

into the pons. Hemorrhages or a thrombosis often give limited symptoms. One such case is now under our observation. This patient, a physician of 45 years, suddenly developed numbness on the entire left side of the body with a complete motor right fifth nerve palsy and a partial weakness of the right sixth nerve. He had besides limited cerebellar disturbances in the limbs of the involved side which were best brought out by the Bárány tests. In this instance the diagnosis was made by the presence of the fifth and sixth nerve palsy on the side of the lesion, the sensory symptoms on the opposite side due to the irritation of the median lemniscus and the slight cerebellar asynergy on the side of the lesion.

The conclusion can be drawn that in lesions of the middle cerebellar peduncles the associated phenomena consist of the fifth or sixth nerve symptoms on the side of the lesion, with sensory and motor phenomena on the opposite side.

Lesions of the Inferior Cerebellar Peduncles.—Such tumors are very rare and we have never seen one limited to one or both of the inferior peduncles. We have, however, seen a number of tumors of the cerebellum in which there has been an extension of the growth into one or both peduncles. In such growths the associating phenomena, if the lesion extends into the medulla, should consist in implication of the vestibular tract, and disease of the ninth, tenth and twelfth cranial nerves.

Lesions of the Cerebellopontile Angle.—The diagnosis of tumors in this area as a rule is not difficult. We have encountered cases, however, in which such diagnosis had been made, only to find that this angle was invaded secondarily by tumors growing from the cerebellum and more rarely from the pons. The differential diagnosis is important from the surgical standpoint, for it is readily seen that tumors growing from the pons or cerebellum offer little hope for surgical removal.

Tumors growing in this angle are usually fibromatous and more rarely fibrosarcoma. We have encountered a number of cysts, but these are generally parts of gliomatous tumors growing from the cerebellum. Most tumors grow from the eighth, and more rarely from the seventh, fifth, or sixth cranial nerves, respectively. The first symptoms are usually referred to the cranial nerve from which the tumor grows and in the course of time the seventh, sixth and fifth nerves become implicated. Of course, the symptoms depend on the size of the growth. In small tumors no pressure is exerted on either the pons or cerebellum. In a well-developed case, besides the cranial nerve symptoms, because of the pressure on the pons, there are motor symptoms on the opposite side and more rarely sensory phenomena.

Because of the pressure on the anterior part of the cerebellum, both the superior and inferior surfaces are involved, this causing cerebellar asynergy of the limbs on the same side. In cases in which the angle is invaded secondarily from the cerebellum or from the pons, the cerebellar symptoms are always much more marked, and it is because we have not been sufficiently careful in the differentiation of the cerebellar phenomena that errors in diagnosis have arisen.

We wish to emphasize here that in the usual tumor growing from the cerebellopontile angle the cerebellar symptoms are *not very marked* and that unless the tumor is very great the asynergy will be limited only

to the arm and leg on the side of the growth, and that if there is in addition to the cranial nerve symptoms cerebellar asynergy in the trunk and limbs on both sides, it is probable that the tumor grows either from the cerebellum or more rarely from the pons.

Of course, if the progress of the symptoms is carefully noted, in the latter type of case the cerebellar phenomena should occur first and the cranial nerve symptoms last. But unfortunately we are not always permitted a clear-cut history, and it is because of this that we desire to emphasize this important point in differential diagnoses.

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GALVANOMETRIC STUDIES OF THE CEREBELLAR FUNCTION*

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The object of this study has been to ascertain, if possible, the mechanism of the cerebellar function, as deduced from the phenomena consequent to its lesions. It will be necessary, therefore to formulate at the outset a clear conception as to which of such phenomena are essentially cerebellar in origin. The cerebellum is adjacent to so many important structures and forms such intimate connections with the vestibular system and its oculomotor tracts that many of its lesions, experimental, as well as clinical, but especially the latter, very frequently implicate these other structures, producing their corresponding symptoms. On reviewing the literature we find, therefore, that a number of symptoms have from time to time been ascribed to this organ which have not had their genesis in it at all.

THE CEREBELLUM AND SENSORIUM

Lesions of the cerebellum do not produce disturbances of sensibility, and if these are present, they are invariably due to the involvement of the sensory tracts or nerves found in its vicinity. This is attested by such a great number of observations that it may well be considered an indisputable fact.

What was said with regard to general sensibility applies with equal force to that type of sensation which is implied by the general term of the "muscular sense." As is well known, this type of sensation comprises all those sensory reactions which, originating in the motor organs and their accessories (tendons, joints, etc.), provide us with information as to their state of tension, their direction in active and passive movements, and incidentally aid us in the appreciation of our posture in space. The cerebellum has been thought to be the center of the muscle sense by such noted observers as Lussana,¹¹ Hitzig, Munk¹² and of late Lewandowsky, and the spinocerebellar tracts have been identified by them as the carriers of the so-called proprioceptive stimuli. It is an undeniable fact, how-

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* Because of lack of space this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints. A copy of the latter will be sent by the author on receipt of a stamped, addressed envelope.

11. Lussana, F.: Jour. de la physiologie de l'Homme, 1862, v, 418; vi, 1863, 169, etc.

12. Munk, Quoted by André-Thomas: Cerebellar Functions, English Trans. by W. C. Herring, New York, 1912.

ever, that pure cerebellar lesions do not give rise to loss or impairment of the muscle sense and, if this is present, there is invariably found an associated lesion of the posterior columns or their nuclei.¹³ Patients with cerebellar disease have complete orientation toward the surrounding world, are fully conscious of all changes in posture during active and passive movements and present no Romberg sign (swaying on being deprived of the aid of vision). As pointed out by Luciani, the dog deprived of a lateral lobe of the cerebellum even anticipates the disturbances that will ensue on walking or prehension of food, and to obviate a fall seeks out some support for the side of the lesion.

