

for that purpose I have put the greatest trust in using a saturated solution of iodine with cataphoresis, using the negative pole in order to force it through the tissues. In this way I have obtained the same constitutional conditions and diminished the inflammatory, so necessary before active operations are undertaken. It is my firm conviction that if such conditions are properly treated it is necessary to give more attention to the local condition. I refer now to the keeping in absolute hygienic condition all the teeth of our patients. For my part, I have found it impossible to give the time necessary, and I have for years trained a female assistant, who devotes her entire attention to the cleansing of individual teeth. I agree thoroughly in the idea that only one, or two, or three teeth ought to be cleansed at a time, and not attempt to cover too much surface. Following this method, I feel at the present moment that my patients can have their mouths kept in the hygienic condition all the teeth of our patients. For my part, only if sufficient dental attention is given. I can not imagine any one present who is able to take the time which is necessary to do this work effectively. I throw this suggestion out for the benefit of those who have not already adopted such a plan. I am able to keep a young woman busy at nothing else but this one particular thing, and she has trained her fingers so well in this that I have many of my friends, my professional colleagues, come to her for this work because she has become so adept with an experience of seven or eight years.

DR. E. S. TALBOT, Chicago—Dr. Brown brought up one point not down on the program, and that is natural absorption. I believe that in the evolution of man the absorption of the second set of teeth is in accord with the process. In the lower vertebrates there is a succession of teeth. When one set has done its work it is lost and a new set takes its place; this continues throughout the life of the animal. Advance in evolution finds man with only two sets of teeth. Natural absorption takes place in every individual. This absorption is called osteomalacia. It takes place in the lower animals such as carnivora, monkey, horse and cow. The alveolar processes being transitory structures, disease of any kind due to trophic changes is liable to produce absorption. This may take place in children as young as 12 years, as I can prove by photographs and skulls in my possession.

DR. G. V. I. BROWN, Milwaukee—If the teeth came together so that there would be no motion, there would be comparatively little pyorrhea. When the occlusal surfaces are such that there must be a slight adjustment of the position of the teeth, every time the mouth is closed, the slight movement constantly repeated causes inflammation and a series of disturbances which develop the disease.

DR. J. H. SALISBURY, Chicago—Dr. Litch stated that he had difficulty in getting his patients to drink the amount of water Dr. Talbot prescribed. That may be due, in some cases, to the fact that the water is retained in the stomach, and so causes disturbance. Absorption of water from the stomach is practically unnatural, and if for any reason the stomach does not empty itself water will remain there and give more or less trouble. That failure to empty itself is due to the fact that the intestines are full. In that case it would be desirable to see that the intestines are emptied at proper intervals. Constipation in some cases may be the cause, or in other cases it may be due to dilatation of the stomach and causes which prevent the passage of liquid from the stomach to the intestines.

The criticism raised in regard to the continued use of drugs arose from a misconception. The continual use of drugs was not advocated. It was not at all necessary to use them but for the purposes of arousing the various organs to activity; as for instance, to empty the intestines a cathartic or two or three doses would be very desirable, but in general natural means to stimulate the powers of the system should be depended on, rather than the action of drugs. As a general practitioner, I am much interested in this question, not only from the standpoint of etiology, but also from the impression that it is a matter of vital importance. The profession should understand the possible deleterious effects upon the general system of a continued swallowing of food and mucus laden with pathogenic organisms derived from the teeth. Both the medical profession and the laity should be impressed with the very serious

importance of attending to this matter, that no very serious changes take place in the stomach, and consequently in the blood.

CEREBRAL LOCALIZATION.*

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Fifty years after Gall had published his doctrine of cerebral localization, the scientific medical world still believed with Flourens that "not only did all perceptions, all volitions, all intellectual faculties, reside exclusively in the brain, but that all faculties occupied the same place in it. That as soon as one of them disappeared from a lesion of any one point of the brain proper, they all disappeared; that as soon as one returned after the healing of that lesion, all returned. That the ability to perceive and to will, therefore, constituted essentially but one faculty; and that that faculty resided essentially in a single organ." Again fifty years had elapsed and a chart of the surface of a brain could be fitly compared to "a political map of Germany at the end of the seventeenth century." So greatly has our knowledge of cerebral localization advanced in those years that it seems impossible to give more than a most cursory review of what is known, in the course of one evening. Many important and interesting theoretical considerations will necessarily have to be neglected entirely.

