



The Topography of the Brain Jean-Martin Charcot

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As late as twenty years ago physiologists and clinicians agreed in declaring the cortex of the brain to be functionally, homogeneous. Flourens's experiments had decisively negated Gall's very ingenious but purely hypothetical conception, and any effort to prove localization would, at that day, have seemed like a reversion to a system already tried and condemned. It was freely admitted that, from experiments made on pigeons, one might infer the mode of brain functionment in man. Medicine was under the yoke of the then dominant teachings of physiology, nor was there so much as a thought of reaction. Clinical observers, indeed, had long before known that motor troubles consequent on a lesion of the brain imply localization of such lesion in the hemisphere on the side opposite to that paralyzed; but that was then the sum of the topographical diagnosis.

Broca, in 1863, showed that the impairment of the power of articulate speech, which he calls aphemia, is connected with a brain affection that is always localized in a clearly circumscribed region of the left hemisphere. At first the fact was called in question. When proofs had been multiplied in its favor men contented them selves with simply admitting it, little noting that this very definite localization was a first attack on Flourens's doctrine, which must now undergo revision. But the topographical anatomy of the cerebral convolutions was then too little known to enable one to "find his bearings" on the surface of the brain, and the reaction against Flourens's ideas would at that time have met with insurmountable obstacles.

The thorough researches of Leuret and Gratiolet, and of their successors, Ecker, Broca, Gromier, by making us acquainted with the morphology of the external surface of the brain, removed these first anatomical difficulties. The experiments of Fritsch and Hitzig

in Germany, in 1870, and shortly afterward those of Ferrier, in England, modified the ideas which prevailed. They showed, on the one hand, that the gray matter of the brain is not incapable of excitation, as had been supposed; that electric excitation of this gray matter calls forth motor reactions; on the other hand, they prove -an important point- that the effects produced differ according to the part of the cortex that is excited. From that date, properly speaking, began researches into motor localizations in the brain. Since then such researches have been prosecuted in two directions; for while the physiologists reproduced, with various results, the experiments of Fritsch, of Hitzig, and of Ferrier, the clinicians were also at work. And I may be permitted to say that the researches in this latter direction began in France, and that I have had some share in them. My first researches, made jointly with Professor Pitres, then my interne, were the starting-point for studies that have been for ten years prosecuted with remarkable activity in France, where a great number of investigators have contributed their share of facts, in England by Jackson and Ferrier, in Germany by Nothnagel.

On considering how far we have advanced in the study of localization in the cortex while puruing these two paths experimentation on animals and anatomoclinical observation of man-one is struck with the fact that while among clinicians there is perfect agreement, at least on the essential points, among the physiologists there is marked disagreement. The divergence of views is due, perhaps, mainly to the fact that the experimenters have cared less about determining the relations between a given affection and a lesion of one or another part of the cortex, than about discovering the inner mechanism of the relation between the two. That which, in the eyes of the clinician, whose thoughts are ever of diagnostics, is the point of capital importance, thus becomes an acces-

sory datum for the experimenter, who thinks more about theory. Now, the theories that have been advanced, one after another, to account for the phenomena observed to follow excitation or destruction of the cortex are as numerous as they are uncertain. Take the fundamental facts alleged by Fritsch, Hitzig, and later by Ferrier, viz., that excitation of certain parts of the gray matter determines localized convulsions; that, on the contrary, ablation of these parts produces paralysis; these facts, while admitted in their general tenor, have been interpreted in very different ways. According to some writers, Ferrier, for instance, the cortex comprises true motor centers; others, as Hitzig, Fritsch, Schiif, Munk, hold the excitable points to be sensitive centers, excitation of which determines movement in virtue of a sort of reflex action, while destruction of these centers produces paralysis through loss of conscious sensibility. Many physiologists, as Tamburini, Luciani, and Seppeli, hold this "excitable zone" to be both motor and sensitive. Vulpian held that it is simply the place of convergence or influences emanating from all the other parts of the encephalon, and that it has no activity of its own. Finally, according to Dr. Brown-Squard, the excitable points of the cortex have neither motor nor sensitivo-sensorial functions; excitation applied to them does but traverse them, passing on to organs of movement situated lower down; their destruction does not act by suppression, but by irritation at a distance. Such is the theory of dynamogenic, or inhibitory, action at a distance. As has been justly remarked by François Franck:

"It must be admitted that all the interpretations now conceivable are absolutely provisional; nay, it were rash and illogical to believe that any question whatever touching the mechanism of the brain, and in particular this one, has been definitely settled."

