2] Fortuyn, *Arch. Neurol. and Psychiat.* (London), 6, 221, 1914.

[3] Anat. Anz., Erganzungsheft, 61, 234, 1926.

two adjacent passages, so as to be visible to the animal from the common entrance. The stimuli are alternated in position irregularly on the right or left in successive trials and the animal is trained to go to one of the stimuli for food, in whichever passage it may be displayed. The discrimination box has served for the study of thresholds for brightness but is unreliable for tests of detail vision. For the latter the method is modified so that the animal must jump from a distance against one of two cardboard doors bearing different visual patterns, one of which may be knocked aside to allow him to reach food. This method works rapidly for tests of pattern vision. For testing learning and memory, latch boxes or mazes through which the animal must learn the direct path are used. By these means we may study the limits of visual capacity, such as thresholds, acuity or ability to distinguish complex patterns or to identify pictures with objects, and also the rate at which visual and other associations are formed and the loss of visual and other memories as a result of brain injuries.

The neurological variables which may be studied quantitatively are the locus of lesion with respect to the cortical fields, the absolute extent of destruction, and the time interval between operation and tests. The destruction of cerebral tissue may precede training in tests of the influence of lesions upon the limits of capacity, or may follow in tests of postoperative amnesia. In all cases reported the lesions have been carefully reconstructed from measurements of serial sections.

SPECIFICITY OF FUNCTION IN PATTERN VISION

The rat has a fairly good capacity for pattern vision.[4] He is very near-sighted, the far point being at about 8 cm. His acuity is about 1/60 that of man. With these limitations his vision does not seem essentially different from that of man. He can readily distinguish position, distance, brightness, relative size and complex outlines. If he learns to differentiate between two solid figures he recognizes immediately outlines of them differing in size, or even partial outlines. That is, essentially, he can recognize pictures of objects. He can learn to pick out a variable from a constant part of a complex pattern.

For test of cerebral localization we have made a general survey of the whole cortex, destroying parts, symmetrical on the two hemispheres, with knife or thermocautery.[5] With recovery from the operation the animals were put through a number of tests of vision, then brought to necropsy, and the lesions reconstructed from serial sections. Our tests have now covered about 50 cases with lesions in all parts of the cortex.

Fig. 1 shows the results of the experiments. Destruction of a small region in the lateral part of the visual area completely and permanently abolishes the capacity for detail vision. The animals still jump readily, distinguish the position and distance of single objects and can distinguish between two objects of different size or brightness, but they fail on every test which requires the discrimination of patterns. The defect in vision is no greater if the whole of the occipital cortex is destroyed. Vision for discrete objects, their relative position and brightness is retained after lesions including any part of the cortex.

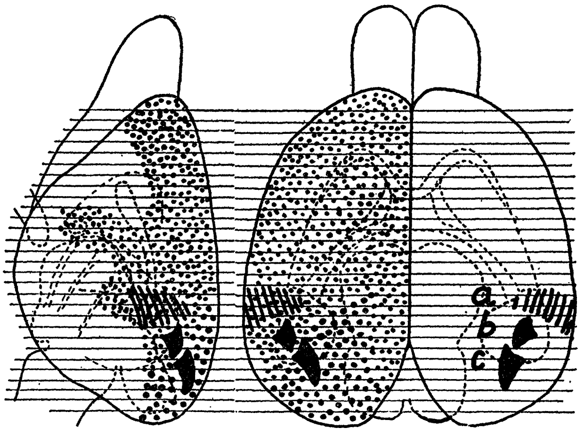

FIG. 1. Visual areas of the cerebral cortex of the rat. The stippled areas represent the regions destroyed by operations, symmetrical on the two hemispheres, without disturbance of the capacity for pattern vision. The coarser stippling indicates the position of the anatomical *area striata*. Destruction of the cortex at b or c on both hemispheres completely abolishes vision for patterns but leaves the capacity to distinguish the position and relative brightness of gross objects, as does the total destruction of the visual cortex. Interruption of the projection fibers at a abolishes all vision for objects but leaves the capacity to distinguish differences in the intensity of light.