We will begin with cortical localization, that part of our subject which is probably of greatest importance from a clinical point of view. The region of the surface of the brain which has been studied most carefully, and about which we consequently know most, is the motor, or Rolandic, area. That portion of the brain which lies anteriorly to this, the so-called prefrontal region, appears to be the seat of the highest mental processes. Its destruction is said to annihilate the power of directing one's attention entirely when the lesion is bilateral, while a unilateral lesion often causes only a transitory mental change. Mental weakness, loss of memory and attention, apathy and childish behavior are common symptoms. A tendency to indulge in rather stupid jokes, the "Witzelsucht" of German authors, is supposed to be particularly characteristic. In irritative lesions Flechsig claims boundless delirium of grandeur and its reverse in the early stages, later on complete apathy and loss of judgment to be particularly frequent. Bruns has taught us that pathologic processes in this region may cause ataxia of the so-called cerebellar type.

Before we enter into the study of the motor region of the brain a few general remarks about cerebral centers and brain palsies may not be out of place. These so-called centers are in all probability not mathematical points, nor even spaces with sharp boundary lines, but rather diffuse areas which perhaps differ somewhat in extent in various individuals. It is, of course, known to all of you that each hemisphere influences in the main the muscles of the opposite side of the body, a point to which I shall return presently. Some centers, however, control muscles on both sides of the body equally, such as the orbiculares oculi, the muscles of mastication, of the larynx, pharynx and to a lesser extent some other ones. Irritative lesions in these may, therefore, cause bilateral spasms, while paralysis is found only

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when the center is destroyed in both hemispheres of the brain. In older literature there are quite a large number of cases recorded in which spasms or paralysis were found on the same side as the brain lesion. Nasse has collected no less than 58 of them, but on account of the unsatisfactory histories and uncertain methods of microscopic examination they are of very doubtful value. There are, however, some more recent observations to which these objections do not apply. One of these I had occasion to watch for some months; it was later published by Dinkler. Here, an immense tumor had destroyed practically all of one hemisphere, and it was in all probability the pressure upon the opposite hemisphere which was responsible for the existing spasms.

Another instance of even greater interest is that reported by Dr. Bidon and quoted by Charcot and Pitres. In this case there was hemiplegia affecting the left side of the body, with complete motor aphasia. The post-mortem showed softening of the lower half of the left Rolandic area and the foot of the third left frontal convolution, and the microscopic examination revealed secondary descending degeneration in the left cerebral peduncle and the left anterior and lateral columns; there was no trace of decussation of the pyramids. That cerebral centers may migrate from one side of the brain to the other was at least made probable by an observation of Oppenheim. His patient had been right-handed up to the age of 17, when an injury to the right hand compelled her to learn to write with the left one. When at the age of 59 a tumor developed in the right hemisphere, it caused left hemiparesis with aphasia. Bramwell has reported a somewhat similar case in an old man.

Since the so-called psychomotor centers are distributed over a relatively large area of the cortex supplied by a number of small arteries, superficial lesions cause monoplegias more often than hemiplegias. On the other hand, monoplegias due to destruction of the white substance of the brain are rather rare, a point which may at times be of considerable value in determining the exact location of a pathologic process.

Cerebral hemiplegia, whether it be due to a superficial or a deep-seated lesion, never means a complete paralysis of one-half of the body. Those muscles which we habitually use together with the same muscles of the opposite side are affected only to a slighter degree. Thus we find paralysis of arm and leg, together with paresis of the lower part of the face, slight weakness only in the muscles of the tongue and trunk, excepting the trapezius, and no demonstrable loss of power at all in the muscles of mastication, articulation and those of the eye. Since we use both legs simultaneously more often than both arms, the weakness is usually more pronounced in the former than in the latter. Paralysis of cortical origin is flaccid in the beginning, and presents late contractures due to secondary degeneration in the pyramidal tracts.

