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Practical Cerebral Localization

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Practical Cerebral Localization.

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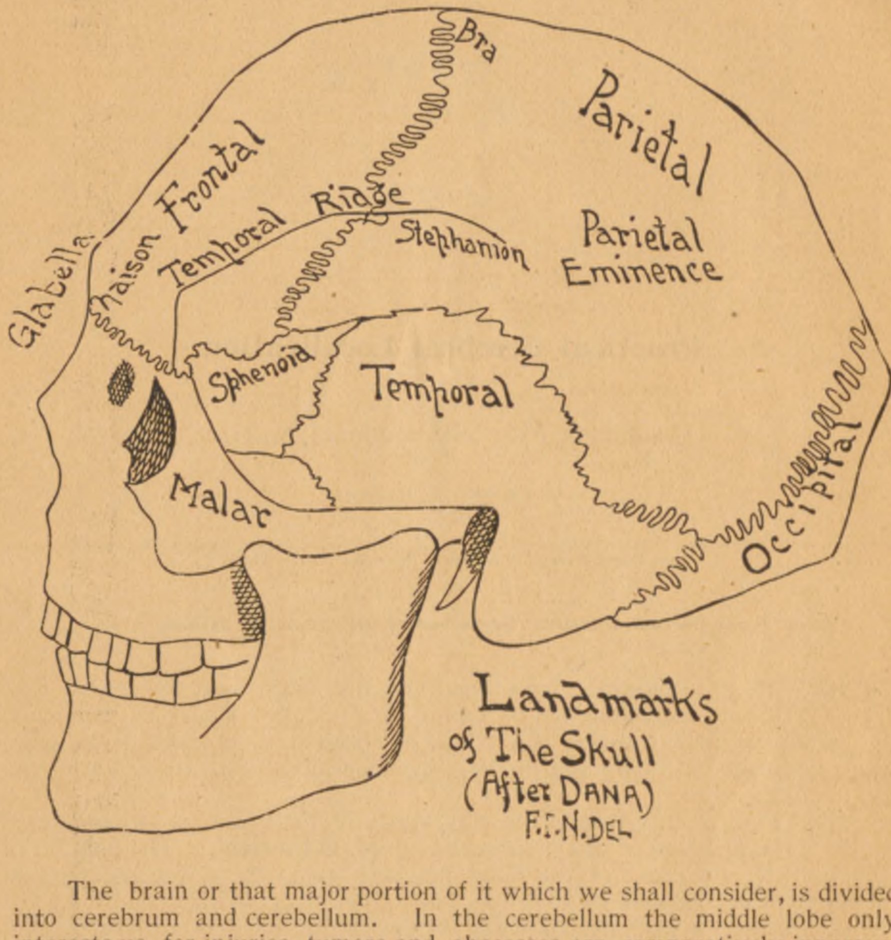
THE odd physiological views regarding the brain and nerves, as held by Hunter, and expressed by him in his principles of surgery, compared with the modern theory of cerebral localization, is an index of the great progress made all along the line of physiological investigation during the past century.

Especially is this true in the physiology of the brain, as demonstrated in the establishment of the localization of brain function. The old idea of the unity of the organ of the mind has been removed and in its stead an assignment of regional location has been given to each individualized function. Further, a strong relationship between brain cells has been discovered, whereby substitution of action in certain cells takes place when disease or accident slowly destroys the physiological functions of their fellows. Such in brief, are the two essentials of cerebral localization, but their practical use to us in removing disease would be nothing, were it not for that greatest of modern surgical principles, asepsis. The skull, so long closed to the surgeon's knife, is now opened freely because of the absence of subsequent septic dangers, when aseptic measures are thoroughly practiced. To recite the various steps leading up to the present status of localization and brain surgery, would tax your patience and consume time, so we will enter at once upon the consideration of the principles of localization and their practical application.



The brain or that major portion of it which we shall consider, is divided into cerebrum and cerebellum. In the cerebellum the middle lobe only interests us, for injuries, tumors and abscesses are comparatively innocous, unless the middle lobe is affected. The cerebrum, the seat of intelligence, is made up of two hemispheres, right and left, subdivided by fissures into lobes, and these in turn are subdivided into convolutions, which may be smaller, larger or reduplicated. The five lobes of the brain are the frontal, parietal, temporo-sphenoidal, occipital and Island of Reil, and are separated by the primary fissures. As no two brains are alike, we have difficulty in locating secondary fissures, but the primary fissures exist in common. The fissure of Sylvius separates the temporo-sphenoidal lobe from the orbital surface of the frontal lobe; its posterior limb separates the temporal from the parietal lobe. The fissure of Rolando separates the frontal and parietal lobes. The parieto-occipital fissure separates the parietal from the occipital lobe.