THE CEREBELLUM AND MOTILITY

The phenomena consequent to cerebellar lesions belongs to the motor sphere and not to the sensory. This is almost universally conceded now by both physiologists and clinicians. The disturbances manifest themselves, however, in such a large variety of phenomena, owing chiefly to concomitant lesions of other structures, that their interpretation has for a long time been exceedingly difficult. Of late, however, thanks to our increasing knowledge of the anatomy and physiology of the cerebellum as well as of its adjacent structures, this difficulty has been largely removed, and we are able to dissociate the true cerebellar phenomena from the others.

FORCED MOVEMENTS, NYSTAGMUS, ETC.

It is pretty definitely established that such phenomena as forced movements, the circus movements and rolling movements in animals and the so-called imperative movements in man, are not cerebellar in origin. And the same is true of the nystagmus, the conjugate deviation of the eyes

and the characteristic attitude of the head so frequently observed after unilateral ablation of the cerebellum in animals and occasionally in cerebellar disease of man.

These phenomena are essentially vestibular in origin, being due to a lesion of the vestibular complex itself or its oculomotor tracts. Muskens,¹⁴ who made an extensive study of these phenomena in their relation to the pathologic findings at necropsy, found that they are associated with lesions in the paracerebellar nuclei, the vestibulomesencephalic tracts in the posterior longitudinal bundle and the descending vestibulospinal tracts. They are, at any rate, not cerebellar symptoms. The whole lateral lobe of the cerebellum may be removed without producing the slightest indication of these phenomena. I have personally performed such operations in about twenty-five animals (dogs and cats) and can say without hesitation that forced movements as well as nystagmus are not cerebellar in origin.

I have seen no instance of rolling movements or a tendency to it in pure cerebellar lesions. They were, however, extremely vehement and almost uncontrollable whenever there was an associated lesion of the vestibular system. These movements in my cases were invariably toward the side of the lesion in accordance with the results of Schiff,¹⁵ Ferrier and Turner,¹⁶ André-Thomas, and Muskens¹⁴ in lesions of the cerebellum, and those of Bechterew¹⁷ and Breuer¹⁸ after section of the eighth nerve.

THE PHENOMENA OF CEREBELLAR LESIONS

The phenomena that are purely cerebellar in genesis are two, the ataxia and the tremor. I am using the term "ataxia" in its general sense to denote irregularity or perversion of muscular action without reference to its nature, and in contradistinction to loss of movement such as is found in paralysis. Personally I have never been convinced as to the existence of hypotonia in cerebellar lesions, a symptom on which Luciani laid so much stress. In my experience, which is in accord with that of Ferrier, it is very inconstant. I have noted it only in very extensive lesions, in lesions which have involved also the vermis and its nuclei. The nucleus tecti, as established by the morphologic studies

of Weidenreich¹⁹ and others, may be looked on as a part of the paracerebellar nuclei, which, from their rôle in the production of decerebrate rigidity (Thiele²⁰) are known to dominate the tonicity of the body musculature. The presence of hypotonia in such severe lesions of the cerebellum is thus easily explained without attributing it to a disturbance of the chief function of the organ. The ataxia and tremor, on the other hand, are constant and marked, and found in lesions limited to the lateral lobes and in the absence of any involvements of the vestibular

complex. And it is to interpret these phenomena that our efforts should be directed in attempting to decipher the complex and mysterious problem of the mechanism of the cerebellar function.

THE INTERPRETATION OF THESE PHENOMENA

There have been numerous theories advanced in explanation of these phenomena. Flourens, Vulpian,²³ Renzi and Dickinson interpreted them as disturbances of coordination, attributing to the cerebellum that function which, according to Foerster's definition of coordination, enables the organism by means of its muscles to produce external effects which fulfil a definite purpose or a set task, accomplishing these effects

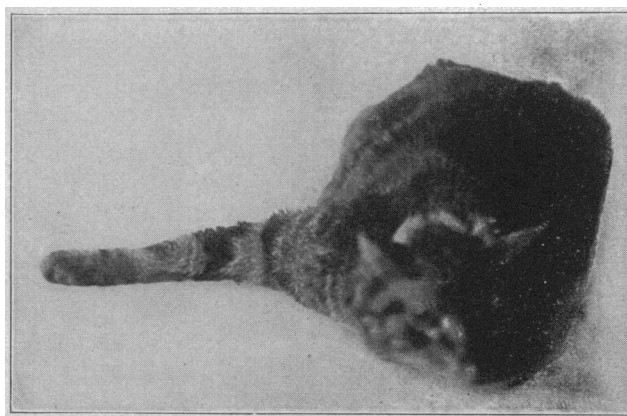


Fig. 1.—Cat after removal of left motor area of the cerebrum and right lateral lobe of the cerebellum. From photograph taken fifteen days after the first operation and fifteen days after the second. Note marked extension of right forelimb on prolonged standing. The extension is not due to a spasmodic condition of the forelimb, as it takes place gradually and is brought about by the weight of the body forcing the paretic limb out.

13. André-Thomas: *Cerebellar Functions*, p. 144.
14. Muskens, L. J. J.: *Jour. Physiol.*, 1904, xxxi, 212; *Brain*, 1914, xxxvi, 352.

15. Schiff, M.: *Lehrbuch der Physiologie*, 1858, I, 354.
16. Ferrier and Turner: *Phil. Tr. Roy. Soc.*, London, 1894, clxxxv, B, p. 730.
17. Bechterew: *Arch. f. d. ges. Physiol.*, 1882, xxx, 318.
18. Breuer: *Arch. f. d. ges. Physiol.*, 1891, xlviii, 250.
19. Weidenreich: *Zeitschr. f. Morphol. u. Anthropol.*, 1899, i, 259.
20. Thiele, F. D.: *Jour. Physiol.*, London, 1905, xxxii, 355.
21. Vulpian: *Leçons sur la physiologie générale et comparée du Système Nerveux*, Paris, 1866.

with the smallest possible waste of energy. Bouillaud,²⁴ Ferrier and Thomas thought the cerebellum to be an organ for the maintenance of body equilibrium and endeavored to explain the phenomena of the lesions on that hypothesis. Babinski,²⁵ basing his opinion on clinical studies, proposed the theory of asynergia of movements, according to which it is essentially lost in cerebellar lesions in the harmonious association of simple movements necessary for the execution of a complicated movement and he endeavored to prove it by certain clinical tests. Babinski admitted, however, that in addition to asynergia there is also a dysmetria which is the cause of the excessive and ill-proportioned movements in cerebellar lesions. As to the asynergy, it is of course clear that it is merely a broad, general term for incoordination, and this conception of the nature of the disturbances aids us but little in understanding the mechanism of their production.