We will next consider the distribution of motor centers in the so-called Rolandic area, which extends over both central convolutions and the paracentral lobule, to a lesser extent into the frontal convolution and the superior parietal lobule. The muscles of face and tongue, and probably also those of mastication, are represented in the lower portions of both, principally the anterior, central convolutions. The upper extremity occupies about the middle one-third of these same convolutions, extending further up anteriorly than posteriorly, while the upper one-third, as well as the paracentral lobule, and probably part of the superior parietal lobe, control the movements of the lower extremities. Experiments

on animals, in part verified by clinical experiences, have taught us a great many details, which you will find on charts in any modern text-book on nervous diseases. I shall mention only a part of the more important ones here.

While monoplegias of cerebral origin affecting only the leg are relatively rare, they have been bilateral in a rather large percentage of the cases recorded. The proximity of the two paracentral lobules explains this rather interesting observation. Charcot has claimed that tubercular lesions have a special tendency to attack the leg center. A curious fact, for which there had been no satisfactory explanation for many years, is the apparent immunity of the upper branch of the facial nerve in cerebral hemiplegia. It was thought that for this portion of the facial nerve a separate cortical center must exist, and Exner and Paneth claim to have located it in animals in the gyrus angularis. A more probable explanation for this immunity has already been given. The muscles supplied by that nerve belong to those which are generally used simultaneously on both sides of the body, and, therefore, each cerebral center very probably controls both upper facial nerves. Paralysis could, therefore, occur only when both cortical areas are destroyed. Furthermore, recent pains-taking investigations by Mirallie have shown that "in every case of cerebral hemiplegia in which the lower facial is paralyzed, the upper branch of that nerve is also affected, but to a lesser degree than the lower branches or than in peripheral facial paralysis."

The cortical center for the muscles of mastication has been located in a number of animals, the dog, ape and rabbit, but, as far as I am aware, there are no clear cases in the human being on record to verify these observations.

The same applies to the center for the rotation of the head. We know where to find it in animals, but are uncertain about its exact location in the human brain.

The cortical center for the larynx was first demonstrated in cats by Ferrier in 1873. His conclusions were confirmed by experiments made on dogs by Soltmann, Krause and Duret, and on apes by Masini and Semon and Horsley. It is in all probability bilateral in man, and located at the lower end of the Rolandic area, in the immediate neighborhood of the centers for speech and face. Cases in the human being verifying these experiments are very rare, and, therefore, one which I have under observation at present may be of some interest. The patient is a man, 45 years of age, who had been ill for one week when he first consulted me. He complained of headache, and stated that about every half hour he had a peculiar attack, during which he suffered from "sticking pains" and a "shaking sensation" in his throat, and was unable to utter a sound. The examination of the central nervous system revealed no objective signs of disease, but there was marked enlargement of all superficial lymphatic glands. At that time the patient denied syphilitic infection, but later acknowledged that it occurred about one year ago. Two laryngologists examined the patient, and could find nothing to explain the laryngeal spasms, from which the patient evidently suffered. He was put on iodid of potash, and after about one week reported that he was worse. I then for the first time could witness one of his attacks. It consisted of a marked laryngeal spasm, followed by aphasia, which lasted but a very few minutes, but recurred frequently. He was sent to the Michael Reese Hospital, and there developed clonic spasms of the right side of the face, which usually followed the

laryngeal attacks and a more permanent motor aphasia. Dr. Hale, who kindly examined the fundus for me, found optic atrophy on both sides. Under vigorous antisyphilitic treatment he improved considerably, was discharged, but returned after about two weeks, complaining of weakness in his right arm. This gradually increased and spread, until now the patient has a right hemiplegia with complete motor aphasia, which does no longer yield to antisyphilitic treatment. He is sinking rapidly, and will probably die within a week or so.¹