The secondary fissures and the convolutions are important considerations but are best spoken of under the head of topography, which we will now take up. The fissures of Sylvius and Rolando are our base-lines from which we will carry on our topographical survey, and it is therefore necessary that we first locate them with a certainty. As in land surveying, we must first determine our land-marks before proceeding in the general survey, so I will invite your attention to this drawing showing the landmarks of the skull. This is copied from Dana's admirable paper on Craniocerebral topography as it appeared in The Medical Record, January 12, 1889, and reproduced in The Journal of Nervous and Mental Diseases, of June, 1890. The Glabella-Nason Bregma, Inion, Stephanion, the principal land-marks, are here well defined. Now, having our land-marks we proceed to use them, calling to our assistance this instrument, the cyrtometer. The one I have here is Wilson's. You see it is a very simple instrument, made of flexible metal, and secured in position by use of the tape.

The broadest arm passes around the forehead, touching the glabella and external angular process, the narrow or longitudinal arm passes backward from the glabella in the middle line to the occiput. This arm is marked with two scales of figures, one scale being located in the anterior half and the other in the posterior half. A narrow strip slides on the longitudinal arm at an angle of 67° with it, extending towards the transverse arm. To use the cystometer, we first shave the scalp, apply the instrument so as to touch the two land-marks, the glabella and external angular process. Measured from the glabella backward the distance of any of the figures in the first scale, is 55.7 per cent of the distance from the glabella to the corresponding figure in the second scale. Now when the instrument is in position, if the figure 1 in posterior scale falls immediately over the inion, we place the sliding arm at figure 1, in anterior scale and we have the beginning of the fissure of Rolando, which extends at angle of 67° downward and forward. This is the direction of the upper two-thirds of the fissure, or 2¼ inches. The lower third 1¼ inches is slightly more vertical. To find the lower end more exactly, lay off a line from the stephanion to the asterion, and another from bregma to the external auditory meatus. The point of intersection will be over the lower end of the fissure. The fissure of Rolando is the same in children as in the adult.

To find the fissure of Sylvius, draw the vertical line from the stephanion to the middle of the zygoma. Draw a horizontal line from the external angular process to the highest part of the squamous suture; continue this back, gradually curving it up till it reaches the parietal eminence. The junction of the two lines will be at the beginning of the fissure of Sylvius. The vertical line indicates nearly the position of the anterior or vertical branch of the fissure, which is, however, directed a little more forward, and is about one inch in length. The posterior part of the line indicates the position of the posterior branch of the fissure. The triangular gyrus and motor-speech centre lie just anterior to the vertical branch of the fissure. The fissure of Sylvius runs nearly horizontally and lies either under or a little above the uppermost part of the parieto-squamous suture. This suture, the external orbital process, and the parietal eminence are the guiding land-marks, by the help of which the surgeon can often operate without marking down lines on the scalp. In children, according to McClellan, the fissure is higher than in the adult. To carry out our cranio-cerebral topography any further would cause a departure from our subject under consideration. To those of you who desire further information I take pleasure in referring you to the paper of Prof. Charles Dana, before mentioned. From the two base-lines we have located, we can by following his concise rules, locate all important regions of the brain cortex.

The relationship between the cranium and cortex is as follows: "Beneath the frontal bone lie nearly all the lowest frontal, 5-6 of the middle and ¾ of the upper convolutions. The temporal bone covers the temporal lobe, except its posterior fifth and anterior extremity. Less than half of the occipital lobe lies under the occipital bone. The rest of the cerebral cortex lies beneath the parietal bone" (Gowers). The boundaries of the areas of the brain have been pretty well established by physical experiments of Ferrier, Horsely, Schafer, Fritch and Hitsig, and the pathological studies of Horsely, Keen, Starr, Park, Mills and others. Especially is this true of motor regions, while vision, hearing, etc., are still in dispute. The occipital lobe is undoubtedly the seat of vision, the temporo-sphenoidal that of hearing, and the higher psychical centers are to be found in the frontal lobe.

Mills says that for practical convenience the brain may be subdivided into five lobes, four of these at least, according to the great general functions subserved, these lobes having, in nearly all directions, well defined fissural boundaries. 1. A higher psychical or inhibitory lobe, in front of the basal and anterior branches of the great Sylvian fissure and on the median surface in front of the anterior bend of the calloso-marginal fissure. 2. A motor lobe, including the posterior parts of the first, second and third frontal, both ascending or central convolutions and the adjoining marginal gyrus on the median surface. 3. A lobe for general or common sensation, including the gyrus fornicatus, the hippocampal convolution, the precuneus and the postero-parietal gyres. 4. A lobe of special senses, including the whole of the occipital and temporal lobes. 5. The Island of Reil or insular lobe.

