

## LENTICULAR ZONE AND ANARTHRIA\*

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The classical ideas concerning the question of aphasia have recently become a subject for reconsideration through the efforts of Pierre Marie.<sup>1</sup> The distinguished French neurologist, after a careful and conscientious study covering a period of several years, has endeavored to replace the old views on aphasia by a new and more simple conception. His contention is that the division of aphasia into a motor and sensory is no more tenable; that there is only one aphasia, namely: sensory aphasia, to which anarthria is sometimes added. Marie, therefore, does not recognize the motor aphasia of Broca with agraphia, but considers only total aphasias.

Since this somewhat startling announcement of Marie, made in 1906, a large number of anatomico-clinical observations on aphasia have been reported by competent observers, some corroborating and others denying the above view. In spite of the large material already accumulated the views differ widely. Indeed, so far it seems that they are even irreconcilable. One class of writers, with the masterly Déjerine<sup>2</sup> at its head, refuses absolutely to accept Marie's ideas and rejects them *in toto*. The other category of very authoritative men adhere to Marie's new conception without the least criticism. The clinico-anatomical case I am reporting demonstrates clearly, I believe, that, while certain features of Marie's revised view on aphasia are not at all in conformity with pathological findings, there are nevertheless some phases which can be explained, if not totally, at least to a large degree, on the basis of his so-called lenticular zone. The anatomical and clinical conditions of the case are particularly conspicuous with regard to the most important point of Marie's contention, namely, with the question of anarthria.

### CASE REPORT

The case is as follows:

*Patient.*—O. A., a colored man, aged 38, laborer, was admitted to the Douglass Memorial Hospital, Aug. 27, 1909, with a complete right hemiplegia. The history

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\* Read at the meeting of the American Neurological Association, held in Washington, May, 2 to 4, 1910.

1. Marie, P.: *Semaine méd.*, 1906, xxvi, 241.

2. Déjerine, J.: *Presse Méd.*, 1906, xiv, 437, 453; *l'Encéphale*, 1907, ii, 400.

shows that at the age of 23 he had had a chancre. Except smallpox, he never had any other disease and always was in good health. About one year before admission he began to feel pain in his right arm. There was a continuous aching and occasionally sharp pain would appear. Four weeks before his admission the pain became more intense. While sitting one day at the table, he made an attempt to rise, but fell, having lost power on the right side. There was no loss of consciousness.

*Examination.*—On admission to the hospital, the following condition was found: The right arm was totally paralyzed; some movements were noticeable in the right leg; the mouth was drawn to the left. While talking, saliva would run out of the right corner of the mouth. The patient had therefore a total right hemiplegia. There was considerable spasticity in the arm and leg. The knee-jerks were increased on both sides, more on the right than on the left. Ankle-clonus was present only on the right. The toe phenomenon by Babinski's method was absent on the right, present on the left; by my method present only on the left side, when the calf-muscles of the right were pressed on. There was a diminution of the sense of touch and pain on the right side. The pupils were equal and normal and reacted to light and accommodation. The eyegrounds were normal. There were no palsies of the ocular muscles. The heart showed a slight dilatation of the left ventricle; the sounds at the apex were somewhat rough. The second aortic sound was markedly accentuated. The speech showed no disturbance of articulation, the words, syllables and letters were distinctly pronounced and without the least hesitation. The speech was impaired at the onset of the apoplectic seizure, but the patient recovered it in twenty-four hours. This information was obtained from a relative with whom the patient lived.

Further investigation into the mental condition and various forms of speech gave the following result. The first disturbance noticed was the recalling of names of certain (not all) objects. The following examples will demonstrate the disorder:

"What is the name of this place" (hospital) ?

In reply, the patient smiles, makes an effort to answer, but cannot find the name.

"Is it a stable?"

"No."

"Is it a kitchen?"

"No."

"Is it an apartment house?"

"No."

"Is it a hospital?"

"Yes."

"What is this man's business" (referring to the resident physician) ?

"I can't say."

"Is he a fireman?"

"No."

"Is he a waiter?"

"No."

"Is he a tailor?"

"No."

