

2. Loss of the vibrating sensation on the legs or on the abdomen and legs or on the abdomen only. Babinski reflexes, loss of abdominal reflexes. (Later signs of disseminated sclerosis.)

3. Girdle pains, often for a long period; then loss of vibrating sensation on the legs, with Babinski reflexes. (Later signs of compression myelitis from tumor of the meninges or vertebrae.)

4. Loss of the vibrating sensation, loss of the tendo-Achillis reflexes, pain in the legs. (Later loss of knee-jerks and other symptoms of peripheral *multiple* neuritis.)

THE RELATIONSHIP BETWEEN CENTRAL AND PERIPHERAL INVOLVEMENT OF THE CRANIAL NERVES.*

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IN the brief time assigned to me it is obviously impossible to deal with all the symptoms caused by endocranial involvement of the cranial nerves. I have therefore selected for consideration some of the symptoms of cranial nerve involvement which present unusual difficulties in diagnosis and about which our knowledge is still incomplete. I propose to confine my remarks to the first, second and eighth nerves and those portions of the fifth and ninth nerves which are concerned with taste. In other words, I am going to consider the cranial nerves that control the special senses in which you are particularly interested.

The various studies that have been carried on to locate the cerebral centers controlling the special senses have cleared up many points that have hitherto been shrouded in mystery, but there still remain a goodly number on which light needs to be thrown. But whether the loss of one of these functions is due to destruction of the end-organ, the cerebral pathways or center is still often most perplexing. You see a large number of such cases where we see *one*. If it would be possible to create a uniform method of study of all such cases, then a Gunn, a Helmholtz or a Hughlings-Jackson, or even a lesser light, might take this vast material, which both in quality and quantity has never hitherto been dreamed of, and cull from it most valuable information. The study of these special senses is beset with unusual difficulties, for here we are dealing with sensory phenomena which are difficult to throw light upon by any experimental observations on animals, since obviously

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indirect methods must be employed to determine a disturbed function in an animal.

The anatomist has quite outstripped the physiologist in this field, for the course of the fiber connections for the most part is quite well known. There are three methods of studying the physiology of these special senses in animals. The first method consists in destroying a portion of the brain and noting whether the animal no longer reacts to a certain sensory stimulus.

In the second method similar observations are made on specially trained animals. An animal is trained to do a certain thing, let us say raise its right forefoot when a certain odor is presented to it, and then after some lesion has been produced one notes whether the animal still does this. This method has been carried out to a high degree of perfection by Kalischer, but there are inherent dangers in the method which throw doubt on its value. It presupposes, it seems to me, that these artificially developed reflexes are so firmly fixed in the animal's mind that a severe trauma, as removal of part of the cerebral cortex, will not influence them unless the function that is being studied is disturbed. Yet forgetfulness has at all times and in all ages been characteristic even of the human race. Even Homer records that the mere eating of the lotus fruit could produce complete amnesia. The pathway by which an artificial reflex is carried out can certainly be more readily inhibited than can the pathway that an animal has always possessed, as in the case of the sucking reflex or the bladder reflex. Therefore, when a certain stimulus does not lead to the reaction to which an animal has been trained, though it suggests that the stimulus is not appreciated, there is always a lurking suspicion which this method of investigation can never dispel that the acquired habit has been lost as a result of the trauma. In man after a severe cerebral injury it is a very common experience to find that the patient's memory for events of all sorts just preceding the accident is lost for weeks and often months. If this is true in man there is every reason why it should also be true in animals.

The third method has been to determine the presence or absence of a salivary reflex when a strong sensory stimulus is produced. This reflex can be elicited with the greatest ease in experiments on monkeys when the anesthetic becomes light, as I have been able to demonstrate to my own satisfaction repeatedly. It therefore makes me feel that the method is too unreliable to be seriously considered.

Up to the present time, therefore, the results of such investigations on animals, many of them very elaborate and careful, have had the serious handicap of uncertainty. I am convinced that the next real advances will be made when accurate clinical observations are subjected to the acid test by research students.