We have here, then, a definite restriction of the function of pattern vision to one small area of the cortex. There is no interference with detail vision in our tests after destruction of any other part of the cortex. In all the tests the animals were normal in all visual tests after the destruction of the motor, or somesthetic, or auditory, or of the greater part of the visual area, including the supposed visual association area.

The localization of the visual field is as precise and as absolute as it is in man. Within the field there may be an accurate representation of different parts of the retina. We have no method for mapping

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[4] Lashley, *Jour. Genet. Psychol.*, 37, 453, 1930.

[5] *Jour. Comp. Neurol.*, in press.

scotomata in the rat and hence can only infer finer specialization from data on higher animals. Since any other part of the cortex than the visual area can be destroyed without disturbance in our tests, it seems clear that the simple motor habits involved do not require the formation of any special transcortical associations with other fields, but are carried out merely by the coordinated activity of the visual cortex and the subcortical visual and motor nuclei. There are indications in the anatomical studies of Poljak and in the experimental work of Jacobsen and myself that this may be the condition in primates also.

If we destroy the optic radiations as they leave the internal capsule, we have a different picture. In all tests requiring jumping the animals behave exactly as do others deprived of their eyes. Yet they are not completely blind. In a simpler situation, the discrimination box, where they must run past the stimulus, they can still distinguish light from darkness and can form habits based upon this discrimination as readily as do normal animals.

All this seems clear enough and consistent with a thoroughgoing theory of localization. Pattern vision is a function of the visual cortex, the identification of the position of single objects is possible through the action of projection fibers to other than the anatomical visual area, and the discrimination of the intensity of lights may be wholly a function of the thalamus and midbrain.

LIMITED SPECIFICITY IN DISCRIMINATION OF BRIGHTNESS

But when we study further the function of brightness vision, that is, the discrimination of light from darkness, in the discrimination box, a difficulty immediately arises for so simple an interpretation. In the absence of the entire visual cortex the habit of light-darkness discrimination is formed at normal rate. We have data now on 113 cases with injuries in the visual cortex and 89 normal controls. There is no significant difference in their rates of learning. But if we train normal animals and then subject them to operation in the visual area, they lose the habit. If the entire visual cortex is destroyed, the postoperative amnesia is complete. The animals can relearn to make the discrimination, but require just as much practice as they did for initial learning before operation. This loss can not be accounted for upon the grounds of cortical blindness, for the animals relearn at normal rate and are obviously not blind. It is not a simple sensory defect, for if it were, the defect should show in the initial learning of animals which lack the visual areas, and these are normal in the formation of this habit. We can only describe the loss as an amnesia, in contradistinction to sensory defect.

The next step in our analysis is to determine the effects of partial destruction of the visual area upon the habit based on brightness discrimination. If only a part of the visual cortex is destroyed, the animal shows amnesia but relearns more rapidly than when all is destroyed. It makes no difference what part of the visual area is involved, the effect is the same. In a series of cases the relationship between the extent of lesion and the degree of amnesia is very close, whereas the locus within the visual cortex is immaterial.[6]

These results can be interpreted, I believe, only by assuming that the visual cortex acts upon the lower visual nuclei in such a way as to facilitate their activities in the performance of functions which they nevertheless can carry out in the absence of the visual cortex. There is still localization, in the sense that the visual area and no other part of the cortex exerts this effect, but the effect is independent of that finer localization which is essential for pattern vision. For this function there are not subordinate localizations of functions within the visual cortex, but the area acts as a unit, each part providing energy or facilitation of the lower centers, as does each other part.

We have closely comparable data for the latch-box. This is learned at normal rate by animals lacking any half of the cortex, yet when it is learned by normal animals an amnesia follows the destruction of any part of the frontal region.

Turning now to another aspect of the visual problem: Testing for threshold of discrimination between different intensities of light we find that animals lacking the visual cortex have on the average a higher threshold than normals and that they are slower in learning to choose between lights whose difference is well above their threshold.[7] The disability here does not seem to be one of actual visual defect, so much as of instability of the visual reactions. The animals seem to fail the tests near threshold values, not so much through inability to discriminate as through inability to react consistently enough to reach the high standard of accuracy required as evidence of discrimination. One or two cases, entirely lacking the visual cortex, showed a threshold as low as normal, after long training.