Certainly the study of these questions is by no means void of interest, and the clinician may not stand indifferent toward the efforts made to determine the instrumental process whereby a given lesion of the cortex produces such or such a convulsion, such or such a paralysis. But he must not forget that this determination is a secondary task; and, in any case, theoretic considerations cannot fairly be suffered to call in question the positive teachings of anatomo-clinical observation.

Then, it is to be borne in mind that experimentation with animals that are nearest to man, still more with those far removed from man on the zoological scale, cannot, however faultless its technique, however definite its results, solve finally the problems raised by the pathology of the human brain. In brain it is, above all, that we differ from animals. That organ attains in man a degree of development and of perfection not reached in any other species. Its functions become complex, while at the same time its morphology undergoes important modifi-

cations. Now, it is perfectly clear that as regards questions of localization morphological details are of the first importance. As for functions, even if we take account only of those common to men and animals, they are not performed in all in the same way. The higher an organism stands in the animal scale, the more strictly are the purely reflex functions subordinated to the functions of the higher centers. A decapitated frog performs with its legs co-ordinated automatic movements; not so a decapitated dog. In the dog, brain lesions, even of considerable extent, produce only incomplete paralysis, often passing away, while in man the like lesions cause incurable functional troubles. These examples are enough to show that, particularly as regards brain functions, the utmost reserve is necessary in drawing inferences from animals to man. The results of experimentation, however ingenious, however skillfully conducted, can give only presumptions more or less strong, but never absolute demonstration.

Hence, the only really decisive data touching the cerebral pathology of man are, in my opinion, those developed according to the principles of the anatomo-clinical method. That method consists in ever confronting the functional disorders observed during life with the lesions discovered and carefully located after death. This is the method that enabled Laennec to throw light on the difficult subject of diagnosing pulmonary affections, and it has also materially helped the diagnosis of diseases of the liver, kidneys, and spinal cord. To it, I may justly say, do we owe whatever definite knowledge we have of brain pathology. As for the localization of certain cerebral functions, here this method is not only the best, but the only one that can be employed. What light, for instance, could experimentation have thrown upon the question as to the seat of the functions of speech-functions which are special to man?

No doubt observations restricted to the domain of man, and deprived of the powerful lever of experimentation, may, at first sight, seem doomed to play a subordinate and inconspicuous role, but that is so only in appearance. As I had occasion to write, some twelve years ago:

"The conditions of a truly spontaneous experiment in man are presented every day in pathological circumstances. To profit by them, we have only to learn to comply with the necessities of a situation no doubt very different in many respects from that which experiment purposely brings about in the animal, but which is not always more complex. If it is true that observations made, in the light of physiology, on man in disease, usually require more time, more patience, than corresponding studies of animals under experiment; if it is true that in man the conditions of the phenomena cannot be, as they are in the laboratory, either modified or

reproduced at the will of the observer ; so, too, is it true that disease often determines in the body of the patient lesions more strictly limited to one organ or one tissue; in other words, more systematic and more compatible with persistence of life, and with the integrity of functions not directly concerned ; consequently they lend themselves better to methodical and protracted analysis than do mutilations produced in animals by even the most skillful physiologist.” (Revue Scientifique, Nov. 11, 1876).