The more careful study and interpretation of subjective sensations, which of course can only be done on human beings, it has seemed to me would lead to advances in this field. By subjective sensations is meant those peculiar phenomena in which a patient complains of certain sensations which are not susceptible of other proof than the patient's assertion. Thus he may say he thinks he smells certain odors when there is nothing around to produce them, or he hears or sees or tastes something when the stimulus to produce these is not present.

The difficulties of the problem are nowhere better illustrated than in the olfactory mechanism. So little is really known on this subject that von Bechterew in his three volumes on the functions of nerve centers devotes but ten pages to the entire subject.

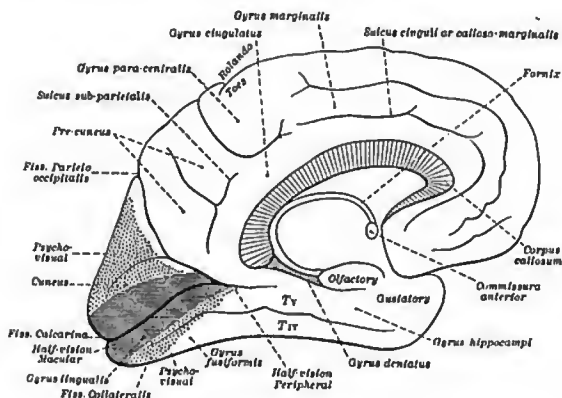


FIG. 1.—From Purves Stewart.

It is now quite generally conceded that the olfactory mechanism after leaving the olfactory bulbs at the base of the frontal lobes passes *via* the fornix to the gyrus uncinatus and pyriformis of the hippocampal lobe (Fig. 1). There is another group of fibers, the fibers of Lancisi, less well developed but of considerable size, which belongs to this mechanism. Our interest lies in differentiating smell disturbances if possible when they occur in these various regions. A lesion in the nose causes loss of all forms of smell, and so does a lesion in the olfactory pathways in the nervous system, unless the lesion, instead of being destructive, is irritative in character. This can be caused by an inflammatory process or merely when there is pressure on the hippocampal lobe; in such a case the patient experiences subjective sensations of smell. I have

believed that the presence of a subjective sensation of smell always means a hippocampal lesion, yet rhinologists have observed patients with this symptom with apparently only a nasal lesion. I know of no instance, however, in which such a case with the lesion supposedly in the nose has had its brain studied. Such studies would be essential to clear up this point. Is the loss of smell resulting from a nasal lesion the same as from a central lesion? Is the recognition of certain odors more inhibited in a peripheral lesion than in a central one? Does unilateral loss of smell occur with a lesion in one hippocampal lobe? How is the power to perceive an odor without the ability to name it to be interpreted? Do fluctuations in the sense of smell occur with lesions in one portion of the tract and not in the other?

Most of these questions still need further elucidation. A lesion in the nose when it leads to loss of smell usually causes complete bilateral loss. On the other hand, a destructive lesion of the *gyrus pyriformis* is said, at least in animals, to cause loss of smell in the nostril of the same side and some diminution of smell on the opposite side. The examination of the sense of smell is so uncertain even on intelligent human beings that tests on animals even by such eminent observers as Ferrier, Luciani and von Bechterew cannot be relied upon, especially when the latter claims to have noted unilateral *diminution* of the sense of smell in a dog. That the uncinate gyrus at least at times controls smell on both sides is illustrated by a case on which I was able to make careful tests (Fig. 2). No sensation of smell was present on either side, yet the lesion was only in one hippocampal lobe and his peripheral mechanism seemed normal.

The presence of subjective sensations of smell is evidence of a cortical lesion, the so-called uncinate fits that are most frequently encountered in pituitary disease, but there our knowledge ends. We are unable to distinguish a lesion of the olfactory bulbs from a lesion in the nose, nor do we know how to recognize a lesion of the fornix or the fibers of Lancisi.

A further point of great interest is how much reliance we may put on the ability of a patient to name odors as contrasted with his ability to recognize the presence of an odor without naming it. Does the naming of an odor constitute more of a mental process, and if it does, is this controlled by a different portion of the brain? If there is any occupation in which the olfactory sense is specially trained as tasting is in tea-tasters or the ear in a musician, the study of a pathologic lesion in the hippocampal lobe in such a patient might be of tremendous value.