According to Holmes and Stewart the cerebellar phenomena are the result of defective cooperation of muscles and their antagonists. The cerebellum, according to this view, effects relaxation of the extensors while the flexors are in a state of contraction, and relaxation of the flexors while extension is in progress, the same being applied to the abductors and adductors.

OBJECTIONS TO THE FOREGOING VIEWS

There are two points in common to all these theories: First, they all ascribe to the cerebellum a function which is totally distinct from that possessed by any other part of the nervous system, a function that is closely allied with motor activity but still different from it in a vague, indefinite way. Secondly, the function of the cerebellum, according to any of these theories, cannot be expressed in the terms of a single neuron effect. It is of course evident that coordination of movement, synergy of movement, etc., an effect which manifests itself simultaneously in a large group of muscles, widely isolated from each other and differing in their mode of action, cannot be the expression of an individual neuron. It must be the resultant effect of a large number of neurons all functioning at the same time. This conception of function is, however, entirely unnecessary for our understanding of the activity of any other nerve structure, in which case each unit, whether motor or sensory, may exhibit its individual function independently of the other neurons and produce a corresponding effect.

Notable among these was Risien Russell, who maintained that unilateral ablation of the cerebellum was in his experiments followed by genuine paralysis on the side of the lesion, entirely identical with that following ablation of the motor area of the cortex. This is, however, denied by the majority of observers. There was no paralysis in the animals operated on by Bouillaud,²⁴ Dalton,²⁶ Longet,²⁷ Munk and

Ferrier and Turner.¹⁸ Personally, I am convinced that pure cerebellar lesions do not give rise to paralysis. One has only to compare the gait of an animal after destruction of the motor area of the cerebrum with that which follows unilateral ablation of the cerebellum to be impressed by the marked dissimilarity between these two conditions. In the former instance, the limbs on the side opposite to the lesion are distinctly paretic; they are unable to support the animal on that side; they bend under it and are frequently placed with the dorsal surface to the ground. In the latter condition, on the other hand, the limbs on the side of the lesion are generally in full extension; they are never placed with the dorsal surface to the ground, but are usually thrown violently forward or outward, and the frequent falls are therefore to be attributed to the ill-proportioned activity of the muscles rather than to loss of their activity.

GOWERS' HYPOTHESIS

Gowers,³³ as far as I can ascertain, was the only one to maintain, on purely theoretical grounds, that the cerebellum has no downward effect whatever, that it exhibits its influence on the cerebrum exclusively, this influence being inhibitory in character, and that cerebellar coordination is nothing else than coordination of

the activity of the rolandic cortex. His arguments in support of this theory were as follows: The cerebellum is connected with the periphery by means of afferent or upbearing tracts exclusively. The only efferent or outgoing tracts known to come from it pass by way of the superior cerebellar peduncle to the opposite hemisphere of the cerebrum, and it is inconceivable that an organ having no outgoing tracts to the periphery could

exert a direct motor effect on it. Continuing he said:

The will may alter any muscular contractions by which the upright posture is maintained, and yet without thus being deranged for an instance, and it is difficult to understand how this is possible if the two motor effects, the cerebral and cerebellar, take place apart, and equally difficult if we assume that they only come together in the subordinate centers in the spinal cord.

He therefore concluded that the cerebellum exhibits its function on the motor cortex of the opposite hemisphere of the cerebrum by way of its efferent tracts passing along the superior cerebellar peduncles, and that this function is inhibitory in character, in view of the fact that evidence at hand tends to show that when one set of cells exerts an influence on another set, this is in the nature of control, as witness the influence of the cortical motor cells on the spinal cells in the anterior horns. We must distinguish, said Gowers, between the continuous action of nerve cells, which by its nature needs control and restraint, and occasional action, which requires stimulation, and conceived as excitation.

According to this conception, the phenomena consequent on cerebellar lesions are only indirectly cerebellar; they are primarily cerebral effects, and, like the

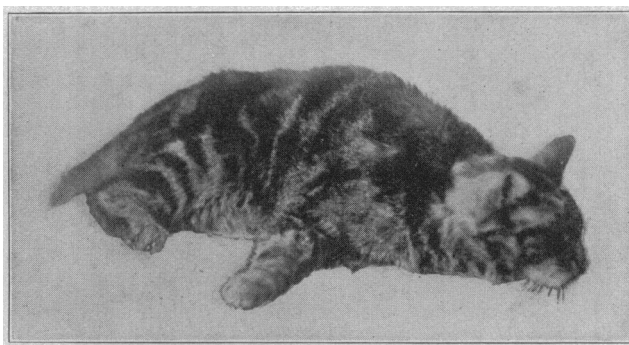


Fig. 2.—Same cat as shown in Figure 1. In standing posture for two or three minutes. Considerable abduction of both limbs on right, paretic side.

24. Bouillaud: Arch. gén. de méd. 1827, xv, 64.

25. Babinski, J.: Rev. neurol., Nov. 9, 1899, p. 784; Feb. 2, 1901, p. 260; April 18, p. 422; May 15, 1902, p. 470; Nov. 15, 1902, p. 1013.

26. Dalton, J. C.: Am. Jour. Med. Sc., 1861, p. 84.

27. Longet: Traité de physiologie, Ed. 3, iii, 466.

33. Gowers, W. R., Lancet, London, 1890, i, 955.

increased reflexes and spasticity after lesions of the upper motor nervous system, are due to removal of inhibition on the nerve cells directly concerned in their production.

It is evident from the foregoing that, according to Luciani, Hughlings Jackson and others, the disturbances of motility in cerebellar lesions are the expression of diminished or lost functional activity on the side of the lesion; according to Gowers, on the other hand, they are the result of exaltation of such activity.