While we are discussing the centers controlling the voluntary muscles of the body, it may perhaps be opportune to say a few words about that most important group of symptoms due to cortical lesions, Jacksonian epilepsy. It appears that quite a number of physicians even to-day consider every localized spasm of an organic nature to be cortical. That is certainly not correct. The true Jacksonian epilepsy is characterized by a number of features which occur but very rarely when other parts but the cortex are irritated, so that if we only know what constitutes an attack of Jacksonian epilepsy we will seldom err in basing our localization upon its presence. The convulsion proper is almost always—Grasset even claims invariably—preceded by a brief tonic spasm, then we see a series of clonic contractions, beginning in an isolated group of muscles, to which it either remains confined, or else it spreads progressively to adjoining groups of muscles, or may involve all of one-half of the body, or even become generalized. The convulsions hardly ever last more than five minutes, and are followed in the part first affected by a feeling of great lassitude, or even by a paresis, which latter persists for some hours, a few days, rarely for a longer time. With each successive attack this paresis becomes more intense and lasts longer; in rare cases we see post-epileptic contractures. The convulsive period is followed in the more severe cases by post-epileptic stertor, or sleep, usually shorter in duration than in so-called idiopathic epilepsy. Other post-epileptic symptoms are delirium, headache, amnesia, vertigo, vomiting, transitory aphasia, hemianopia or myosis. The sensibility usually remains intact. In rare instances, particularly when a syphilitic lesion causes the attacks, we find patches of anesthesia, most commonly on the dorsum of the hands, the outer side of the hips or the forearms. Charcot, Bourneville and others have reported cases in which the spasm consisted of tonic contractions only.² A glance at a chart in your text-books will show you in what way Jacksonian epilepsy may spread. The following sequence is most often seen: Face-tongue, face-arm-leg, arm-face-leg, leg-arm-face. The spasms could never jump from face to lower extremity without affecting the arm also, as the center for the latter lies between those of the two former.

Just as in idiopathic, we at times see epileptic equivalents in the Jacksonian form of epilepsy, which may either alternate with the convulsive attacks or may take their place altogether. One case of the latter type, which I saw some years ago, I referred to briefly in a discussion on epilepsy last year. Here, attacks of visual hallucinations were the first sign of a brain-tumor. In other cases these equivalents consisted in attacks of vertigo, so-called "absence," hallucinations of sight, hearing or of the olfactory sense, localized paresthesiæ

or sudden, impulsive acts. Attacks of migraine ophthalmique have been described; according to Féré they consist of two phases, that of excitation, in which violent circumscribed headache, vertigo, vomiting, convulsions, scintillating scotoma are noted, followed by a period of exhaustion, comprising hemianopia, somnolence, and at times aphasia and hemiplegia.

Such attacks are, as I have already stated, very rare, excepting in cortical lesions. They have, however, been seen in disease of the centrum ovale, the internal capsule, striated bodies and optic thalamus. They occur most frequently in limited lesions, which develop actively and progressively, such as neoplasms, encephalitis or meningitis. It is not necessary for the pathological process to have its origin in the motor area proper, or even in its immediate neighborhood, as long as it causes irritation of those parts. The existence of paresis, or paralysis, makes it probable that the motor region proper is affected; absence of loss of motor power that the lesion is either very superficial or only in parts near the psychomotor centers. The early occurrence of headache points to a disturbance near the surface—irritation of meninges—a sign of considerable practical importance.

The points which are valuable in differentiating Jacksonian from idiopathic epilepsy are the following: The spasm is limited in the beginning of the attack; loss of consciousness occurs late, if at all; the epileptic cry, fall and biting of tongue are usually absent; the post-epileptic coma is slight, of short duration or entirely absent; in the so-called *état de mal* there is no rise of temperature; finally the history of the case, in particular the age at which the first attack occurred.