The known facts in regard to taste are no more certain; in fact, the incontrovertible evidence is practically negligible. Even the peripheral portion of the mechanism offers difficulties of interpretation, and one wonders what the biologic significance of having

two peripheral pathways for carrying impulses of taste may be; the important role that taste plays in digestion may be the cause of this extensive peripheral nerve supply.

The taste fibers passing through the fifth nerve and those in the ninth carry the same sensations, and any differentiation is controlled in the cerebral portion of the pathway. About the ultimate cerebral terminations of the fibers there is much uncertainty, and in this instance the doubt is anatomic as well as physiologic. One group of observers believes the cortical center for taste lies in the hippocampal lobe nearer the convexity of the hemisphere (Fig. 1), while another group believes the center lies in the operculum near the motor center which controls the movements of the tongue and lips (Fig. 3). The most important investigation of this subject has been carried out in von Bechterew's laboratory by several of his assistants. A critical analysis of their work inevitably leads one to the conclusions that testing taste on animals is very

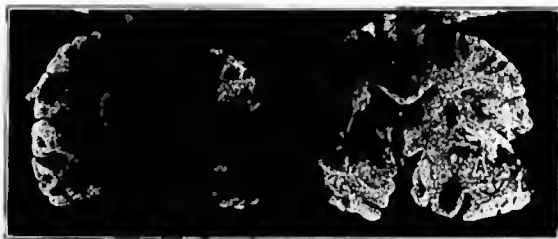


FIG. 2.—The tumor was an infiltrating glioma located at "A."

uncertain. These investigators have concluded that there is more or less marked diminution in the sense of taste and that this *diminution*—not loss, I would have you note—is more marked on the side opposite the lesion than on the same side. Such fine distinctions I feel confident are not possible in animals, since at least in my experience they are hardly ever possible in human beings. Furthermore, all taste tests to be reliable should be carried out with the patient's tongue protruded, since substances diffuse so rapidly on the moist tongue. In these experiments the test substances were introduced with a pipette on one or the other side of the tongue. The clinico-pathologic evidence is excessively meager, so that we are inevitably led to the conclusion that further studies on the sense of taste are urgently needed if we are to be able to make use of these sensory phenomena in more accurately determining the significance of disturbances in taste.

I have thus far dealt with the two special senses about which we know the least. Fortunately our knowledge of sight and hearing

is far greater; in fact, the mass of evidence, both experimental and pathologic, is so huge that a clear analysis of all this work would take much longer than is possible here.

I shall therefore select certain phases which bear particularly on the question of differentiating peripheral from central lesions.

1. Disturbances of ocular movements.
2. Disturbances of vision.

The movements of the eyes are controlled by the third, fourth and sixth nerves and their nuclei in the pons. Movements of the eyes can also be brought about by stimulation or destruction of the anterior corpora quadrigemina, that portion of the optic thalamus known as the pulvinar, the occipitotemporal region of the cortex and a center in the second frontal convolution (Fig. 4).

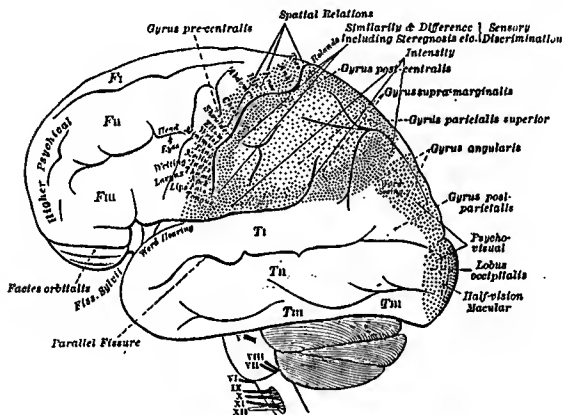


FIG. 3.—From Purves Stewart.

Though we are dealing here with motor phenomena these movements form such an intimate part of the visual mechanism that I feel justified in considering them.