It occurred to me that the problem could be taken out from the realm of pure speculation and solved by means of the galvanometer.

SOLVING THE PROBLEM GALVANOMETRICALLY

It is well known that living tissue in a state of activity exhibits four kinds of phenomena, mechanical, chemical, thermal and electric. Of all these the electric phenomena are the most faithful; they are to be observed, especially so in the case of nerve tissue, when none of the others can be elicited.³⁴ Among the electric phenomena is the state of negativity which excited tissue assumes with relation to the quiescent part thereof. On connecting two such parts with a galvanometer, with the circuit closed, a current is seen to pass through it, as evidenced by the deflection of the needle, from the quiescent to the active part. The former is therefore in its external or galvanometric relations positive to the latter. In completing the circuit and passing through the tissue the current of course passes in the opposite direction, from the active to the resting part. Active tissue, in other words, behaves electrically toward quiescent tissue as the zinc element of a voltaic couple behaves toward the copper element—it is with regard to the outer or galvanometric circuit, negative to the latter. While this electric phenomenon, the so-called action current, is generally studied by means of the negative variation, or the diminution of the preexisting current of injury which accompanies the excitation of the tissue, the latter, says Hermann,³⁵ is only a special case of a more general phenomenon, which is, that active tissue is, properly speaking, invariably negative (or "zincative," according to Waller) to resting tissue quite independently of previous preexisting currents. The action current is also entirely independent of the agency which provoked the activity in the tissue. The stimulus may be chemical, mechanical, thermal or electric; it may be direct or indirect. Gotch and Horsley³⁶ have thus demonstrated that the action current manifests itself in a peripheral nerve, not only on direct excitation of the nerve, but also on stimulation of the contralateral and functionally related motor area of the cerebrum.

That natural nervous activity is, like that resulting from artificial stimulation, accompanied by the development of a negative state, has been demonstrated by A. Beck,³⁷ Alcock and Seemann,³⁸ Einthoven³⁹ and others. Beck removed the brain, spinal cord and sciatic nerves of a frog *en bloc* and placed the preparation on a glass plate. He then applied nonpolarizable electrodes to various parts of the nervous system and connected them with a galvanometer. He found that there was uniformly an electrical difference of such a nature that the proximal, more active parts of the nervous system were negative to the distal parts. Alcock and Seemann and Einthoven studied the impulses which traverse the vagus during natural respiration and found that a state of negativity invariably passes along the nerve with each inspiratory excursion. The latter also observed minor effects which accompanied the large waves of the inspiratory acts and corresponded with rhythmic contractions of the heart. A natural corollary to this should be that, if active tissue on one side of the body were connected by means of a galvanometer with the corresponding quiescent tissue on the other side, the former would be negative to the latter. That this is so for direct stimulation has been demonstrated by Hermann and Luchsinger, Waller and others in their study of skin currents. On connecting the pads of the hind paws of a cat with the galvanometer and stimulating the sciatic nerve for one of these, the corresponding pad has been found to be negative to the other. In this case the current in its internal circuit has to traverse, of course, from one side of the body to the other. That this holds true for indirect stimulation of tissue I have proved to my satisfaction by the following experiment:

With the cat under ether anesthesia, by means of a tracheal cannula I carefully exposed and isolated both sciatics. After drying all hemorrhage, I freed the nerves from contact with

any of the surrounding tissue—which of course had been injured during the operation—and placed them on the boots of nonpolarizable boot electrodes, which had been soaked in Ringer's solution for the preceding twenty-four hours. I connected these with a galvanometer with a contact key in the circuit. The galvanometer employed during this and the other experiments to be described was of the Thomson type and manufactured by Elliot Brothers, London. It had a resistance of 20.74 ohms and its sensitiveness was such that at 37.5 cm. of scale distance 1 cm. deflection, registered 0.0000154 ampere of current, so that its constant (K) was 0.00155 ampere ($K = \frac{2 I \times D}{d}$).

While an experiment was in progress the nerves were kept moist with cotton sponges soaked in normal salt solution at a temperature of 38 C., which were removed during each observation so as to prevent any possible short-circuiting. The sponges were always applied to both nerves at the same time in order to avoid any possible differences of surface temperature.

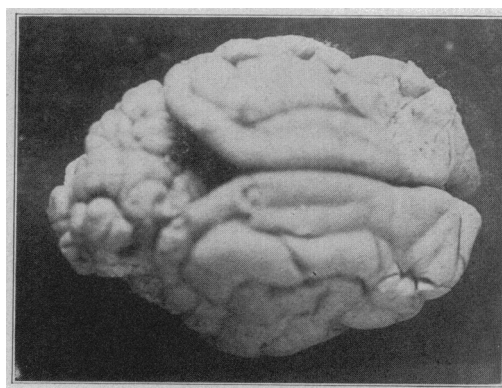


Fig. 3.—Brain of cat shown in Figures 1 and 2.

34. Waller, A. D.: Eight Lectures on the Signs of Life, New York, 1903, E. P. Dutton & Co., p. 10; On Skin Currents. Observations on Cats, Proc. Roy. Soc., 1901, lxi, 171.

35. Hermann, L.: Handbuch der Physiologie, ii, part 1, p. 194; Borutau, D.: Arch. f. d. ges. Physiol., 1901, lxxxiv, 95.

36. Gotch, F., and Horsley, V.: On the Mammalian Nervous System, Its Functions and Their Localization Determined by an Electrical Method, Phil. Tr. Roy Soc. London, 1891, clxxxii, B, p. 332.