But in order to be employed with profit, anatomo-clinical observations must not be gathered at haphazard. On the contrary, they have to be tested methodically and classified according to certain rules that I have taken pains to define from the beginning of my studies on cerebral localizations. It is plain, for instance, as I have elsewhere said, that irritative lesions are a very different thing from destructive lesions; nor must we confound lesions newly produced (accompanied, as they almost necessarily are, by phenomena having their seat either near by or at a distance) with old lesions, in which the morbid process being, in a measure, at an end, is now clinically represented only by the mere inactivity of the parts that have been diseased or destroyed. Just because these distinctions have not been sufficiently noted by authors, most of the old observations are useless as regards the question of localizations. When we add that in these observations the designation of the lesioned convolutions is commonly vague and lacking in precision, it is seen that such data give but little light. Hence, as Nothnagel justly says of the many cases of brain lesions that are recorded, having been collected in the course of ages, unfortunately only a very few can bear criticism or warrant conclusions. But while we must distrust the old data, we may well accept those which in these latter years have been carefully collected by authors who understand the exigencies of the anatomo-clinical method. By taking their stand upon these clinicians have been able to formulate the propositions to which I am now to call attention, and which form the groundwork of topographical diagnosis in the pathology of the brain. In this summary statement I intend absolutely to avoid reference to facts that are not perfectly established, for instance, those bearing on sensitive localizations; I will mention only such as may be regarded as firmly and definitively settled.

When a brain lesion, whether cortical or of any other sort, is accompanied by motor paralysis, the seat of the paralysis is always on the side opposite to that of the lesion. This proposition is universally accepted by physicians, and in clinics it may be said to have the force of a law. I would not have referred to this elementary truth had not some physiologists in these latter days ventured to call it in question, or at least sought to lessen its diagnostic value by citing in opposition to it alleged contradictory facts. But when these observations are subjected

to criticism, it is easily seen that they have no such force as they have been credited with. In the record of a clinical case there may easily occur an error as to the side affected “right” instead of “left” and vice versa. To some such lapsus, as I can show, is to be referred the apparent anomalousness of some, at least, of the facts alleged in opposition to the law of chiasm; hence, in my opinion, no weight is to be attached to cases, even modern cases, in which authors have not taken pains to insist explicitly on this anomaly.

And even were it proved that in a few cases, that are surely exceptional, the paralysis and the lesion producing it are both on the same side of the body, it would be necessary, before drawing an inference from such facts, to make sure that they are not to be explained by an abnormal arrangement of the nerve conductors. This calls for a few words of explanation. We know that the centrifugal, or motor, fibers proceeding from the brain decussate, those of the right crossing those of the left side at a certain point in their course before they enter, first, the spinal cord and then the muscles. This decussation takes place at the level of the Pyramids of the bulb it gives the reason why a lesion of the right side of the brain produces paralysis of the left side of the body, and vice versa. But normally time decussation is incomplete for though most of the motor fibers that constitute the pyramid pass into the spinal cord of the opposite side, some of them take the straight course and enter the anterior spinal cord of the same side. These fibers are, under ordinary conditions, very few in number. But it may happen, in case of an exception anatomic arrangement, that the fibers taking the straight course are more numerous than those which cross. Of course in such a case a lesion of the brain would be explained by an anomaly of structure, but that would give no ground of inference against the law of decussation, which still holds good in the immense majority of cases. Even granting, therefore -a thing that has yet to be proved- that this law is subject to exceptions, these exceptions are so rare that, as far as clinical diagnosis is concerned, we may leave them out of account, and hold it for a well-established truth that a paralysis of cerebral origin presupposes a lesion of the hemisphere of the opposite side. If I have mentioned incidentally the objections brought against a proposition long since become classic in nerve pathology, it was in order to show the danger of accepting theories, for so a man may be led to question the most indisputable clinical facts.

Turn we now to the study of disorders consequent on lesions of the cortex. Hemiplegia, i. e., paralysis of the movements concerned with the face and with the two members of one side of the body, is often the consequence of these lesions. But not all lesions of the cortex are accompanied by hemiplegia; they are so only when certain conditions as to the extent of the lesion, and par-

ticularly as to its seat, are present.

Now, anatomico-clinical research shows that even considerable alterations in the gray matter of the brain cause no motor disturbance when they are localized in certain regions. These regions include the sphenoidal, occipital, and inferior parietal lobes of the pli courbe and of the insula, the orbital lobule, and the anterior portion of the first, second, and third frontal convolutions. These portions of the brain may be destroyed by softening, may be compressed or irritated by tumors, by bony splinters, or by effusion of blood, without in the least affecting the motility. The case is totally different if the region destroyed is that corresponding to the two ascending frontal and parietal convolutions and the adjoining replis, viz., the paracentral lobule, the foot of the first three frontal convolutions, and of the superior and inferior parietal lobules. In such cases we always find hemiplegia of the side opposite to that of the lesion. Here, then, we have a striking contrast between the gravity of the symptoms produced by lesions of this zone and the marked harmlessness, at least the latency of effects as regards the phenomena of movement, in the case of lesions to other portions of the cortex.