In this case the visual cortex seems, speaking figuratively, to keep the subcortical visual centers on the job, possibly by a facilitation which maintains an increased responsiveness or increased excitability

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[6] Lashley, *Jour. Comp. Neurol.*, 41, 1, 1926.

[7] Lashley, *Jour. Genet. Psychol.*, 37, 461, 1930.

without contributing to the specific integrations involved.

This sort of general facilitating action of one center upon another is not unfamiliar in neurological theory, since it forms the basis of Monakow's theory of diaschisis. Our results for the visual field depart from Monakow's theory first in that the functions lost from withdrawal of facilitation do not recover spontaneously, but may be reacquired by reeducation, second, in that the effect may be specific for integrations already formed in learning and need not involve the capacity to form those associations, and third, that the severity of diaschisis is proportional to the amount of tissue destroyed.

There are indications that similar conditions underlie many of the symptoms of brain lesions in higher animals and man. The cerebral paralyses give a picture which is the inverse of the preceding. We trained a group of rhesus monkeys to open puzzle boxes by turning a crank, opening a gate hasp, and the like.[8] Their retention of these problems was found to be perfect after three months. They were then subjected to operation involving removal of both precentral gyri. The resultant diplegia cleared up after three months to permit of fairly deft movements of the hands. During the recovery period the animals did not see the problem boxes. About four months after the operations their ability to open the boxes was tested. All solved the latches practically at once, without random activity, and used the same methods that they had employed before the destruction of the motor cortex.

In this case we seem to be dealing with two mechanisms which have somewhat the relations of the visual cortex and lateral geniculate bodies. Destruction of the motor cortex does not abolish the specific integrations involved in the manipulative habits, but only withdraws a certain necessary mass of facilitation from the total system. This facilitative control is recovered (largely by reeducation, as shown by Odin and Franz[9]), and with the recovery, the organized patterns of movement may again become functional without training. It seems that we must be dealing with two mechanisms, one of which is responsible for the integration of movements in the habit, the other for a facilitation of final common paths to make them responsive to the excitations from the former. Both are almost certainly cortical functions and both are essential conditions of the normal activity.

I suspect that some of the phenomena of motor aphasia or anarthria fall in this class of disturbances. From experiments upon movements of the tongue in speech and in silent thinking we have evidence that overt speech includes both a general increase in the tonus of the vocal organs and also a specific innervation which determines the patterns. The tonic innervation holds the tongue forward in the speaking position and in this position even during silent thinking there are sometimes involuntary movements of speech. With relaxation the tongue drops to the back of the mouth and the involuntary movements disappear. The suggestion is that Broca's area or Marie's quadrilateral provide a tonic innervation which makes the lower motor centers responsive to weaker impulses descending from the temporal and angular gyri.

A survey of the literature on motor aphasia which we have been making indicates that the severity and duration of symptoms after injury to the left third frontal convolution are proportional to the extent of destruction and independent of locus within this region. The assumption that the function of Broca's area and of Marie's quadrilateral is a non-specific facilitation of lower centers may help to clear up some of the difficulties of localization which have been encountered in study of these areas.

NON-VISUAL FUNCTIONS OF THE "VISUAL" CORTEX

In the experiments which I have thus far discussed the function of the visual cortex is visual, although exercised in various ways. We have now to consider what seems to be an entirely different function of this same area. If we subject animals to cerebral lesions and then train them in a fairly complex maze, we find that they learn much more slowly than do normal controls.[10] This slowness of learning appears, no matter in which part of the cortex the lesion occurs. The degree of retardation seems proportionate to the amount of tissue destroyed, irrespective of the locus of injury.

Fig. 2 illustrates the relationship between the extent of destruction of the cortex and the amount of training necessary to perfect the habit. The correlation is 0.84 which is as high as that between any two measures of learning ability or of intelligence that we have. The lesions included in the graph cover all parts of the neocortex and, within the limits of their statistical reliability, the data indicate that equal amounts of destruction in the motor, somesthetic, auditory or visual areas are attended by equal amounts of retardation in learning.