Common or tactile sensibility is supposed by some authors to be localized in the psychomotor area. Some even go so far as to claim that destruction of these parts does not cause any disturbance of motility primarily, but only in so far as disturbances of sensibility interfere with the voluntary use of our muscles. Others again claim that the motor and sensory centers are distinct and separate. One of the latter group, Ferrier, has collected 284 cases in which paralysis was caused by cortical lesions, and has found that in 100 of these nothing was said about the condition of sensibility, that in 121 it was found intact, and in the remaining 63 affected. Counting only those cases in which the condition of sensibility is mentioned—and it seems extremely probable that in some of the first 100 it was not mentioned, because intact—we would find it normal in about two-thirds of all cases. Where anesthesia does exist, it often does not correspond in extent to the area of paralysis, and is usually more transitory. This has induced Charcot and Pitres to conclude that in most cases of anesthesia with cortical hemiplegia the former is purely functional, "analogous, if not identical, with hysterical anesthesia," just as we see a purely functional loss of sensibility in organic paralysis of spinal or peripheral origin. (Erb's case of progressive muscular atrophy, Gowers' case of peripheral facial paralysis, etc.)

Goltz has found that in dogs even the ablation of an entire hemisphere causes only hypæsthesia, not anesthesia, and he himself admits that this may possibly be due to destruction of fibers below the cortex.

Horsley found that when parts of the motor cortex were removed in man slight loss of tactile sensation, difficulty in localization stimuli, and loss of muscular sense resulted. Most authors, I believe, concede that the latter, the muscular sense, is most intimately associated with the Rolandic area, but it can not be denied that

¹ The patient has since died. No additional symptoms of any importance developed, excepting that just before death the temperature reached 108°. Unfortunately we could not make a post-mortem examination.

² Other types of epilepsy may of course occur in cortical lesions, but they are not characteristic.

even it may remain intact when the cortex is destroyed by disease. Ferrier has sought for another localization of common sensibility, and claims to have found it in the hippocampus major and gyrus fornicatus. Observations in the human being, tending to prove the correctness of his views, are not known to us. The entire problem of cortical localization of sensibility, you see, still remains to be solved.

One of the clinically most important centers of the Rolandic area we find in the posterior of the third left—in right-handed persons—frontal convolution, the seat of the faculty of articulated speech. Its destruction deprives the individual of the power of speech, with conservation of the faculty to understand, read and write. Broca is generally thought to have discovered this important fact, while in reality he was only one of several, each of whom contributed his share. When he published his first "memoire" he did not, apparently, know that this center was found in the left hemisphere only, a fact which had been noted by Dax twenty-five years before; but the most important work of the latter had been noticed by few, so that Broca received all the credit. In about the same region, as nearly as we can locate it to-day, is the center for musical expression, which, however, may remain intact in cases of motor aphasia. Lesions in the second left frontal convolution probably cause agraphia, i. e., loss of the memory for written images. On account of the proximity of this center to Broca's convolution, motor aphasia and agraphia are very often found in the same case.

Some authorities have claimed to have found other centers in the motor area, but their observations are all lacking convincing confirmation. Thus, Herrington claims to have located a center for the contraction of the sphincter ani in the paracentral lobe, while an area controlling movements of the bladder is supposed to be located in the front part of the gyrus sigmoides. According to Raudnitz, stimulation of the vasomotor centers in the Rolandic region decreases the temperature in the corresponding extremity, while destructive lesions cause the opposite effect.

Unverricht and Preobraschewsky claim to have found a center controlling respiration near the fissure which separates the second from the third frontal convolution.

About the symptoms caused by disease of the cortex of the insula we know practically nothing. Perrier states that they consist in paralysis of arm and leg, without involvement of face or trunk, that the paralysis is very much more marked in the upper than in the lower extremity, that it is flaccid, without contractures, is neither accompanied by disturbances of sensibility, nor by any characteristic changes in cutaneous or deep reflexes.

Parietal lobe.—The most important portion of this lobe is the gyrus angularis. It appears to have some relation to our visual apparatus. Irritative lesions occasionally give rise to optical illusions or flashes of light, followed by temporary amblyopia, while destructive pathologic processes, more particularly in the left hemisphere, usually cause word-blindness. Bruns calls attention to the occurrence of hemi- or mono-ataxy in lesions of the parietal lobe and believes with many other observers that this part of the brain is intimately associated with the muscular sense.