"Is he a doctor?"

"Yes."

"What is your work?"

"I can't tell."

"Do you do writing?"

"No."

"Do you cook?"

"No."

"Are you a laborer?"

"Yes."

"In what state is Philadelphia?"

No answer.

"Is it in Maryland?"

"No."

"Is it in New York?"

"No."

"Is it in Virginia?"

"No."

"Is it in New Jersey?"

"No."

"Is it in Pennsylvania?"

"Yes."

"What is this" (watch is shown) ?

No answer.

"Is it a key?"

"No."

"Is it a lock?"

"No."

"Is it a ball?"

"No."

"Is it a watch?"

"Yes."

Several other objects were then shown, such as key, knife, penholder, etc. The patient named some of them promptly, some with delay.

The patient presented a certain degree of word-blindness. The *Saturday Evening Post* and *Illustrated Weekly Magazine* were shown him and he was asked to read the heading. He read: "Sanitarium Evening Postal and Illustrated Weekly Manager." Further test for reading showed that he missed words, syllables, letters. A partial alexia was therefore present. The test for writing could not be performed, as the right hand was completely paralyzed, the patient being right-handed. This mental condition showed at times some lapses.

"How old are you?"

"I am 24." (He is 38.)

"How long have you been married?"

"Sixteen years."

"How old were you when you got married?"

"Sixteen years."

"Are you happy?"

"Well no, I am sick. I think I have the old folks, I don't owe anybody."

The patient also repeated continually that he heard the Lord's voice and saw angels and spirits.

His memory was also somewhat deficient; he could not tell from one meal to another what was given him to eat. He also made mistakes in the days of the week. In spite of these deficiencies he could give prompt and clear answers on the subject of his illness, on the functions of his sphincters, on his comfort and discomfort; he was able to converse with the nurse and resident physician on the weather and on his family.

To sum up, he presented at the time of his admission to the hospital and for two subsequent months, complete right hemiplegia, alexia, verbal amnesia. The latter two phenomena were not complete. But what is particularly important is the total absence of motor aphasia and of dysarthria.

*Course of Disease.*—For two months the patient's condition remained unaltered. One morning he was found unconscious and agitated with convulsive movement in his right arm and leg and frothing at the mouth. The attack lasted a minute, but for two subsequent hours the arm and leg remained in a state of extreme rigidity. Moreover, the least touch of the leg or arm would produce a convulsive movement. From the moment he lost consciousness, and during the following twenty-four hours of his life, he presented a conjugate deviation of the head and eyes to the right. An attempt to turn his head to the left would not correct the direction of the eyes. During the first few hours he could not voluntarily turn the head to the left, but in the last few hours he was able to make attempts to do it, but only attempts, as he was unable to hold it in the right direction longer than for a fraction of a minute. The same can be said about the eyes. In addition to this phenomenon, the patient was unable to protrude his tongue, move his lips or utter a word. Soon he presented difficulty of swallowing and on the twenty-fifth hour he expired.

*Autopsy.*—This was performed by Drs. Bailey and Diu Guid. The brain and cord were removed. The brain presents a slightly thickened dura and along the superior longitudinal fissure on both sides subdural osseous plaques are seen; the latter are strongly adherent to the dura and pia, especially on the left side. The frontal lobes are small and a longitudinal groove is observed on the right frontal lobe at the level of transition of the frontal to the orbital lobe. The entire posterior half of the left hemisphere is congested, and the superior third of the left Rolandic area is softer than that of the right hemisphere. There is nothing special at the base. A high transverse anteroposterior section of the brain shows a more congested state of the left hemisphere than of the right. The anterior two-thirds of the left internal capsule, as is seen in Figure 1, and the antero-external portion of the left optic thalamus show marked softening and destruction.

A second lower transverse anteroposterior section shows the following condition (Fig. 2): The entire left internal capsule is totally softened. The anterior portion of it presents a shriveled, folded mass. The lenticular nucleus, the external capsule and the head and tail of the caudate nucleus are completely destroyed. The very posterior portion of the capsule and a part of the inferior longitudinal bundle surrounding the posterior cornu of the lateral ventricle are also involved. The left optic thalamus is found very much paler than the right, and the part of it adjacent to the capsule is also softened. A particularly deep destruction is seen in the portion of the softened area which is in contact with the subcortical tissue of the insula, where a deep depression is seen. The softening also extends at a certain distance into the supramarginal gyrus, but the white matter of the angular gyrus is apparently intact.