37. Beck, A.: Centralbl. f. Physiol., Nov. 8, 1890.

38. Alcock, N. D., and Seemann, J.: Arch. f. d. ges. Physiol. (Pflüger's), 1905, cviii.

39. Einthoven, W.: Ueber Vagusströme, Arch. f. d. ges. Physiol. (Pflüger's), 1908, cxxiv, 246.

With the two sciatic nerves of the animal in the circuit as described, I made a number of observations on making and breaking the circuit, and was satisfied that they were equipotential, that is, that there was no current passing through the galvanometer. I then trephined the skull over the right motor area, both carotids having been ligated early in the operation, and exposed the cortex. The left sciatic promptly became negative to the right, the galvanometer registering each time the circuit was closed, a deflection of 1 or 2 cm., evidently as a result of irritation of the cortex by the exposure and the application of normal salt solution to its surface. The deflections then gradually diminished in range and were finally reduced to only 1 or 2 mm. I then stimulated the motor cortex by the faradic current and determined the area for flexion of the hind limb. Having fastened that limb securely to the board so as to prevent all movement of its muscles, I made observations of the galvanometer while the cortical motor area was being faradized. The left sciatic continued to be negative to the right, the deflection increasing to about 0.5 to 1 cm. I kept up these observations for about an hour and not once did the deflection take place in the opposite direction. I then trephined the skull over the left motor cortex and, as before, stimulated the hind-limb area there while the right hemisphere was at rest. The direction of the deflection was promptly changed, indicating that the right sciatic nerve became negative to the left. While these results were merely what was to be expected, their demonstration is of value in that it suggests to us the means whereby we are able to determine with more or less precision the comparative state of activity on the two sides of the body when one side is hyperfunctionating.

Applying this method toward the solution of our problem with reference to the mechanism of the cerebellar function, I carried out the following series of experiments: I removed in one group of cats the right lobe of the cerebellum; in another group the left lobe and kept them under observation for one, two and three weeks. I did not study them galvanometrically during the first week for two reasons. First, the animals were too much depressed and frequently in a state of collapse, and it was not thought wise to submit them to a second operation requiring prolonged anesthesia. Secondly, as my object was to determine the electric potential on the two sides, a state of negativity on the side of the lesion soon after the cerebellar operation would not indicate anything, as it might be due to irritation of the adjacent structures in the cerebellum. I therefore postponed such studies until all the irritative phenomena of Luciani had passed off. Out of all the animals operated on I selected for these studies only seven, three with the right lateral lobe removed and four with the left, as I discarded all those which did not show marked unilateral ataxia and whose wounds did not heal by first intention. Subsequent necropsies have shown that in each case almost an entire lateral lobe had been removed, leaving the vermis intact. The galvanometric studies were carried out in

these animals in the same manner as in the experiment recorded above, only that in these animals no artificial stimulation was employed. The two ulnar or the two sciatic nerves were placed on the nonpolarizable electrodes which were in a circuit with the galvanometer, and observations made as to the presence or absence of a current and its direction. While the animals so studied were comparatively few, the results were so constant and so uniform for each kind of lesion that I feel justified in making certain deductions. The nerves on the side of the lesion, whether ulnar or sciatic, were invariably and persistently negative or "zincative" to those of the healthy side. The observations were kept up for two and three hours, and not once was there a deviation from this rule. Closing the circuit promptly brought about a deflection in the galvanometer, the direction of which indicated that a current was passing through it from the healthy side to the injured side. On changing, by means of a reverser, the poles of the circuit, the deflection, of course, took place in the opposite direction, always, however, indicating the same difference of potential between the two sides.

While the direction of the deflection was uniform, its extent was extremely variable, ranging from 2 cm. to 2 mm. within a very short space of time. This

apparently depends on the condition of the animal and the state of narcosis, which, it should be remembered, virtually abolishes all nervous activity. That the anesthesia was a factor in determining the extent of the deflection was noted repeatedly when a deflection of only 2 or 3 mm. during deep narcosis became gradually increased on the withdrawal of anesthesia.

Could this difference in potential be due to differ-

ences in surface temperature of the two nerves, to differences in the concentration of the salt solution, or to other accidental causes? To determine this point I cut the nerves, leaving only the peripheral portions in contact with the electrodes. The deflections of the galvanometer ceased in each case, showing that the current previously observed was not due to such causes and also that it was not peripheral in origin. In carrying out this experiment care of course should be taken that the cross-sectional surface of the nerves is not in contact with the electrodes.

The question of course arises, what is the origin of this state of negativity or functional hyperactivity on the side of the lesion? It could not have been due to irritation of the cerebellar structures adjacent to the lesion as the same condition obtained three weeks after the cerebellar operation, when all possible irritative phenomena had passed off. It could not have been due to hyperactivity of the spinal cells on the side of the lesion, as, aside from the fact that these cells do not seem to possess any automatic activity, the spinal animal responding only to external stimuli, there was no evidence in my experiments of any hyperactivity of these cells. The reflexes were equal on both sides and there was no evidence of spasticity on the side of the lesion.



Fig. 4.—Cat two weeks after the removal of right lateral lobe of the cerebellum. Note its ability to support the body by its nonparetic limbs.

It may be assumed, however, that the hyperactivity originates in the paracerebellar nuclei, especially Deiters' nucleus. These nuclei, like the vagal cells in the medulla are known to possess automatic activity, as proved by the decerebrate rigidity following transection of the mesencephalon. Thiele²⁰ in an extensive study of this rigidity has shown that it originates in these nuclei and that the cerebellum exerts an inhibitory effect on them. Stimulation of the cerebellum produces immediate relaxation of such rigidity, as demonstrated by Sherrington⁴⁰ as well as Lowenthal and Horsley.⁴¹

It may also be assumed that the hyperactivity has its origin in the midbrain or the basal ganglia and that the inhibitory effect of the cerebellum is exerted on these structures. While later investigations tend to deny these structures a motor function, there is evidence that they participate in the production of movement to some extent.

And, finally, it may be supposed, as Gowers theoretically assumed, that there is hyperactivity on the side of the cerebellar lesion which originates in the contralateral motor area of the cerebellum.