The characteristic picture of an ophthalmoplegia needs no consideration here. The differentiation of irritative lesions of the peripheral nerves from lesions of the cerebral regions just mentioned, offer however, a real problem.

Experimentally it has been shown that stimulation of the anterior corpora quadrigemina brings about a spasm of accommodation and conjugate deviation of both eyes to the opposite side. This conjugate deviation may be to the side, upward or downward, depending on which portion of the quadrigeminal body is stimu-

lated; marked convergence of both eyes can also be brought about by stimulation of this region. All these movements can also be produced by stimulating the occipital cortex, the frontal region and the optic nerve, but if the corpora quadrigemina are destroyed stimulation of the occipital lobe no longer produces these movements, though they still can be produced by stimulation of the frontal center.

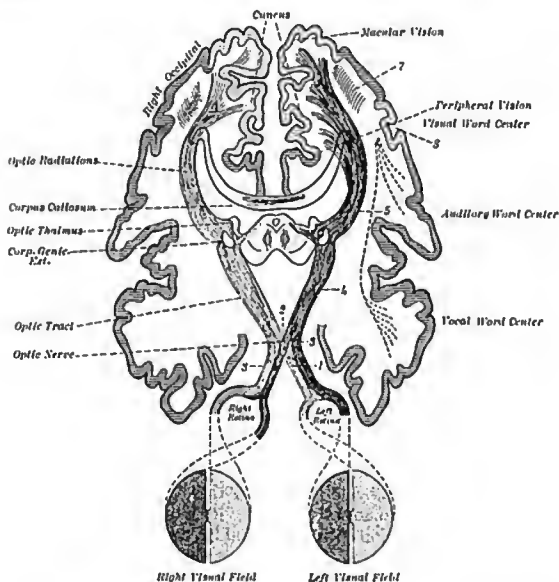


FIG. 4.—From Purves Stewart.

This is clear evidence to physiologists that the corpora quadrigemina are merely reflex centers and not primary centers, but this explanation does not materially help the clinician to differentiate lesions of these various regions.

Is our knowledge of these various regions adequate to enable us to recognize when they are diseased, and if so what are the salient points? That most accurate differentiation would be of great value is illustrated by the common example of conjugate deviation of the eyes which is noted in cerebral hemorrhage due to vascular disease and cerebral hemorrhage due to trauma.

When the symptoms are compared in parallel columns the dif-

ferences seem (see table) quite clear, but when applied to a particular clinical case the problem is by no means simple. For example, the

Frontal.	Occipital lobe.	Anterior corpora quadrigemina.	Optic nerve.
Conjugate deviation of the eyes. Vision intact.	Conjugate deviation of the eyes. Homonymous hemianopsia or homonymous visual impressions.	Conjugate deviation of the eyes. Homonymous hemianopsia.*	Partial, complete or no ophthalmoplegia depending on involvement of 3d, 4th or 6th nerves.
Weakness or twitching of face of opposite side.	No motor involvement.	Hemiparesis or no motor disturbance depending on size of lesion.	Blindness of one eye or contraction of field depending on degree of involvement of nerve.
Pupillary reflexes undisturbed.	Pupillary reflexes undisturbed. Cortical blindness (Seelenblindheit).	Pupillary reflexes abolished.	No motor involvement. Pupillary reflexes abolished if blindness complete.

significant differential point between a frontal and occipital lesion is a field defect, but so often unconsciousness is present to thwart one in distinguishing frontal from occipital conjugate deviation. Experimentally Gerver, in Bechterew's laboratory, observed that ocular movements were more readily produced by frontal-lobe stimulation than by stimulation of the occipital lobe. How to make use of this interesting experimental observation in a clinical case I am at a loss to know. There are a few associated symptoms that may be helpful in clarifying these confusing pictures. If, associated with conjugate deviation, there are irritative motor phenomena, Jacksonian convulsions, it is more probable that the conjugate deviation arises from the frontal region rather than from the occipital region, since the frontal center lies so near the motor cortex. On the other hand, visual hallucinations would point rather to an occipital lesion, while the presence of high temperatures must always suggest the possibility of a ventricular involvement and consequently a lesion of the corpora quadrigemina, which protrude into the ventricle. The evidence of so-called heat centers in the brain is not to my mind conclusive in spite of the extensive studies of Barbour. In some unpublished experiments carried on in my laboratory by Captain Philip Green the evidence was not so positive that we felt convinced of the existence of a heat center or of its definite location. In other words, we are in the strange situation that though knowing quite accurately the functions of some of these centers we are often unable to determine by our present methods which center is involved.