#### DISCUSSION OF CASE

If we consider here the boundaries of the so-called lenticular zone as designated by Marie, namely, a square area limited anteriorly by the white substance of the third frontal convolution, posteriorly by Wernicke's area, externally by the insula, internally by the wall of the third ventricle, and if we consequently consider the fact that the lenticular zone com-

prises the external capsule, the lenticular and caudate nuclei, the anterior and posterior segments of the internal capsule, also the optic thalamus, we must conclude that in the present case the entire lenticular zone was destroyed with the exception of the inner two-thirds of the thalamus.

According to Marie the lenticular nucleus plays an enormous rôle in phonation and in coordination of movements indispensable for articulated speech. Indeed, he says that the lenticulostriated ganglia either by themselves or through their afferent and efferent pathways represent in the mechanism of language a body far more important from a motor standpoint than the cortical center of the old Broca's view. If, however, we refer to the anatomy of the lenticular and caudate nuclei and particularly to secondary degenerations, we know that no fibers from these nuclei enter into the composition of the cerebral peduncles, and if disturbances of speech occur in lesions of the lenticular nuclei, such lesions are always accompanied by lesions of the adjacent internal capsule. It is a well-known fact that an involvement of the knee and the adjacent portion of the posterior segment of the internal capsule gives disturbances of speech of a paralytic nature, namely, dysarthria. Déjerine has shown that through this portion of the internal capsule pass fibers coming from the operculum, which is the motor center for the lips, tongue and larynx. A unilateral lesion of this center produces dysarthria or anarthria, which are well known to be paralytic disturbances of phonation and articulation of words. In the classical motor aphasia, on the contrary, there is no paralysis of the organs involved in the mechanism of phonation and articulation. In the latter, not only the spoken language is altered, but also the inner language; such a patient has no spontaneity in bringing before him auditory images; the few words that he pronounces he utters correctly, although he may accentuate syllables or letters. The dysarthric, or anarthric, on the contrary, pronounces very deficiently, but his inner language is perfectly preserved.

Applying this information to the present case, we see that Marie's contention as to the function of the lenticulostriated body cannot be confirmed entirely by the pathological findings of the present case. Indeed, the destruction of the lenticular and caudate nuclei was complete, and yet the patient did not present the least indication of dysarthria. His speech, as far as the articulation is concerned, was clear and distinct. The patient did, however, present a partial word-blindness and partial verbal amnesia, all symptoms of sensory aphasia. If we assume that, in describing his area of speech, Marie meant that a lesion in any spot of the lenticular zone will produce symptoms of aphasia (sensory or motor),



Fig 1.—Upper section showing partial destruction of the lenticular zone (see text) viz.: two thirds of left internal capsule, antero-external portion of thalamus and tissue surrounding the internal capsule.