To determine this point, I carried out the following experiments: In one cat I removed a very large portion of the right hemisphere of the cerebrum but left the motor area intact. In three others I removed only the motor area. The first cat showed no paralysis on the opposite side; the three others showed it to a very marked degree. Ten to fifteen days after the cerebral operation I removed in these cats the lateral lobe of the cerebellum, contralaterally to the previous lesions (of the three last, two had the right motor area of the cerebrum and the left lateral lobe of the cerebellum removed, and one the left motor area of the cerebrum and the right lateral lobe of the cerebellum) and kept them under observation for fifteen days more. The first cat showed ataxia on the side of the cerebellar lesion, but, as before, no paralysis. The three last, on the other hand, were markedly paralyzed on that side. They showed no ataxia, but did show considerable tremor, involving head, trunk and paretic limbs. The presence of the tremor in these cases, after destruction of motor area, would indicate that this symptom is not entirely cerebral in origin, a supposition advanced, on theoretical grounds, by Gordon Holmes⁴² and Kinnier Wilson.⁴³

Galvanometrically these cats gave the following results: The first one, with the motor area intact, although with a large portion of the cerebrum removed, showed a persistent and invariable negativity on the side of the cerebellar lesion, identically the same as in those animals in which a cerebellar lesion only had been produced. The three others, on the other hand, with a lateral lobe of the cerebellum removed and the additional extirpation of the contralateral motor area

of the cerebrum, gave no galvanometric deflection whatever. I kept up my observations in these animals for nearly three hours and not once was there a deviation from this rule. Fearing that there was no deflection obtained in the last animals because of a possible fault in the circuit, I cut the sciatic nerve and applied the nonpolarizable electrodes to it in such a manner that one was in contact with the longitudinal surface and the other with the cross section. A current of injury was promptly obtained, showing that there was no such fault there.

The results obtained thus tend to show that the hyperactivity on the side of the cerebellar lesion, as shown by its being electrically negative ("zincative") to the other side, originates in the contralateral motor area of the cerebrum; that following unilateral ablation of the cerebellum the contralateral motor area of the cerebrum becomes hyperexcitable, has been found by Risien Russell⁹ and confirmed in part by Luciani.⁴ Their results were denied by Bianchi.⁴⁴ Cerebellar incoordination is, therefore, in accord with Gowers' hypothesis, only indirectly cerebellar; it is primarily a cerebral effect, an expression of its hyperactivity consequent to the removal of inhibition or control by the opposite half of the cerebellum. The hyperactivity manifests itself in all the

movements in that they are excessive, forcible, unchecked and go beyond the mark. In walking the animal throws its foot violently forward; in seizing food its paw goes beyond it, etc. Clinically, as Babinski pointed out, cerebellar patients, in performing the finger-to-nose test, are unable to stop the movement when the point aimed at is reached, and the finger goes beyond it, violently striking the jaw.

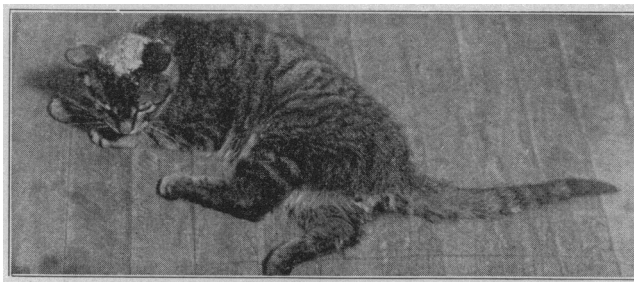


Fig. 5.—Cat shown in Figure 4, after remaining in that posture for two to three minutes. Animal falls over to the right, owing to hyperactivity of the muscles supporting the body on that side (see text).

Similarly, when tracing on a sheet of paper a line which should be stopped at a certain point, the pen goes way beyond this limit. The symptom of adiadosokinesis is therefore best explained by assuming it to be a result of the dysmetria and excess of each component movement, such as pronation and supination, a view suggested by Thomas, rather than that it is due to a delay in the excitomotor activity, as thought by Babinski, the discoverer of this phenomenon.

This conception of the cerebellar function is also in accord with certain well-established anatomic and clinical facts. The cerebellum develops embryologically from the rhomboidal lip of His, along the sensory tracts, as an accessory to them. Anatomically it is too, as Edinger, Sherrington, Gowers and others pointed out, a sensory organ. The effects of its lesions are, however, motor in character. This contradiction between its anatomy and physiology does not exist if we assume the motor phenomena to be cerebral in origin, and the function of the cerebellum to be that of control and restraint, such, to use a crude simile, as that of the vagus, which is also a sensory structure.⁴⁵

40. Sherrington, C. S.: *Proc. Roy. Soc. London*, 1896, ix, 382; *Integrative Action of the Nervous System*, N. Y., 1906.

41. Lowenthal and Horsley: *Proc. Roy. Soc. London*, 1897, lxi, 20.

42. Holmes, Gordon: *Brain*, 1904-1905, xxvii, 364.

43. Wilson, S. A. Kinnier: *The Anatomy and Physiology of the Corpus Striatum*, *Brain*, 1914, xxxvi, 478; *Progressive Lenticular Degeneration*, *Brain*, 1911, xxxiv, 465.

44. Bianchi, quoted by Sherrington: Schäfer's Text-Book, ii, 904.

45. It was held by Waller, Schiff and others that the inhibitory fibers of the vagus originate in the nucleus of the spinal accessory. This seems to be disproved, however, by later investigations. (Luciani: *Human Physiology*, i, Chapter 9.)

Another mystery cleared up by this conception of the cerebellar function is the path by which the cerebellar effects exhibit themselves on the periphery. As is well known, in view of the fact that the cerebellum has no descending tracts to the spinal cord, and also that the effects of its lesions manifest themselves homolaterally and not contralaterally, as is the case in lesions in all other motor and sensory tracts, it was found necessary to conceive an unusual and circuitous course for the transmission of its impulses. It is assumed that, originating in the deep nuclei of the cerebellum, the impulses first ascend by way of the cerebellomesencephalic tracts to the red nucleus of the opposite side and there are relayed, and finally descend by way of Monakow's bundle, or rubrospinal tract, crossing again to reach the spinal on the side whence they first started. This course, besides being different from that of all other motor and sensory structures which, as a rule, cross to the opposite side to exhibit their effect there, and, if homolateral in function, as the direct pyramidal tract or the vestibulospinal tract, go by direct route to the same side, is also untenable in the light of certain anatomic facts. The cerebellum attains its largest development in man, whereas the rubrospinal tract, by which its impulses are supposed to be carried, has been demonstrated only in animals and is in man at best only rudimentary.