The act of vision, to which we turn next, would seem to be a sub-

ject in which most if not all the problems had been solved. I know of no other subject on which the concentrated efforts of such a group of great minds has been focussed. To name but some of these investigators: Flourens, Munk, Ferrier, Goltz, Luciani, Hitzig, Monakow, Bechterew and your honored guest from Holland. Yet there are a number of questions that still are unsettled.

In weighing the experimental evidence and determining the apparently great differences in some results, one point seems to have received insufficient attention, namely, that most mammals on account of the anatomic position of their eyes have complete monocular vision or at most partial binocular vision, but none of them except the monkeys have practically complete binocular vision as occurs in the human being, where each eye practically covers the same field of vision. The diametrically opposite results obtained on dogs by different investigators might readily be explained by the different type of dog used. Thus a dog with a long snout, like a hound or setter, can have no overlapping of his visual fields, while the bull-dog with the abbreviated nose undoubtedly has at least partial binocular vision. There is no doubt whatever in the case of man that each eye has representation in each visual cortex, and the most recent observations indicate though it is not absolutely proven that each macula is only represented in one cortex. It is also certain that various types of homonymous hemianopsia are produced by lesions of one optic tract after it leaves the chiasm, and similar defects may be produced at various points along this pathway (Fig. 4). Though phases of these questions are not absolutely clear the most profitable fields for investigation are the functions of the visual cortex and its location. Combining the anatomic studies of Campbell and Brodmann with the physiologic observations we know that the occipital region contains the cortical center of vision and that this area overlies Gennari's streak (Fig. 4, "A"), but what the functions of this cortex are is a debatable question (Fig. 5).

Von Bechterew believes that there are two centers, one lying on the mesial surface and one on the convexity, and that each of these centers has two separate functions, one controlling the peripheral vision and the other central vision. He furthermore believes that the center on the convexity is a higher one than that on the mesial surface and that the loss of this function leads to what the Germans have called "Seelenblindheit", the inability to interpret visual impressions. An integral part of this conception is the ability to interpret written words and musical notes. Apparently there is some further differentiation between the right and the left visual cortex, and one is forced to the conclusion that probably the left cortex in right-handed individuals plays a greater role in the interpretation of such visual impressions. In spite of these fairly well-established facts it remains difficult to differentiate

a lesion of the lateral geniculate body and the pulvinar from lesions in the optic radiation as they stream through Gennari's streak into the occipital cortex. Some observers claim that the geniculate body is more concerned with macular disturbances than the visual cortex, though the recent studies by Gordon Holmes

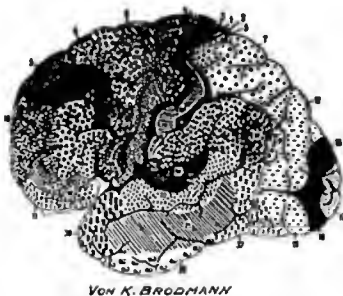
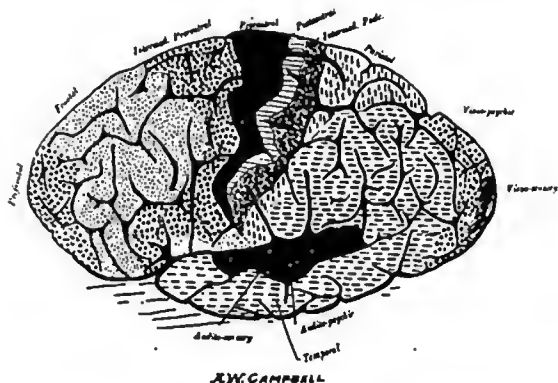


FIG. 5.—Areas of cortical function.

and by Lyster on gunshot wounds of the occipital region seem to establish the fact that the macula is represented in the cortex as is the rest of the retinal field, but each macula is represented in but one cortex—the cortex of the same side. There are those who feel that this question in regard to the macula is not yet proven. If it is so, however, the special function of the geniculate body

becomes more of a mystery than ever. We know that a lesion of this body produces a homonymous hemianopsia indistinguishable from a hemianopsia produced by lesion of the optic tract at various other points.