This contrast has been so often noted and verified in clinics that we can have no hesitation in admitting the existence, now well established, of a motor zone in the cortex. This zone occupies, as we have seen, pretty nearly the middle portion of the external surface of each hemisphere; the region anterior or posterior to this does not, directly at least, control movements.

This fact, resulting from a careful comparison of the symptoms observed during life and of the necroscopic lesions of the cortex, is further confirmed by anatomico-clinical observations of another order. The fact is well known that a nerve fiber degenerates when separated from its trophic center, which, in the case of motor fibers, is the motor cell whence these fibers emanate. On the other hand, we know that, as a sequel of certain cerebral lesions, there is developed in the peduncles, bulb, and spinal cord a degenerescence of the centrifugal or motor nerve tubes. Turck first brought this to light in 1851. Soon afterward I verified the exactitude of this observation in my researches with Vulpian. The labors of my pupils, Bouchard, Pitres, Brissaud, in France, and those of Flechsig, in Germany, have settled the determining conditions and the topography of this degenerescence "secondary" degenerescence, as it is called. Now, not all lesions of the cortex are equally capable of producing secondary degenerescence. This special point I distinctly called attention to in one of my lectures in 1873. I attach the more importance to what I said then, because the question of cortical localizations in man had not yet been raised, and there could be no suspicion that my statement was put

forward to strengthen a theory. I said:

"Cerebral lesions en foyer, considered with respect to the seat they occupy, are not all equally capable of determining the production of consequent scleroses. Thus, among these lesions there are some which are never followed by descending sclerosis, while others are dead certain, so to speak, to produce it.

It results from my observations that extensive superficial softening, when it occupies either the occipital lobe, or the posterior portions of the temporal lobe, or the sphenoidal lobe, or, finally, the anterior regions of the frontal lobe, is not followed by consecutive fasciculated sclerosis; while such sclerosis, on the contrary, regularly appears when the foyer compromises the two ascending convolutions (ascending parietal and ascending frontal) and the contiguous parts of the parietal and frontal lobes."

Research has, during the past ten years, confirmed the exactitude of the foregoing propositions. We may, therefore, hold it as certain that secondary degenerescence is never seen except after cortical lesions; that when these lesions are in the zone which we have called the motor zone, that fact of itself suffices to prove that there is no direct relation between the motor conductors and the regions of the gray matter of the brain which we have called the latent zone, destruction of which does not cause paralytic effects.

I might cite more arguments to prove the reality of the motor zone of the cortex; in particular, I might recall the fact, demonstrated by Betz, Mierzezewski, and other authors, that its structure differs perceptibly from that of the adjoining regions, and that this zone has a mode of development peculiar to itself, as shown by Parrot. But whatever the force of these new proofs, I do not dwell upon them here, wishing to stand on the ground of clinical observation exclusively. On that ground the reality and the independence of a motor zone are universally recognized and accepted to-day.

The question now arises whether this zone is functionally homogeneous, or whether, on the contrary, it is not resolvable into distinct centers, each concerned with the movements of some special part of the body. Let us see what is to be learned on this point by the anatomico-clinical method. Motor paralyzes resulting from lesions of the cortex do not always assume the form of hemiplegia. They may, affect the face, the arm, or the leg; in that case there is "monoplegia", or, as Nothnagel terms it, "parcellary, paralysis." We must observe that monoplegia does not necessarily depend on lesion of the cortex. Besides cases of monoplegia due to hysteria there are some that are due to affections of the motor conductors at points in their course more or less distant from the