The symptoms of organic brain disease and their relationship to localization are both general and special. General symptoms: Headache or cephalalgia is of very common occurrence; we meet it at all times and under all conditions. So common is it that its value as a diagnostic symptom is often overlooked. In considering it symptomatically we will take it regionally, first mentioning that its value depends on intensity, permanency and character of pain. We know that headaches of the same origin, in different individuals present different phases, but taking the headache as an associated symptom we can pretty firmly establish our diagnosis. Frontal headache is principally sympathetic in origin, indicating eye strain, or neurotic nasal disturbance. In migraine frontal headache may be pronounced, but usually it attacks one side of the forehead.

In organic cerebellar disease we find frontal headache as a marked symptom. In cerebral syphilis, where gumma exists in the frontal region, we have localized frontal headache, which is more severe at night. Temporal headache is found in disease of the temporal bone as in caries of the petrous portion; also in brain tumor in syphilitic brain disease, and in abscess of temporo-sphenoidal lobes. It may be reflex in origin also. Coronal headache seldom indicates brain disease but is reflex and calls attention to the digestive tract and pelvic viscera. Organic basillar disease of the brain will occasion occipital headache. Localized headache in organic brain disease only exists when the pressure from the lesion involves tissues and nerves. If the deep centers of the brain are involved, headache will be general.

In cases of epilepsy, cerebral hemorrhage, abscess, etc., we may have a continuous general headache; as a rule, however, the attacks are periodical. Vertigo, optic neuritis and convulsions are other general symptoms of great importance. Vertigo, when paroxysmal, is usually associated with nausea and vomiting. I recall one case where vertigo was always accompanied by a desire to go to stool; the attack would end in free vomiting and purging. Vertigo, when continuous, and by this I mean, manifested on least change of position, is often witnessed in Jacksonian epilepsy, in adults. Vertigo is allied to epilepsy; has often an aura and is followed by lassitude and even loss of consciousness. Several very pronounced cases of vertigo have come under my observation during the past year, one case of cerebellar disease (abscess of middle lobe), one of tumor (middle lobe cerebellum). In several cases of cerebral syphilis, in one case of abscess of the temporo-sphenoidal lobe, and in focal epilepsy I found it a frequent associated symptom.

Optic neuritis may be the first symptom leading to the attention of the physician being called to cerebral disease. The patient first notices a failure of vision and when examined for defect the opthalmoscope will reveal neuritis. A considerable degree may exist before vision is affected. The neuritis is then probably subsiding as the acuity of vision may not be affected during the inflammatory stage. To decide whether the defect in vision is the neuritis or the intra-cranial disease, we must compare the degree of neuritis and the degree of affected vision. Neuritis, says Jacoby, is pathognomonic of brain tumor. Where defective vision exists, let me urge upon you opthalmoscopic examination in all cases, especially when cortical disease is suspected. Here are presented the first symptoms of grave organic brain disease.

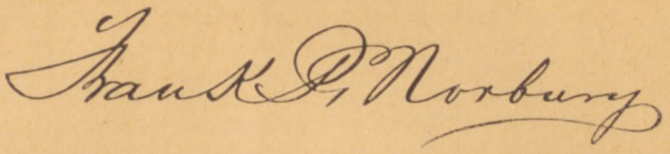
A case in point: A young man, telegraph operator, noticed acuity of vision diminishing; could not see to write very well; called on an ophthalmologist who diagnosed optic neuritis and referred him to a neurologist in Chicago, for further examination. He diagnosed gummatous brain tumor, which diagnosis proved correct; as subsequent history showed. The young man denied ever having had syphilis, and as he was then expecting to be married soon, refused constant treatment, recommended to him. He was married, and in due time other symptoms of syphilis appeared; he became insane; was under my care three years. He died from extension of paralytic symptoms. His case was one of the most unique and interesting of organic disturbances of the brain, with heterogenous focal symptoms, I have ever studied.

The special cerebral symptoms are either those of irritation, destruction, local pressure or reflex, manifested in spasm, or paralysis, or both. Their intensity and duration depend greatly upon the manner of onset of the lesion, whether gradual or sudden. In the former case, partial repair may take place during the continuence of the destructive process and the symptoms may not be so well defined; in the latter, their extent may in themselves be misleading as to the real value of the lesion, so it is well to be observing in the consideration of special symptoms in making our diagnosis, keeping in view the fact that the mode of onset indicates the nature of the lesion and oftentimes its location as well, "inasmuch as certain lesions are more common than others in certain parts." Mono-spasms are the result of local irritative lesions of the cortex, the pathology of which was first clearly defined by Hughlings Jackson. The irritation of tissue surrounding a local lesion is sufficient to keep up a discharge of nervous energy, and where the discharges are frequent, temporary paralysis of the muscles implicated in the region follows. The causes of local lesions are variable, but they are principally encephalitis, tubercle, syphilis or spicula of bone. Syphilis causes the majority of cases of mono-spasms which ultimately become monoplegia by the irritating lesion, becoming destructive by its extension. Spasm may continue after paralysis has resulted. The clinical varieties of mono-spasm are as follows: Crural (limited to the leg), brachial (limited to the arm) facial (limited to the face) oculo-motor (limited to the muscles of the eye).