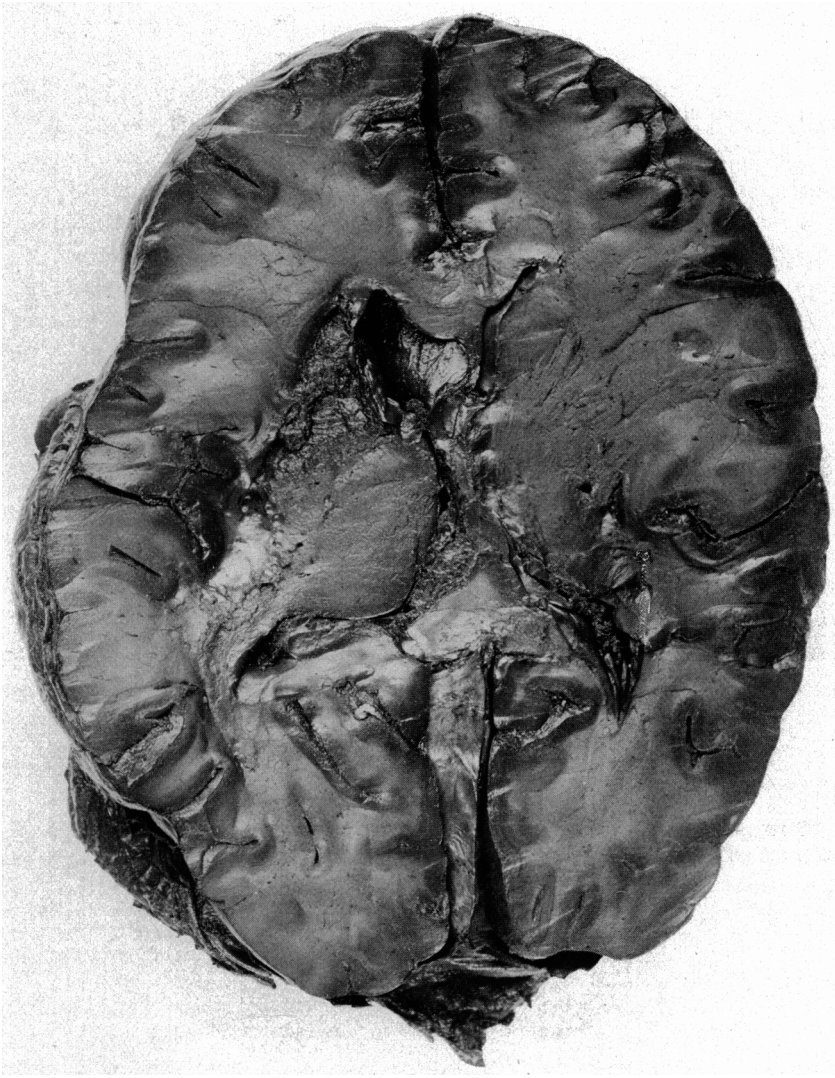


Fig. 2.—Lower section showing complete destruction of the lenticular zone (see text) viz.: entire left internal capsule, lenticular nucleus, external capsule, head and tail of caudate nucleus, part of inferior longitudinal bundle.

the present case justifies his view to some extent. The few symptoms of sensory aphasia observed in this case correspond in reality to Marie's contention, but are at variance with the old conception concerning Wernicke's zone. Indeed the angular gyrus, the supramarginal gyrus, the posterior portions of the first two temporal convolutions, the insula and the frontal convolution are all found here intact, macroscopically and microscopically. In support of Marie's conception of sensory aphasia can be mentioned also the condition of the inferior longitudinal bundle, which in my case was partly destroyed. As is well known, a lesion of this fasciculus plays, according to Marie, an important rôle in the causation of aphasia.

On the other hand, when a complete softening of the entire lenticular zone fails to produce symptoms of anarthria, on which Marie lays so much stress, and fails to present a complete picture of sensory aphasia, his doctrine does not possess so solid a basis, as one might fancy in listening to his arguments. It would, therefore, appear to be weakened by the pathological findings of the present case.

#### CONCLUSION

The conclusion to which this observation leads is that while the so-called lenticular zone of Marie may play a certain rôle in sensory aphasia, its rôle is not considerable. As to its being a center for anarthria, the present case proves that its destruction does not interfere with phonation and articulation of spoken words. Consequently, if Marie's conception of aphasia may be applicable to a certain series of cases, as he has shown, it does not hold its ground in every case in which the sensory or motor speech may become involved.

NOTE:—Since this work has been prepared, I came across Von Gehuchten's observation reported before the Académie Royale de Médecine de Belgique Jan. 29, 1910. The latter had a clinico-anatomical case almost identical with mine. There was also a vast focus of softening in the lenticular zone of the left hemisphere. It destroyed the entire external segment of the lenticular nucleus with the corresponding portion of the external capsule, the middle portion of the posterior segment of the internal capsule; it also destroyed the nucleus caudatus. The patient presented during life a right hemiplegia, but not a trace of anarthria, contrary to what might have been expected according to the views of Pierre Marie.

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