According to our conception, however, each half of the cerebellum exhibits its function on the opposite hemisphere of the cerebrum, its tracts crossing to the other side like those of all other sensory tracts. Its indirect effects, in the form of regulated and well-proportioned movements, manifest themselves on its own side, as, originating in the motor cortex of that hemisphere of the cerebrum, the motor impulses, passing by way of the pyramidal tracts cross to the opposite side before reaching the "final common path" in the spinal cord.

Finally, there is a good deal of evidence that there is a structural linkage between the cerebral hemisphere of one side and the cerebellar of the other side, and that the cerebellum is subservient to the cerebrum. It has been observed clinically in cases of congenital or acquired atrophy of the cerebrum, and experimentally after destruction of the motor cortex, especially if the optic thalamus was involved in the lesion, that there was simultaneously in the congenital lesions or after some time in the acquired variety, an atrophy or degeneration of the opposite lateral lobe of the cerebellum. These cases are described under the name of crossed hemiatrophy.

With reference to the tremor it appears to be largely dependent on an interaction between the cerebellum and the midbrain structures. I am engaged in some experiments with reference to these points at present and hope to communicate the results at some time in the future.

To sum up, the cerebellum is a complex structure having no direct effect on the periphery, but acting primarily on the motor cortex, the paracerebellar nuclei, and probably also the basal ganglia and ruber. Its primary effects are those of inhibiting, controlling

and regulating the activity of these latter structures. Its ultimate effects are appropriate and rhythmic muscular action.

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ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. GREY, WEISENBERG AND WORK, AND MEYERS

DR. W. F. SCHALLER, San Francisco: Dr. Work brought out the relationship between certain symptoms and definite localization of lesions. Certainly this is progress in the right direction. It is not a question only of posterior fossa disease characterized by a syndrome, it is a question of exact anatomic localization. The only way we shall make an advance in this work is by correlating the symptomatology and pathology; by studying our specimens in macroscopic and microscopic serial sections.

Dr. Meyers does not believe that nystagmus is primarily an expression of disturbed cerebellar function. I am inclined to favor this view, and regard vestibular nystagmus as a reflex and as the motor expression of a stimulus somewhere along the vestibulo-ocular path: labyrinth, vestibular nerve, Deiters' nucleus, posterior longitudinal bundle and oculomotor nuclei. This stimulus may be due to a focal lesion, or to pressure. Dr. Grey in his conclusions stated that in posterior fossa tumor without nystagmus he was inclined

to believe that the tumor was intracerebellar; and that in cases of extracerebellar tumor he generally found nystagmus. May I ask Dr. Grey if the reason for this was not that in cases of extracerebellar tumor the intracranial pressure was increased and the nystagmus was due to disturbance of function by pressure? If there be a nystagmus following cerebellar lesions proper, I believe it is due to involvement of the vermis in relation to its efferent fibers to Deiters', i. e., cerebellovestibular tract (*Hacken Bundel, faisceau en crochet*) which forms an arm of the nystagmus reflex in that stimuli are received in this way from the muscles and joints and pass to the vermis before

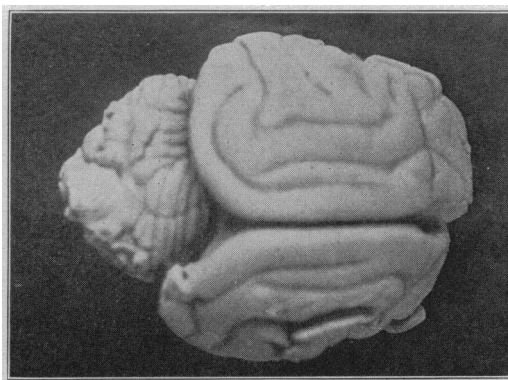


Fig. 6.—Brain of cat shown in Figures 4 and 5.

acting on Deiters' nucleus. To illustrate these views I wish briefly to mention three posterior fossa cases recently studied clinically and anatomically. In the first case there was no nystagmus, no intracranial pressure. The lesion was a vascular one, limited to the white matter of both cerebellar hemispheres and involving the dentate nucleus on one side. There was marked ataxia of the cerebellar type. The vestibulo-ocular reflex path was not involved; hence absence of nystagmus. The second case was that of a right sided cerebellopontine angle tumor in which the usual signs of intracranial pressure, vertigo, nausea, headache and choked disks were absent. The tumor had caused a considerable atrophy of the cerebellar hemisphere on the same side and also the pons, and by replacing these structures had not materially added to the contents of the posterior fossa; hence absence of intracranial pressure. Ataxia of cerebellar type was marked. A slight nystagmus more marked to the right was explained by direct involvement of the vestibulo-ocular reflex. The third case was a tumor in the fourth ventricle arising from the inferior tela. The marked nystagmus could be explained by direct pressure of the growth on Deiters' nucleus. Ataxia was absent.

DR. A. L. SKOOG, Kansas City: The subject of cerebellar tumor is always interesting, and might be broadened to include any of the lesions in the cerebellum. The subject of cerebellar function has received a great deal of attention in the last few years and many new facts have been presented. However, as indicated by Dr. Work, many of the state-

ments are really theoretical, which perhaps makes the subject still more interesting. As stated by several of the speakers, it invites the possibility of error in diagnosis. It has been stated that glioma is the most frequent type of tumor found in the cerebellum. This is in accordance with my own view. The glioma is a type of tumor which always springs from glia tissue, but there are those gliomas which are more dense than others, those that have a tendency to be better outlined. The type, however, is the infiltrating one and at times they are rather difficult to differentiate from some simple degenerating conditions of the central nervous tissue. I have always been interested to know whether there is any difference in symptomatology, even though the tumor be located in the same place, between the soft and infiltrating and the more dense one. That might be a reason for the difference in symptomatology which was brought out in the discussion.