The interpretation and recognition of colors remains a still more uncertain question. The accepted theories of light perception all assume that the differentiation is carried out in the retina, but it seems that the cortex must exercise a further controlling influence. This is suggested by the cases in which subjective visual disturbances are noted during an attack of focal epilepsy which is due to an irritative focus in the occipital region. These rapidly passing color impressions that such patients experience are difficult to record. They are so rare that one observer rarely sees many, consequently the method of studying the phenomena is not uniform. Whether the succession of colors is always the same has as far as I know not been determined. On account of the intricacy of the problem we are offered here a most fascinating field for further investigation.

We come finally to a consideration of the sensations which are transmitted through the eighth cranial nerve. In considering these we must keep in mind that we are dealing with two separate and entirely distinct functions, those carried by the auditory portion and those by the vestibular portion.

The difference between the functions of the central and peripheral portions of the auditory mechanism is so much more clearly recognized than in the case of the other special senses that our attention is centered upon other aspects which offer greater difficulties in diagnosis.

1. The interpretation of those auditory impressions that are associated with speech and an appreciation of musical sounds.

2. The difference between disturbances of the median geniculate body, the posterior corpora quadrigemina and the auditory cortex.

The cases in which there is a partial impairment of hearing afford such opportunities for study. The first temporal convolution contains the auditory center; apparently it is divided into at least two portions, one of which controls tone and sound impressions while the other controls word impressions. Since man alone uses speech, the existence of this center in the posterior portion of the first temporal convolution can be only convincingly recognized by pathologic studies on human brains; the attempts to differentiate these centers in dogs, by supposing that howling is mere phonation, while barking, whining and snarling are akin to articulate speech, seem to me rather farfetched.

The appreciation of sounds, musical or otherwise, seems to be a bilateral cerebral function, while the *understanding* of language is confined to the left cortex in right-handed individuals.

This idea one does not at first readily accept, for speech seems

a far more universal attribute of man than does a musical sense; but a distinction must be drawn between the mere perception of sounds and the highly specialized development of the musical sense that is observed in musicians. This latter function seems to be confined to the left temporal convolution, as is speech, but the mere appreciation of sound has unquestionably bilateral representation. An individual can still hear sounds if his left auditory center is destroyed. The interpretation of auditory impressions, however, is carried out by the center in the left temporal lobe. In addition, a valuable diagnostic sign is the presence of subjective auditory phenomena. Such an individual may complain of hearing a variety of sounds or voices. These sounds may vary from time to time or always be the same. A patient with a localized tuber-



FIG. 6.—L. E. M. Surgical No. 7755. Tuberculoma, 4 x 5 cm. in size, removed from auditory speech center. Present eighteen years.

culous process, a solitary tubercle (Fig. 6), said that he had for years thought he heard church bells ringing. Just why he should have had only this subjective sensation when the greater part of his auditory center was involved is difficult to explain. For diagnostic purposes it is of great importance to keep in mind that immediately underneath the cortical center of hearing lies a region—Wernicke's field—in which the fibers from the olfactory, gustatory and auditory centers lie close to one another. In consequence an irritative lesion may produce subjective sensations arising from these three centers.

The functions of the median geniculate bodies and of the posterior corpora quadrigemina are by no means clear (Fig. 4). Gerver is of the opinion that through these centers movements of the eyes

and ears are probably brought about, but the evidence is not convincing. The best opportunity for studying these regions, particularly the posterior quadrigeminal bodies, may be found in observations on early cases of pineal gland tumor. In the few cases of this sort that have been studied no minute investigations that might elucidate this point have been carried on.