It is possible, though not proved definitely, that centers for the movement of the eyeball and upper lid may be in this region. Charcot and Pitres publish an inter-

esting case in which a small focus of superficial hemorrhagic softening in the inferior parietal lobe caused ptosis. But Surmount has collected 61 cases of disease in the parietal lobe, in only 11 of which ptosis had been found. Conjugate deviation of the eyes and the head is also supposed in some instances to have been due to lesions in this region. But it is a rare symptom, occurs most frequently in cases of general meningitis, and is usually transitory in hemiplegias, so that we have few data for definite localization. It may also occur in disease of the mesencephalon. When it is cortical in its origin, the patient is said to look toward the convulsed extremities in case of irritation; at the lesion, in case of destruction, a law which is exactly reversed in case the mesencephalon is affected.

The cortex of the occipital lobe contains the visual centers. Their exact location is still somewhat doubtful. The symptoms characteristic of cortical lesions in this region are the following: The visual disturbance is homonymous, there are no ophthalmoscopic changes—unless due to increased cerebral pressure—the pupillary reflexes remain intact, even if a pencil of light be thrown upon the blind half of the retina only. Visual hallucinations or scintillating scotoma frequently precede or coexist with the hemianopia. It is claimed by some that in the occipital lobe are to be found the psychomotor centers for the eye-muscles and that lesions limited to its anterior portion may cause color hemianopia.

In the temporal lobe we find the auditory centers. Destruction of the left superior temporo-sphenoidal lobe causes loss of the auditory images of words, word-deafness; destruction of both temporal lobes total deafness, while irritative processes in the superior temporo-sphenoidal gyrus often give rise to auditory discharges and subjective auditory sensations, so that a spasm which begins with an auditory aura always suggests that localization.

On the lower surface of the brain are supposed to be centers of the olfactory and gustatory senses, on the region of the gyrus uncinatus. Whether the fibers preceding from them cross or not has not been ascertained definitely; for those of the sense of smell it seems very improbable.

If the large mass of white matter under the cortex is the seat of disease there may either be no focal symptoms at all, or else those of a cortical lesion or of one in the internal capsule, according to the seat of the pathologic process. Jacksonian epilepsy, however, is but rarely met with.

In the capsule interna motor and sensory tracts lie closely together, so that hemiplegia and hemianesthesia must occur in extensive lesions. The order of representation of movement is from before back: opening and turning of eyes, opening of mouth, turning of head and eyes simultaneously, turning of head alone, moving of tongue, of angle of mouth, of shoulder, elbow, wrist, fingers, thumb, hip, ankle, knee, hallux, toes. Back of the motor tracts are the fibers conducting sensibility. The group of symptoms which is characteristic for disease of the internal capsule is hemiplegia with hemianesthesia including hemianopia, but without disturbance of the pupillary reactions to light. This hemiplegia differs from that due to cortical lesions in this, that the loss of sensibility is often not only more pronounced, but also more permanent, that all qualities of sensibility are usually affected and that all of the mucous membrane with the exception of part of that

of the eye may be anesthetic. Hearing, smell, taste often suffer as in hysteria. Post- or pre-hemiplegic hemichorea or hemiathetosis are almost pathognomonic of disease in this region. Grasset has also observed hemiataxy. In most of the cases in which intense neuralgiform pain occurred on the paralyzed side, the internal capsule or its immediate neighborhood was the seat of the lesion.

About the functions of the optic thalami but little is known. They are supposed to control to some extent mimic expression; so that destructive lesions would cause loss of this function, while irritative lesions may cause spasmodic crying and laughing, particularly if situated in the anterior portion of this ganglion. In most cases the symptoms will be about those just described for disease of the internal capsule and due to involvement of these paths: hemiplegia, hemianesthesia, hemichorea, etc. A very pronounced atrophy of paralyzed muscles, developing rapidly with quantitative and qualitative changes in their reaction to the electric current, was supposed by some to indicate trouble in the optic thalami, but the same phenomena have been seen in disease of the cortex.

Destruction of the striated bodies causes no focal symptoms as far as we know. These ganglia are said to contain thermic centers, but as there are some in other parts of the brain, we can not utilize this fact in localizing.