DR. G. W. ROBINSON, Kansas City: Dr. Work speaks of the diagnosis of tumors in the posterior cranial fossa. I should like to ask Dr. Work to speak on the differentiation between these and those situated more anteriorly and giving cerebellar symptoms. I recall two or three patients with lesions (tumor or trauma) of the frontal lobe, chiefly of the middle frontal convolutions, the apraxic center, giving a contralateral cerebellar syndrome. Another condition I have observed recently has been a patient with chronic progressive cerebellar tremor, without ataxia or nystagmus. I should like some of the men who read these excellent papers to tell us, if possible, the location of the lesion giving this particular symptom. I have recently observed two patients who had shown no nystagmus. At necropsy one had a degeneration of the right cerebellar hemisphere resulting from an encephalitis (associated with typhoid fever). The other had a large cyst involving the right cerebellar hemisphere.

DR. JOHN S. TURNER, Dallas, Texas: These cases of cerebellar disturbance are very interesting to all of us and I rise to report a case and to present a question and to ask some of the gentlemen in closing to touch on it. It is in reference to a case recently occurring in our part of the country which became almost a medicolegal case, but by having the records properly presented a damage suit was prevented. The case came under my observation with all the typical symptoms of cerebellar tumor—nystagmus and other eye symptoms. The patient was a girl of 18, being able to go about, but not seeing very well, and having headaches, vomiting, titubation, disturbed coordination and considerable emaciation running over a long period of time. After observing the symptoms and making a tentative diagnosis, I sent her to a physician to look over the eye conditions. He confirmed my diagnosis in so far as the eyes were concerned, using some atropin to further dilate the pupil. Immediately after the dilatation the patient became blind and has never seen since that moment. Her headaches ceased, she gained in weight and was physically much better, but was totally blind. A damage suit was threatened against the physician for making the patient blind, but the records showing that she was partially so, and the opinion that she would ultimately become blind anyway prevented the suit.

DR. CECIL E. REYNOLDS, Los Angeles: Dr. Work laid stress on the paralysis of the sixth nerve in cerebellar lesions, and I think it is very important. I remember a case of Dr. Charles A. Ballance of London at the Great Ormond Street Hospital in 1905, in which there was weakness of the sixth nerve on the left side and a bulging of the occipital fossa on the right side. The optic neuritis was more advanced on the left side. Mr. Ballance operated on the side which bulged, but the tumor was eventually discovered on the same side as the sixth nerve paresis. In my experience the signs of intracranial pressure have been more intense in cerebellar lesions and more early than in cerebral lesions, and this fact in a measure militates against the value of sixth nerve paralysis as a localizing sign in cerebellar disease, because in general increase of intracranial pressure the sixth nerve is often first affected by reason of that general pressure.

I would like to ask some of the gentlemen who have spoken as to their experience of the value of skew-deviation as a localizing sign. Dr. Robinson mentioned a confusion of frontal lobe lesions with cerebellar lesions. I have seen several cases in which a frontal lobe lesion was mistaken for a cerebellar lesion, and it appears to me that in cerebellar lesions the head is more twisted, with the chin pointing to the shoulder of the affected side, whereas in frontal lobe lesions the eyes deviate to one side or the other without much tilting of the head. Also I have noticed that in addition to hebetude out of proportion to the pressure, there is usually a slow and deliberate Babinski's sign in frontal lobe lesions, more sluggish than that usually seen in lesions of the pyramidal tract.

DR. GEORGE A. MOLEEN, Denver: Just one remark on what Dr. Work said with reference to the extension of tumors from the thalamus downward. Is it not the rule to find unilateral paralysis of the third nerve prior to the involvement of conjugate deviation, the center of which lies pretty close to the sixth center in the pons? From this we presume there is a communicating fiber to the third, and in these extending tumors it would seem fair to expect a unilateral lesion of the third before the affection or disturbance of conjugate deviation would occur.

DR. PHILIP WORK, Pueblo, Colo.: We found in working up our pathologic material on this subject that cases which we thought definitely cases of tumor of one or other peduncle proved to be cases in which the involvement had gone past the peduncle, and the diagnosis and localization of symptoms has been hindered by the fact that these cases are so far advanced before they come to section. I personally believe that the matter of function of the peduncles—and coincident with it the establishment of a definite symptomatology—can only be hoped for when we have seen a sufficient number of early cases come to section. This is predicated on a series of necropsies from coincident cases in which the brain lesion was discovered incidentally in routine examination. The differential diagnosis of cerebellar and frontal tumor has been mentioned by one gentleman, but is too big a subject, I feel, to take up here. I have in mind several cases in our own series in which that identical question came up. In two cases in which the clinical diagnosis was made of cerebellar trouble, section showed frontal trouble, with definite cerebellar symptoms. These cases are being worked up at the present time, and we hope to be able to report something of value when we get them correlated. The loss of sight spoken of in the medicolegal case would seem to me to be due to the advanced state of the optic condition. The fact that the other symptoms abated more or less I do not attempt to reconcile.

DR. ERNEST GREY, Boston: Dr. Schaller has referred to increased intracranial pressure as an important factor in the production of nystagmus. This is the view of Bárány and others, and in our earlier work we inclined to the same belief. Recent observations, however, have led us to seek another explanation. We found nystagmus present in a group of patients with subtentorial new growths, some of whom had greatly increased intracranial pressure, while others had practically normal pressure. We have had a similar series of patients with the same variations in the degree of intracranial tension; none showed any nystagmus.

DR. I. LEON MEYERS, Chicago: Something has been mentioned in regard to nystagmus. I can simply add what I said in my paper—in my experience true cerebellar lesions do not give rise to nystagmus unless there is additional involvement of the vestibular complex. Skew-deviation has been observed in a few experiments on dogs during the time of anesthesia, but was lost after the animal awakened. Frederick Batten in a paper published in *Brain* in 1903 determined that the position of the head is not diagnostic of cerebellar lesions. It appears that the position of the head is due to the loss of knowledge of special relationship and is vestibular in origin. In my judgment the cerebellum is purely an organ which exerts an influence on other nerve cells in the brain structures—not on the periphery; the control it exerts is chiefly in the motor sphere.