If normal animals are first trained in this habit and then are subjected to cerebral lesions they show loss of the habit irrespective of the locus of the lesion and in proportion to the extent of destruction. That is,

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[8] Lashley, *Arch. of Neurol. and Psychiat.*, 12, 249, 1924.

[9] *Psychobiol.*, 1, 33, 1917.

[10] Lashley, "Brain Mechanisms and Intelligence," Chicago, 1929.

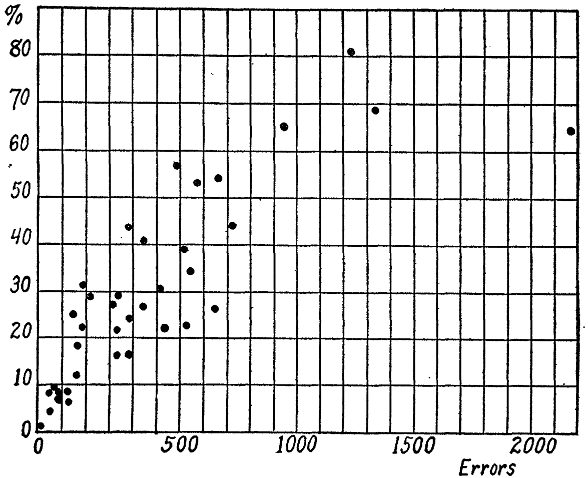

FIG. 2. The relation between the extent of cerebral lesion and the amount of practice necessary for the learning of a complex maze. The ordinates represent the percentage of the neocortex destroyed; the abscissae, the number of errors made during training to a constant standard of efficiency. The graph includes about an equal number of cases with lesions in each of the chief cytoarchitectural areas. (*After Lashley, 1929.*)

every part of the cortex plays a part both in learning and in retention.

Like other cerebral fields, the visual cortex contributes to the learning and retention of this habit of threading the maze. But in this its function is apparently not primarily visual, as shown by experiments like the following: We trained a group of animals until they reached a certain standard of accuracy in running the maze. We then blinded them by enucleation of the eyes. This produced no inaccuracy of performance. In the first tests after blinding the animals make errorless records in the maze. Another set of animals was blinded before training, and learned the maze without seeing it. They were then subjected to lesions within the visual cortex. They showed a loss of the habit—a loss as great as the loss in seeing animals after the same type of lesion. They were retrained and required an amount of practice for relearning which seemed proportionate to the extent of injury within the visual field.

These facts, that blindness does not interfere with efficiency in this activity whereas destruction of the visual cortex in blind animals does seriously interfere, indicate that in this habit the visual cortex has some important non-visual function. Since in this same manner each of the cortical fields seems to contribute equally to the maze habit, we have been led to the conclusion that the various parts of the cerebral cortex exert a mutual influence, each contributing, perhaps by some sort of facilitation, to the functional efficiency of the others, or to the functional efficiency of some lower centers which all the cortical areas supply in common.

Whether there are cortical integrative processes in the maze habit or only a general facilitation of subcortical integrative processes is not certain. There are indications, however, that the same non-specific quantitative relations obtain in the integrative activities of subcortical nuclei. Thus, injuries to the lateral geniculate bodies result in a slowing of the formation of habits based upon brightness discrimination and within our limited data the retardation seems roughly proportional to the amount of destruction.

We have been inclined to interpret this mass facilitation in the cortex as somehow underlying the intelligent activities of the organism. When we compare the effect of the same amounts of cerebral destruction upon functions of varying difficulty we find that, in general, the more complicated the function the more it is affected by a given amount of destruction. For the simplest habit (a maze with one blind alley) the extensive lesions produce little more retardation than the slight, whereas for a more complex maze the retardation is almost proportional to the square of the extent of destruction. For still more difficult functions, which seem to require of the animal something analogous to reasoning in man, Dr. Maier finds that small lesions produce more marked effects and a destruction of 20 per cent. of the cortex may entirely abolish the function.[11]

These experiments give us a picture of a cortical field in which for some activities the efficiency of performance is proportional to the quantity of tissue available and is independent of any known specialization of the parts. Moreover it seems fairly established that the complex functions of such a field may be seriously hampered by lesions which leave the simpler functions almost undisturbed.