The posterior quadrigeminal body also has some connections with the vestibular portion of the eighth nerve. In some investigations in my laboratory with Dr. B. Y. Alvis we have found definite anatomic connections with Deiters's nucleus, but these studies threw no light on the vestibular function of the quadrigeminal body.

The importance of differentiating the central and peripheral portions of cranial nerves is brought home to us most strikingly when we note the enormous literature that has grown up about the vestibular portion of the eighth nerve. It is, I think, fair to say that practically the entire subject of neuro-otology occupies itself with this differentiation. Interest has been centered on the functions of the nerve tracts after they leave the labyrinth.

The finer movements of coordination are conceded to be functions of the cerebellum. Bárány has located certain centers for these movements in the lateral lobes of the cerebellum. His evidence comes primarily from the study of cases of cerebellar tumors in which after operation he has frozen the overlying skin with ethyl chloride and thus affected the cerebellum. Victor Horsley and Clarke showed how inert the cerebellar cortex was. Electric currents of great strength produced no symptoms that they could recognize. I noted no disturbances when I stimulated the cerebellum on a conscious patient some years ago. This of course does not mean that such centers do not exist. The methods of examination were different. The electric current has a stimulating effect while the freezing method has a temporary paralytic effect. My own observations on one patient have the further objection that the position of a patient lying on his face during a cerebellar operation is awkward for purposes of examination. Some recent clinical studies by Gordon Holmes on gunshot wounds of the cerebellum, however, do not support Bárány's contentions that there are separate centers in the cerebellum that control movements of the extremities. Any statement from such a careful observer as Holmes carries great weight. The pass-pointing and ocular movements that are produced by stimulation of the semicircular canals are not brought about by any one center but by interaction of several centers.

Movements of the eyes may be produced by action of the ocular nuclei, an area in the frontal lobe, the lateral geniculate bodies and the anterior quadrigeminal bodies; movements of the extremities are brought about by the anterior horn cells in the cord, by

the motor cortex and probably also by the cerebellum; vestibular stimuli are transmitted from Deiters's nucleus to the roof nucleus of the cerebellum, the olivary bodies, the posterior corpora quadrigemina. Through this maze of tracts and nuclei Jones and Mills have had the great courage to attempt to explain the mechanism by which the Bárány phenomena are produced (Fig. 7). Dr. Alvis and I have worked for several years with the idea of throwing

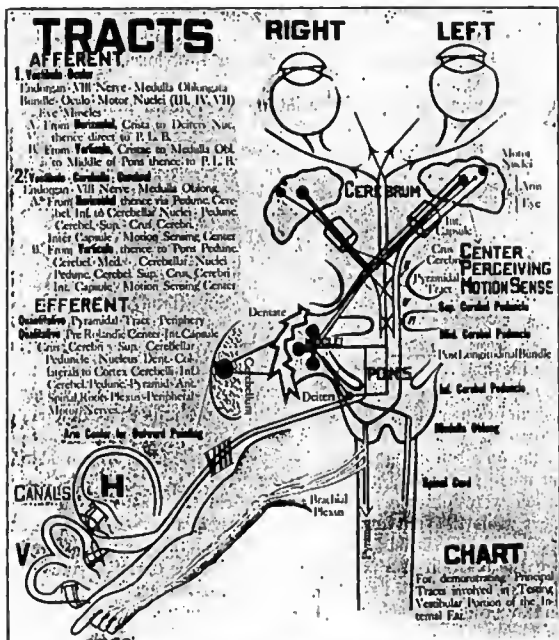


FIG. 7.—Tracts involved in testing vestibular portion of the internal ear.

some further light on the anatomy and physiology of this problem. We believe our work confirms certain points, disproves some others, but leaves other questions in a rather unsettled state. We have found a definite connection between Deiters's nucleus and the posterior corpora quadrigemina. No fibers could be demonstrated passing from Deiters's nucleus to any of the lateral lobes of the cerebellum in which the centers described by Bárány lie. Isolated lesions of Deiters's nucleus (Fig. 8) or the vestibular nerve never

produce the violent circus movements which had come to be considered as a result of von Bechterew's work diagnostic of Deiters's nucleus lesions. Such movements when they occur always are due to a lesion of the neighboring inferior or superior cerebellar peduncles.