Now, where the lesion is such as to cause an extension of the spasm to other regions, as where several of the mono-spasms merge together, we have then defined epilepsy, for consciousness is then involved. Convulsions do not become epileptic until consciousness is lost. This is a diagnostic point (but disputed). Where destructive process is extensive, hemiplegia will result. Voluntary power is thus lost by involvement of the motor centers in the central convolutions. The hemiplegia will be on the opposite side if the brain is anatomically normal, that is, if the fibres decussate; if they do not the hemiplegia will be on the same side as the lesion. Rigidity generally follows as a secondary result, due to degeneration of the pyramidal tract. Partial lesions may exist and involve either the face, leg or arm. These are very common, first, says Gowers, on account of the wide extent of the central region, and secondly because the region is supplied by different arterial branches and softening from arterial occlusion is a common lesion. Monoplegia always suggests cortical disease. The leg is an exception, as it can be affected only when there is involvement of the paracentral lobule. Lesions situated high up in the subdivision of the motor area may affect just special parts of an extremity, etc., as a lesion in the highest part of the arm region affects the shoulder more than the rest of the arm. When irritating lesions alone exist convulsions occur, beginning locally, as I have before stated, and may extend to both regions. By watching for the commencement of the convulsion we may localize the lesion, as for instance if the hand or fingers, we locate it in the middle third of the central convolution. This local commencement of spasm diagnoses cortical lesion. Speech defects also mark location of lesion. "To express our thoughts we need memory of words, to know how to say them, a proper condition of the organs of voice and articulation." A defect, involving any one of the above qualifications will produce aphasia in some form. Amnesic aphasia, or loss of the memory of words, is a common form and often seen in organic brain disease. Occasionally we find word-blindness, familiar objects lose their identity or their name, the noun, may be lost and in its place we have the verb to express its peculiarity, like the word "fire" is forgotten and "that which burns" is used to express it. Or the wrong word is used because of a similarity of objects in shape, size, etc., as in a case I saw in consultation recently, the patient, in trying to speak of capsules which her physician had left her, called them "potatoes." Her case involved more than word-blindness, however, but on the aphasic symptoms depended the diagnosis, which subsequent history proved correct.

Word-blindness is not the only sensory disturbance we have, for now deafness is noticed as well, certain sounds are lost, or all sounds are lost or perverted. The sound of words may be heard but may be utterly unintelligible. I have seen such cases among feeble minded children, and on one or two occasions, in insane patients. The lesion in such cases is to be found in the posterior half of the first and second temporal convolutions of the left hemisphere in right-handed persons. The lesion in word-blindness is probably in the angular gyrus. Most cases of amnesic aphasia have associated motor aphasia or ataxic aphasis. This form of aphasia (motor) is most frequently seen. The patient understands what is said to him, remembers it, remembers what he reads, recognizes his friends, objects, etc., but cannot express his ideas. He may be able to use a simple word or a sentence which he is apt to use on all occasions. This form of aphasia is frequently seen in embolism of the middle cerebral artery. It is also a symptom in cerebral tumors, traumatic injury to the brain, when hemorrhage by compression causes it. It is noticed in double athetosis, sometimes in adult chorea. In porencephalus or congenital defect it is a frequent associated symptom. Embolism in elderly people is heralded first by loss of consciousness, then with return of consciousness, hemiplegia with aphasia is noticed.

Frequently some poor fellow, the victim of embolism, is found on the street, taken in by the police, and because he cannot speak, is locked up for being drunk. He may be found dead in his cell in the morning. I knew of such a case. It is interesting to note in most aphasiacs, the connection between disease of the left hemisphere and ataxic aphasia. It is explained because of the development of the left hemisphere in right-handed people and the reverse in the left-handed. The localization of motor aphasia is to be made carefully; we will find the third frontal or Broca's convolution always involved, together with the Island of Reil in complicated cases. Aphasia is an important consideration from a medico-legal standpoint in cerebral disease and should be thoroughly investigated. I have thus briefly reviewed the important points of cerebral localization, and in closing, ask that in the examination of all cases presenting focal symptoms, plenty of time be taken and comparisons made, so that localization may have the credit it deserves in the diagnosis of cerebral diseases.



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