We have further evidence on this latter point from the experiments of Dr. Carlyle Jacobsen with monkeys.[12] The animals were taught to open various latch-boxes and their rate of learning measured for simple and for combination locks. They were then subjected to destruction of the frontal lobes of the cerebrum. After the operation they remembered quite well the problem boxes on which there was a single latch to be opened and learned new problems of this type at normal rate, but when the boxes combined several of the same simple latches, the animals were unable to recall the solutions and were much retarded in relearning them. Destruction of the frontal areas generally left the capacity to deal with simple situations undisturbed yet interfered greatly

[11] *Jour. Comp. Neurol.*, in press.

[12] *Jour. Comp. Neurol.*, in press.

with the ability to handle combinations of these same simple situations. Something of the same effect appeared after destruction of the parietal association areas.

Many symptoms in man are suggestive of the same type of organization. In organic dementia we may find the ability to execute each of several simple tasks unimpaired, along with an inability to follow instructions which call for the successive execution of these same tasks. In the agrammatic aphasia of Pick there may be little or no amnesia for words but an inability to combine the words in grammatical sequence.

Boumann and Gruenbaum[13] have defined the more general defect of aphasia as an inability to keep in mind the several elements of a complex situation and at the same time to manipulate the elements in thought. With our animals there is an inability to deal with problems which present several elements at once.

ANATOMICAL AND FUNCTIONAL LEVELS OF ORGANIZATION

Our results with mazes of different complexity point to a functioning of cortical fields at different levels of organization. We have already clear evidence that different levels of complexity of organization may correspond to different anatomical levels (as in the case of brightness vision mediated by the thalamus and pattern vision by the cortex) and this has been accepted as a characteristic neural arrangement; the existence of hierarchies of organization in different anatomical loci. But our results with activities which probably represent the highest levels of integration of which the animal is capable suggest that for these activities there is not separate anatomical localization, but that anatomically the mechanism for the highest integrations is coextensive with the mechanisms for simpler ones. The simplification of behavior after cerebral lesions is in these cases not the result of destruction of a super-associative center, but of destruction of tissue anywhere within the cortex. The limiting condition for efficiency is the surface area or mass of cortical tissue and not the specific anatomical relations of the parts.

This is essentially the problem of the relative fragility of functions. Where one function is eliminated by a lesion which leaves other similar functions intact, it has been customary to postulate their separate localization, as in the case of color and pattern vision in man. But it seems also possible that such differential fragility may result from disturbances within a single area and that one limiting condition of the complexity of integration is the amount of available tissue.

NON-SPECIFICITY OF HISTOLOGICAL ELEMENTS

Thus far I have dealt with gross relations of parts of the visual and other areas. What of the finer relations and the specialization of the histological elements? The separate projection of parts of the retina upon the calcarine region of higher mammals is well established and there may well be a point-to-point correspondence between the ganglion cells of the retina and the cells of the stripe of Vicq d'Azyr of the striate area. But does this mean a determination of the reaction through the specialization of these cells?

We blindfold one eye of a rat and train him to react to one of two visual stimuli. We then transfer the bandage to the other eye and test his ability to react to the stimuli with the untrained eye. The response is perfect without training. Here we have a reaction learned with one set of receptors and executed immediately with a different set. Of course the corresponding cells of the two retinae may excite the same cells of the central system, so the experiment is not crucial.

But, if we train the animal to jump to a white erect triangle and to avoid an inverted one and then confront him for the first time with outlines of these figures in smaller size, he will choose the correct outline without error. Here none of the retinal cells and consequently none of the cells of the projection area which were stimulated by the contour of the figures during training are similarly stimulated by the contours of the test figures. The habit is formed by one set of cells and immediately executed by another. It seems clear in this case that the reaction is not dependent upon the particular cells stimulated. Within limits, any cells of the visual projection area if excited in specific relation to one another can mediate the performance of an habitual act, regardless of whether they have been similarly excited during learning.