The connections between Deiters's nucleus and the ocular nuclei as described by Fraser were not demonstrable, so that the vestibulo-ocular phenomena must be brought about by a different pathway.

We were unable to determine whether the course of the fibers from the vertical and horizontal canals followed different pathways or not on their way to the roof nucleus of the cerebellum. Again, it was not possible to demonstrate, what on theoretical

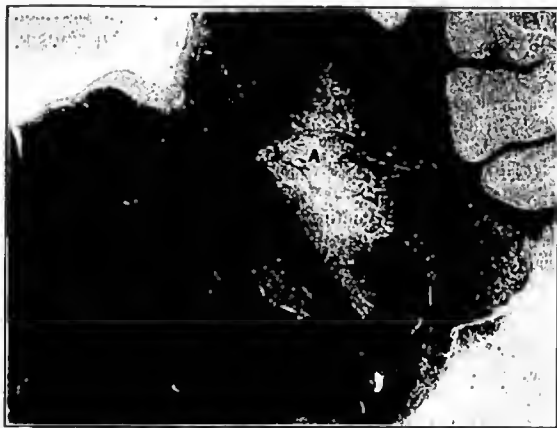


FIG. 8.—Lesion of Deiter's nucleus at A.

grounds we believe, that each semicircular canal is controlled by a separate group of cells in the Deiters's group. The most important single fact that has been established is that the tracts by which these phenomena are carried out lie immediately about the aqueduct of Sylvius and are therefore most easily affected by changes in intracranial pressure. Internal hydrocephalus is an almost invariable accompaniment of spreading processes in the posterior fossa, and the pressure of the obstructed cerebrospinal fluid frequently brings about the symptoms that have been considered evidences of local lesions. This complicating factor tends to make our knowledge of the functions of the different portions of the vestibular mechanism less certain than we have been led to believe.

If we consider for a moment what has been said this evening it must be apparent that I have passed over for the most part the facts that are well known and have tried to bring out in high relief some of the unfinished or thus far unanswered problems on the borderline of your specialties and neurology. To the earnest student these are the questions which it is most desirable to grapple with. The more difficult of solution the more worth while they are. The vast material that passes through the hands of the members of this organization contains within itself the evidence that will clear up these disputed points. To collect, correlate and subject to critical analysis all this material by uniform methods would be a fundamental and enduring service.

ORTHOSTATIC ALBUMINURIA: THE STUDY OF AN UNUSUAL CASE.*

By WYNDHAM B. BLANTON, M.D.,

RICHMOND, VIRGINIA.

CASE HISTORY. Female, white, aged twenty-three years, single, school teacher.

Chief Complaint. Fainting.

Family History. Father living and well; mother died at forty-eight years of paralysis; one sister living and well; no brothers. No family history of rheumatism, gout, diabetes, cancer, tuberculosis or nephritis.

Past History. Patient has had the common diseases of childhood. Has not had scarlet fever, diphtheria or many sore-throats. Ten years ago she suffered with a suppuration of the glands of the neck. Eight years ago a chronically diseased appendix was removed. She has had pneumonia. Several years ago she contracted the "fever" in South America.

Personal History. Sleeps from 10 P.M. to 7.30 A.M. Is fond of outdoor exercise and takes more than the average quantity. Teaches school. Meals are regular; eats slowly, moderately and sparingly of meat. Drinks one cup of coffee a day. Menstruation established at twelve, regular, lasting four or five days. A moderate amount of pain precedes.

Present Illness. Has been given to fainting all her life. Fainted one month ago at the theater; again last night when she witnessed a harrowing picture; again today she felt that she was going to faint in school. Alarmed at the frequency of these attacks, she is seeking advice. She has recently lost some weight—from 106 to 95 pounds—and does not feel as strong as she did. She has also tingling in the hands and the feet.

* Read before the Richmond Academy of Medicine, November 8, 1921.