The diagnosis of a lesion in the corpus callosum is also usually impossible. The following signs are supposed to be of some importance in case of tumor:

1. The existence of symptoms of an organic cerebral trouble which develop slowly and increase constantly in intensity.
2. Hemiparesis, usually combined with more or less weakness of the opposite side of the body.
3. Very pronounced dementia, which contrasts strikingly with the insignificance or total absence of the so-called general symptoms of tumor—headache, vomiting, convulsions, optic neuritis.
4. Existence of dysarthria.
5. Absence of any symptoms suggesting any other localization.

Of these diseases which may affect the lateral ventricles directly we can recognize with some degree of certainty only hemorrhage. Its onset is naturally very sudden and violent, the coma is very profound from the beginning, convulsions and contractures are frequent, while paralysis is in some cases entirely absent, in others general. Vomiting is relatively infrequent. Recovery is rare; in the majority of instances the patients died within a few hours.

The symptom which characterizes disease in the region of the third ventricle is the nuclear ophthalmoplegia. The nuclei of the eye-muscles are arranged so that the cells for the ciliary and sphincter iridis muscles are placed anteriorly, those of the trochlear and abducens nerves posteriorly. If an ophthalmoplegia is bilateral and affects all ocular muscles, whether they be paralyzed or only paretic, it is almost certainly nuclear in its origin; the same symptoms may occur, but are very rare in lesions at the base. If an ophthalmoplegia is unilateral and affects all muscles of that one side it may be due either to disease in the region of the third ventricle or to some disturbance at the point at which the nerves enter the orbit, more probably the latter, and then often combined with some degree of exophthalmus.

The cerebellum is relatively often the seat of disease.

Tumors, particularly tubercles, are often met with in children, and sarcomata and cystic growths are not at all rare. The most important topical symptom is the so-called cerebellar ataxy, a peculiar reeling gait, closely resembling that of an intoxicated person, with a tendency to fall toward the affected side. Another form of disturbance of co-ordination has been seen in some few cases of cerebellar lesions, an ataxy which differs in no way from that of tabes. Second in importance as a cerebellar symptom comes vertigo; it is true that a disturbance in almost any other part of the brain may cause this phenomenon, but in disease of the cerebellum it usually appears early and throughout the course of the malady remains a very prominent and annoying feature. Exactly the same is to be said of vomiting and headache. If a tumor, etc., affects only the cerebellum and not the neighboring parts there is no distinct paralysis, but a certain amount of weakness is often found in the extremities corresponding to the side on which the lesion is located. Some authors even go so far as to hold a paresis of the muscles of the trunk and legs responsible for the ataxy. In quite a few cases intention tremor of the upper extremities has been observed, the occipital region is often sensitive to percussion, and the knee-jerk is relatively often not obtainable. Paralysis of ocular muscles, paretic nystagmus, dilatation or immobility of the pupils, paralysis and anesthesia are very common, but they are due to pressure on neighboring parts, not to the cerebellar lesion proper. Formerly it was thought that the occurrence of cerebellar ataxy always meant involvement of the central lobe of the cerebellum, the vermis superior, but we know now that this is incorrect. I have myself seen a case in which an angiosarcoma had almost totally destroyed that part and there had never been any indication of inco-ordination.

Lesions in the anterior corpora quadrigemina may cause associated movements of the extrinsic ocular muscles, those in the posterior bodies paralysis of these muscles, particularly of the *recti superiores et inferiores*. The intrinsic muscles are often intact in such cases, as their nuclei are placed anteriorly to the parts we are now discussing. Ataxy, both of the cerebellar and the tabetic type, occurs frequently, particularly in the form first mentioned and in a few instances choreiform movements have been recorded. Deafness is rather a common symptom, crossed in unilateral disease. Whether the hemianopia which has been seen, is due to disease of the corpora quadrigemina proper or to involvement of the external geniculate bodies is doubtful; the fact, however, remains that it is a frequent symptom. It is thought by some that this region contains centers which control perspiration.