I have not time to present other examples of this same condition, but I believe that in every reaction, above the level of a spinal reflex to protopathic stimulation, the adequate stimulus is a pattern which is effective when applied anywhere upon the sensory surface and the motor response involves an equally variable grouping of motor neurons. That is, no two repetitions of the same instinctive or habitual act need involve the same pathways of conduction through the central nervous system, or the same nerve fibers excited in the same way.

Professor Herrick[14] has recently defined two types of localization.

> First, a known localization of stable structural elements whose functions also are known, and, second, a localiza-

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[13] *Zsch. f. d. ges. Neurol. u. Psychiat.*, 96, 481, 1925.

[14] Proc. Nat. Acad. Sci., 16, 643, 1930.

tion of fields within which various recurring patterns of performance or schemata, are known to be fabricated and within which inhibition, modification, or conditioning of these patterns takes place.

The second of these is the only type of localization which can be defined in the adult organism. In it the strict localization even of reflex units seems impossible. For spinal reflexes the conception of simple point-to-point connections is proving inadequate and giving place to a less specific and more dynamic interpretation. Subliminal effects, overlap of fields of influence of neurons, and the like have led Sherrington[15] to the conclusion:

> Though trains of impulses are the sole reactions which enter and leave the central nervous system, nervous impulses are not the sole reactions functioning within that system. States of excitement which can sum together, and states of inhibition which can sum together, and states which represent the algebraic summation of these two, are among the central reactions. The motoneurone lies at a focus of interplay of these reactions and its motor unit gives their net upshot always expressed in terms of motor impulses and contraction.

Thus, wherever we turn in the study of the central nervous processes we are confronted by the same problem. Just as our data show that for some functions it is massed relations of facilitation and not the specialization of separate parts which is responsible for efficiency of performance, so within the finely localized areas the ultimate element of organization can not be the single cell and its specific anatomical connections but is the interplay of organized patterns of excitation in which *relative* position and mass of excitation play a dominant rôle.

INTERPRETATIONS

The picture of cerebral functions arising from this work is not a simple one and it is still far too early to attempt any complete account of the cerebral nervous mechanisms. The point which stands out most clearly is the fact that the laws governing the activity of cerebral areas vary according to the functions involved. For pattern vision, the spacial distribution of the visual cortex is of fundamental importance and the different parts contribute diversely to the reaction. But for brightness vision, although the cerebral visual area as a whole plays an important part, its individual parts do not have a differential function but contribute equally in some unknown way to the maintenance of the habit of discrimination. In more complex functions, such as the learning and retention of the maze habit, the visual area seems essential yet non-specific, contributing facilitation to the total neural organization yet essentially equivalent to other cortical areas. The same area may at times function as a highly differentiated system, at others as a unitary mass.

There can be little question of the facts in each case. The experimental and clinical evidence for such diverse modes of functioning seems conclusive. Our task is to find the conditions under which the different types of neural activity occur and to analyze the interactions among them. Any claim to certain knowledge of the mechanisms of cerebral function would be presumptuous, but from the known facts we may gain suggestions which will be of value in the formulation of further research and in giving at least a vague notion of how some of the simpler cerebral integrations are brought about.

The structural elements, projection and association fibers, determine the main lines of conduction and limit the regions of major excitation. The afferent projection fibers transmit their excitations to diverse parts of the cortex and, at least in the case of vision, kinesthesis and touch, reproduce on the cortex something of the spacial attributes of the stimulus. We have seen that the adequate stimulus in such cases is not the specific cells activated, but the pattern of excitation which may shift over the sensory surface and likewise over the cortical field. In such a pattern the relative intensity, distance of separation and frequency of excitations seem to be the only constant factors, determining in turn the direction and steepness of gradient of electrical and chemical processes within the system.

These patterns are certainly not transmitted in duplicate beyond the sensory projection surfaces. They give rise to specific patterns of movement but these do not reproduce the sensory pattern and we can not assume a direct connection between them over preformed and specifically differentiated paths. Yet we must assume that specific sensitivity of motor patterns to sensory patterns does exist.