convolutions. But we, of course, have to do only with monoplegia caused by lesion of the cortex. Now can we, from the localization of a monoplegia, infer the seat of the affection which produces it? In 1883 I was led to conclude, from researches made in conjunction with Mr. Pitres, that the cortical motor centers for the two members of the opposite side are situated in the paracentral lobule and in the superior two-thirds of the ascending convolutions; that the centers for the movements of the lower part of the face are situated in the upper third of the ascending convolutions, near the fissure of Sylvius; that very likely the center for the isolated movements of the arm lies in the middle third of the ascending parietal convolution of the opposite side. Nothnagel reached these same conclusions through a close analysis of a multitude of facts, and they are confirmed by observations published since 1883. This is specially true as regards the motor center of the inferior members, the localization of which has been determined with the utmost exactitude. Sundry recent facts, particularly those, at my instance, collected by one of my pupils, Mr. G. Ballet, have, in fact, shown that the paracentral lobule, with the uppermost part of the frontal and ascending parietal convolutions, has specially to do with the motility of the femur and crus. Hence, when a case occurs of monoplegia of the inferior member referable to a lesion of the cortex, we can affirm that a lesion localized at the points mentioned is the cause.

Paralysis is not the only manifestation which enables us to diagnose a lesion of the cortex and to point out its seat. Alongside of the "deficit" symptoms, so called, must be ranged the "excitation" symptoms, which are also of the very highest diagnostic value in nervous clinics. The symptoms of this second group are manifold, and have diverse clinical significations. I will refer here only to convulsions of cortical origin, commonly known as partial epilepsy, or Jackson's epilepsy. A French author, Bravais, first described, in 1827, under the name of hemiplegic epilepsy, a variety of epileptiform convulsions that begin in one member, or on one side of the face, and which continue to be limited to one of the lateral halves of the body. Bravais did good service in isolating the clinical type, but to Hughlings Jackson, of the London Hospital, belongs the credit of having shown its significance and of having brought to light the relations between partial epilepsy and lesions of the cortex of the brain. I give a few details. Partial epilepsy consists sometimes of simple tremor, again of violent convulsions like those of true epilepsy, and producing a condition that may in a moment end in death. The general characteristic of the spasms is, that they begin in some isolated group of muscles, and are thence gradually propagated to other muscles of the same member, or of the whole body, before the patient loses consciousness. The loss of consciousness, however, is not fatal, as in true epilepsy; it may continue during the lifetime. Clinicians are now fully

agreed as to the semeiological value of partial epilepsy, and the latest observers have confirmed the fundamental propositions put forth by me in 1883, in a work in which I had as collaborateur Mr. Pitres. The following points may be regarded as fully established: In the great majority of cases partial epilepsy results from lesions of the cortex. It but seldom follows lesions of the central portions of the brain. The affections which most readily produce it are limited affections with quick and progressive evolution (neoplasm, superficial encephalitis, meningitis, whether acute or chronic). Partial epilepsy is never observed in cases of extensive lesions that suddenly overspread the whole area of the motor zone. The lesions which produce it are usually in the motor zone itself, but they may lie outside of it, provided the affection is capable of irritating the elements of the motor convolutions. Thus, then, the topography of the lesions in this case is less fixed than in the case of permanent paralysis. That is why cortical paralysis can exist either with or without epileptiform convulsions, and vice versa. The principles that should guide the clinician are as follows: When, in the intervals between attacks, the patient subject to epileptiform convulsions presents no sort of paralytic phenomena, then the lesion is in the vicinity of the motor zone of the cortex. Partial epilepsy begins either in the arm or in the leg or in the face; but we cannot fix by, an absolute rule the seat of the cerebral lesion in its relation to the way the convulsions make their appearance. Still, the epileptiform convulsions which begin in the muscles of the members are generally produced by lesions situated at the level of the upper two-thirds of the motor zone, or in its vicinity; those which begin in the muscles of the face are commonly the result of lesions occupying the inferior extremity of the motor zone, or the neighboring parts.

It is seen that, from the point of view of exact topographic diagnosis, the epileptiform convulsions have less value than the paralysis, yet they authorize us to affirm almost with certainty that they have to do with a lesion of the cortex.