Disease of the hypophysis is thought to be the cause of acromegaly, although in some cases of this disease the pituitary body has been found intact. Only recently I saw a case of psammoma of this region with stunted growth and with but very few of the symptoms which are supposed to be characteristic for this localization. They are: Pain in the frontal and temporal region, shooting toward the orbits; early involvement of both eyes in form of an amblyopia with temporal hemianopia, followed later by so-called primary optic atrophy, paralytic squint, somnolence, weakness in the lower extremities, occasionally dementia and diabetes mellitus or insipidus.

If the crura cerebri are affected we most frequently find that type of alternating hemiplegia, which is known under the name of hemiplegia alterans superior, i. e., paralysis of the oculomotor nerve on one side, that of the

lesion, and of the muscles of the lower face and the tongue and the extremities on the opposite side. Ptosis is usually pronounced and often one of the first, if not the first, symptom. The intrinsic eye-muscles usually remain intact. In some cases paralysis of the extrinsic muscles of the eye has been associated with a tremor of the opposite side of the body, resembling either the intention tremor of disseminated sclerosis or that of paralysis agitans; in other instances the oculomotor nerve of one side and the contralateral facial—lower branches—have been paralyzed. Frequent micturition and incontinence have been noted in a number of cases. It is rather common to see the disease begin in one crus and spread to the other one.

Another type of alternating hemiplegia is seen, when the pons is the seat of a pathologic process. Here it is the facial on the side of the lesion and the extremities and hypoglossal nerve on the other side of the body that are affected. Paresthesia and anesthesia of the latter half of the body may or may not occur. Another form of alternating hemiplegia due to disease of the pons, but less frequent than the one just mentioned, is that of the abducens on one, of the extremities on the other side. Again, in other cases the abducens of one side and the rectus internus of the other one are paralyzed, so that both eyes are turned toward the seat of the disease. In a third, the rarest, form of alternating hemiplegia finally the trigeminus—its motor or sensory branches or both together—are paralyzed on one side, the extremities on the other. Occasionally we see trophic disturbances in the trigeminal region, such as neuroparalytic keratitis. The hearing is quite frequently affected to some degree, although complete deafness is exceptional.

It is a matter of course that various combinations of the types just described may occur and that such an alternating hemiplegia is possible only in unilateral lesions. It is needless to enter into a description of the symptoms caused by bilateral lesions; a moment's thought will show what they must be; but I might add that the nerves in the medulla oblongata are usually affected more or less, thus giving rise to the additional symptoms of bulbar paralysis, difficulty in swallowing, dysarthria, paralysis of soft palate, pharyngeal and laryngeal muscles. Among the rarer symptoms the following may be worth mentioning: Albuminuria, mellituria, polyuria, and very pronounced rise in temperature. Convulsions resembling those of epilepsy are rarely seen, but more tonic contractions, especially of the muscles of the trunk and neck are not uncommon. Choked disc usually appears very late in pontine lesions and there is but little disturbance of the intellect.

The medulla oblongata contains the nuclei of the ninth to twelfth cerebral nerves, sensory, motor and vasomotor paths and centers controlling the actions of heart and respiration. Hence lesions of this part cause partial or total deafness, paralysis of the soft palate and of the larynx, of the vocal cord, disturbance of cardiac action and of respiration, paralysis and atrophy of the tongue with dysarthria and paralysis of motility and sensibility in the extremities. Since the nuclei of these cerebral nerves are mostly placed close together near the median line, unilateral paralysis or alternating hemiplegia must necessarily be infrequent. Diabetes mellitus, polyuria or albuminuria are common.

This is rather a hasty and incomplete review of what is known of cerebral localization. I would add but one point, and that is in attempting to localize a lesion in the brain we should always be prepared for surprises. I

have mentioned a few of the curious cases I have seen, such as the case with convulsions on the side of the cerebral tumor, the tumor of the hypophysis with stunted growth, and as a warning to those who would still be too confident of our ability to determine the seat of brain-disease would say that I have seen typical Jacksonian epilepsy on the same side of the body in which the disease was located in an abscess of the cerebellum.

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