We know that many of the details of motor integration are organized within the motor nuclei themselves and that the cortex does little more than activate these motor patterns, determining which of several integrated systems will respond. There are indications, further, from embryologic studies that, in their growth and early functioning, the motor systems are sensitive to the general direction of polarization of the body. It is not inconceivable that the cortex determines the reactions of lower centers, not by activating individual cells or cell groups but by determining the general direction and degree of polarization of the motor centers. There are suggestions of this in the results of

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[15] Proc. Roy. Soc., 1929, B, 105, 332.

Weiss[16] with transplanted limbs of Amblystoma, in the ready shift from one limb to another in the execution of semi-skilled movements,[17] in the tendency of athetoid movements in hemiplegia to reproduce the general direction and rhythm of movements on the sound side and the like. It is as though the influences descending from the higher centers tend to call out the same direction and rhythm of movement from any motor center which they reach, regardless of whether specific associations with that center have been previously formed or not.

Turning back to patterns of excitation in the cortex, we find them projected from sensory surfaces to cortical fields in such a way that the *relative* position and intensity of activity in the various parts alone are stable. The chief interconnection of the cortical fields is through arcuate fibers which form a veritable feltwork and give little suggestion of any specific projection of one field upon another. Similarly, there are indications that the adaptive control of lower centers is in part by way of the diffusely distributed extrapyramidal fibers and the arcuate fibers of the cord. As examples, I have already cited the fact that pattern vision is mediated by the efferent fibers from the visual cortex only and may mention as an additional example that we now have animals which have learned semi-skilled acts after the section in the cervical region of all the long ascending and descending fiber tracts of the cord.

Such facts suggest that integrations above the level of the sensory fields may be in part a matter of diffusion of impulses through a fairly homogeneous matrix. In the case of any physical analogy which we can draw with this condition, such as chemical diffusion, wave motion, or the spread of timed volleys of nerve impulses through a homogeneous cellular network, there will arise within the matrix a definite and stable interference pattern in which, although the transmitted energy is in a state of constant flux, the lines or points of maximal and minimal summation and interference maintain a constant position. Such an interference pattern would in turn be capable of exciting specific groups of efferent cells and determining a definite pattern of motor innervation, or, if less specific, of altering the general polarization of lower centers and so modifying their functional activity.

This is a possible mechanism which would permit of some such degree of plasticity as the results of our experiments seem to require. It would allow of the excitation of definite motor patterns by sensory patterns of entirely different form, without the intervention of specific neural connections. It is, of course, still purely hypothetical, but it is an hypothesis which is in harmony both with the facts of localization and with the apparently contradictory facts of equipotentiality and mass action of neural tissue.

This is only a part of the whole story. There is evidence that the activity of every nervous center is conditioned by a variety of factors. Its general level of excitability varies and a low threshold or state of tonic activity is maintained by excitation from many sources. Steadiness and continuity of discharge are likewise maintained by agencies other than those which are concerned in the specific patterning of reactions. In addition, there are many indications of a preliminary integration or setting of nervous mechanisms which may then maintain a potential organization until activated by excitation from other sources. Thus it is possible, by brief stimulation of a motor point in the cortex, to modify the motor responses elicited on stimulation of distant points and such an altered excitability or motor set may persist for an hour or more before it gives way to the original pattern of response.

These processes, which we may term priming, steadying and preparatory adjustment, seem to be subthreshold for overt activity: some sort of partial activation or tonic excitation of centers. In addition to these, there are activating mechanisms which perhaps impose additional patterns of integration or perhaps only raise the tonic excitation above the threshold for motor expression.

In the motor field we have evidence that many structures participate in activity without actually determining the specific pattern of skilled movement. Thus the cerebellum, the striate complex, and probably the motor cortex contribute to the readiness and steadiness of response, though their destruction does not abolish the pattern of skilled movement. We do not know the prevalence of such facilitating activities, but our work suggests that the whole cerebral cortex, perhaps every part of the nervous system may, in addition to its specific functions, exercise such general facilitating effects upon other parts. This may account in some measure for the quantitative relations found between extent of lesion and efficiency of performance, the extent of facilitation depending upon the number of cells activated.