The first fact clearly established in cortical localization was, as I have said, that published by Broca in 1861. That author showed that disturbance of the faculty of articulate speech, since called aphemias, motor aphasia, and logoplegia, depends on a lesion of the foot of the third left frontal convolution. Latterly, the question of affections of speech, of aphasia, has been thoroughly investigated again. A more searching and a more exact clinical analysis has shown that there is ground for thinking that there are four sorts of affections corresponding to the loss, partial or total, of one of the four processes by means of which we enter into relations with our fellow men. These four processes are speaking, writing, hearing (of words), and reading. The former two serve us in expressing and transmitting our thoughts; the other two serve us in

understanding and receiving the thoughts of others. Each of these four mental operations may be impaired, either separately or in conjunction with the others. Abolition of articulate speech is called Broca's aphasia, or motor aphasia; abolition of the power of writing is agraphia; of that of hearing words is word deafness; of that of reading, word blindness. Now, as each of these operations has its physical independence, so each has its organ, its special center in the cortex. The lesion which produces motor aphasia is not that which produces word blindness; the one on which depends word deafness is not that which causes agraphia. As yet the precise seat of the four centers cannot be fixed. As regards two of them localization may be regarded as certain; for the other two it is still hypothetical, or, at least, only probable.

Before we point out these different localizations it is important to remind the reader that the left hemisphere of the brain, to the exclusion of the right hemisphere, governs the functions of speech. This fact, glimpsed by Dax, brought clearly to view by Broca with respect to aphemia, holds good also with regard to the other forms of aphasia. Sometimes, indeed, motor aphasia has been found to result from lesion of the right hemisphere, but in such cases the patients are invariably left-handed persons, that is to say, persons in whom the right cerebral hemisphere predominates. But such cases are exceptional; apart from them the rule is, that we speak, write, read, understand words with the left brain. Nor is this surprising, when we consider that, as Gratiolet has shown, the left brain develops earlier than the right; hence, when the infant begins to understand and to utter words, it must use rather the hemisphere that is better fitted for performing these functions.

I come now to the localization of the centers. Two of them, as I have said, those the destruction of which is followed by agraphia and word blindness, have not yet been determined with absolute certainty. The observations hitherto made must be multiplied, but as far as they go they lend the highest probability to the inference that the center which presides over writing is situated at the foot of the second frontal convolution, and that the center which presides over reading occupies the inferior parietal lobule, with or without the co-operation of the lobule of the pli courbe. We have far more decisive data with regard to the seats of the other two centers. Broca's researches have proved indisputably that the center for articulate speech occupies the foot of the third frontal convolution; the observations that are brought forward to contradict this cannot stand criticism. As for the region of the cortex, lesion of which produces word deafness, that certainly, as Nothnagel held as early as 1879, occupies the first frontal convolution. An analytical comparison of the seventeen cases recorded by Seppeli justifies this conclusion.

Such are the most important and the best-grounded of the localizations discovered through the anatomo-clinical method. At first they were not received without calling forth some opposition; and though most clinicians were quick to accept these localizations, at least with regard to motility and the functions of language, there were, as a matter of course, a few who rejected them. But the apparently contradictory facts brought forward by these few opponents could not bear methodical and rigorous criticism. To-day one need but consult the principal medical journals, and in particular the publications of the Paris Anatomical Society, in order to form a just estimate of the number and the force of the data on which are based the localizations of which I have spoken. New observations are daily confirming these localizations, and these observations would surely be more numerous still, but just now the publication of facts confirmatory of the propositions we have formulated is neglected. These propositions no longer meet with any serious contradiction among clinicians. A few physiologists still call them in question, but they do so on the ground of certain purely theoretical conceptions which, as I have shown, have nothing to do with the very definite results of the anatomo-clinical method. As Vulpian justly said:

"All the progress pathology has made remains as a permanent acquisition, whatever opinion be held as to the cortical centers of cerebration. Whether these centers exist or do not exist, it is henceforth indisputable that a lesion of the posterior portion of the left third frontal convolution causes impairment of language; that a destructive lesion of the superior portion of the ascending convolutions produces paralysis of the leg of the opposite side; and that lesion of the middle parts of the same convolutions is followed by paralysis of the arm of the opposite side. No less indisputable is it that certain irritative lesions of these same parts give rise to convulsive symptoms. These facts are highly important for the clinician, and their value is entirely independent, I repeat, of all questions as to the existence of centers of motor cerebration or other centers in the gray cortex of the brain."

It is well to recall these words of a savant who was at once a great physiologist and a great clinician.