The apparent limitation of possible complexity of function by the available amount of tissue seems to raise a different problem. The limitation can not be due to restriction of the number of possible conditioned reflex paths available, for the animal can form as separate habits all of the elements which can not be combined in one reaction. The difficulty is in dealing with a number of elements at the same time.

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[16] *Jour. Comp. Neurol.*, 40, 241, 1926.

[17] Lashley, *Psychol. Rev.*, 31, 369, 1924.

A possible clue to the situation here comes from experimental biology. In the regeneration of hydroids the number of tentacles regenerated is correlated with the size of the regenerating mass of tissue. Child has shown that each separate structure develops from a nodal point in the system of gradients within the mass. He has suggested that there is a minimal distance of separation for the development of diverse gradients limiting the number of structures which can be formed by a small piece of tissue.

In the simultaneous integration of a number of activities the cortex must present a large number of nodal points of excitation and it is possible that the number and distribution of these within the association areas is determined not by specific connections but by the polarization effects of the various localized excitations within the sensory projection fields. In such a case the number of nodes of excitation and of diverse gradient fields would be definitely limited by the factor of separation and of available mass of tissue.

I have indulged in this highly speculative discussion primarily to show that the notion of decentralization or of cerebral function without absolute anatomical localization need not involve an abandonment of recognized physiological principles or a denial of the known facts of localization. The chief advantage of the strict theories of localization has been their definiteness and comprehensibility. Those of us who have felt the inadequacy of such theories have had to fall back upon expressions like mass action, stress patterns, dynamic effects, melodies of movement, vigilance or nervous energy; all highly metaphorical and unproductive of experimental problems. Yet the facts demand something of this sort. The evidence seems conclusive that in various cortical functions there is every degree of specialization from a limited point-to-point correspondence of cells to a condition of absolute non-specificity. Not only is there diversity in the modes of action of different parts of the cortex but a single area, highly specialized and differentiated for one activity may be wholly undifferentiated with respect to another in which it also participates. We have not a choice between a theory of localization and a theory of decentralization, but must develop a wider view which recognizes the importance and interdependence of both modes of integration.

The principles to which I have appealed in the foregoing sketch, the production of gradients of activity and their influence upon organic processes, the development of stable patterns of interference in the transmission of different forces through a homogeneous matrix, are as well established in biological thought as are the principles of conduction within the nerve fiber or the interaction of nervous impulses within a spinal center. They will be capable of test with further improvement in our methods of studying the electrical phenomena of nerve conduction. Whether these specific suggestions prove right or wrong, they indicate, I believe, the direction to which we must turn our investigations, if we are to develop an adequate cerebral physiology.

OBITUARY

IGNATIUS URBAN

THE really capable and active systematic botanists of the world are so woefully few that the removal of a single one vacates a niche that usually remains unfilled. Such losses seem to have been more than normally frequent during the past year. In 1930 the world was deprived of Dr. Adolf Engler, dean of German botanists. Only a fortnight ago news was received of the death in Copenhagen of Dr. C. H. Ostenfeld. On January 7 the Botanical Garden and Museum of Berlin-Dahlem was robbed by death of another of its most brilliant men, Dr. Ignatius Urban.

Dr. Urban's special field was the flora of the West Indies, to which he devoted forty busy years. He found the Antillean flora in chaos, and left it in order. It is safe to say that for no other part of America is there available in convenient form so well ordered a mass of exact information as exists for the West Indian flora in the nine volumes of the "Symbolae Antillanae."

Those volumes by no means represent the whole extent of Dr. Urban's work, for he published many papers in German and Swedish journals. The "Symbolae" contain a vast amount of ably presented information regarding West Indian plants—descriptions of new species, monographs of genera and critical notes upon nomenclature, besides chapters upon botanical history and bibliography and plant geography. One of Dr. Urban's greatest services to science was his careful solution of the status of many vague names appearing in early literature but long neglected. He did more than any other man to place nomenclature of tropical American botany upon a solid and sane basis. His floras of Porto Rico and Hispaniola, which constitute two volumes of the "Symbolae," must be consulted almost daily by students of tropical plants.

Few botanists of all time have accomplished so much and that so uniformly well. Whoever consults Dr. Urban's own pages of the "Symbolae" will be