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Remarks on the Seat of Spoken Language, Followed by a Case of Aphasia (1861)

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This work and the subsequent case which I am here presenting support Mr. [Jean B.] Bouillaud's ideas on the seat of the faculty of language. This question, which is both a physiological and a pathological one, merits more attention than most physicians have been willing to accord it. It is a very delicate matter, very obscure, and somewhat complicated; in fact, so much so that I feel I should make some introductory remarks before going into the fact which I have observed.

It is known that phrenologists place the ability to speak in the area slightly anterior to the brain, in one of the convolutions resting on the orbital arch. This opinion which had been accepted without sufficient proof, and which rested on a very imperfect analysis of the phenomenon of language, would doubtlessly have disappeared with the rest of the system, had Mr. Bouillaud not rescued it from the shipwreck, by making large modifications and by surrounding it with a halo of proof borrowed mainly from pathology. Without considering language a simple phenomenon depending on a single cerebral organ and without attempting to circumscribe the organ within a space of a few millimeters as Gall had done, this professor [Bouillaud] was led to admit by numerous clinical facts, followed by autopsies, that certain lesions in the hemispheres abolish language without destroying intelligence and that these lesions always occur in the frontal lobes of the brain. He concluded that somewhere in those lobes, there are one or more convolutions which dominate some essential element of speech and then, without any further specification, he placed in the frontal lobes the faculty of spoken language which should not be confused with the general faculty of language as such. There are indeed several kinds of language.

Kann, J. (1950) A translation of Broca's original article on the location of the speech center. *Journal of Speech and Hearing Disorders*, 15, pp.16–20. (With permission).

Any system of signals permitting the expression of an idea in a more or less intelligible, complete or rapid manner, is a language in the most general sense of the word: speech, mimicry, shorthand, figurative writing, phonetic writing, etc., are all respectively types of languages.

There is a general faculty of language which presides over all modes of expression of thought, and which may be defined as the faculty to establish a constant relationship between an idea and a signal, whether this signal be a sound, a gesture, a figure, or any type of tracing. Furthermore, each type of language necessitates an organ of 'emission' and an organ of 'reception.' The organs of reception are the ear, eye, or even sense of touch. Organs of emission are set into play by voluntary muscles, as those of the larynx, tongue, face or upper extremities, etc. All regular language supposes then an integration of: (1) A certain number of muscles, their respective motor nerves and the central organ from which these nerves arise. (2) A certain external sense organ, the sensory nerve leaving it and the area of the central nervous system in which the nerve ends. (3) Finally that area of the brain controlling a general faculty of language as we have defined it.

[After this definition Mr. Broca continues his introduction with general remarks which are of minor interest here. Following is the actual report to the Academie of a case of aphasia or, as Broca calls it, aphemia. Case: Aphemia for 21 years, produced by chronic and progressive softening of the second and third convolutions of the superior part of the left frontal lobe.]

On April 11, 1861, the general infirmary of Bicetre, Surgical Service admitted a man, 51, named Leborgne, suffering from a gangrenous condition of the lower right extremity, extending from the toe to the groin. To all the questions which I put to him concerning the origin of his illness, he answered only by the monosyllabic 'tan' repeated twice in succession and accompanied by a gesture of the left

hand. I looked for more information on this man who had lived at Bicetre for 21 years. We interrogated his superiors, the comrades of his division and those relatives who came to see him, and here is the result of our inquiry: Since early youth, he had had attacks of epilepsy, but he was able to become a lastmaker and remained thus until he was 30 years of age. At that time, he lost his ability to speak and was therefore admitted to the hospital at Bicetre. It was not known whether the loss of speech came about slowly or suddenly or if any other symptom accompanied the onset of the disorder. When he arrived in Bicetre, he had not spoken for two or three months. He was perfectly well oriented and intelligent and did not differ from a healthy man expect in the ability to speak. He walked about the hospital where he was known as 'Tan.' He understood everything said to him; even his hearing was excellent; but no matter what question was addressed to him, he always answered: 'tan, tan' accompanied by various gestures designed to make himself understood. When his interrogators did not understand his mimicry, he became easily angered and added to his vocabulary an oath, the same mentioned before when discussing the case of Mr. [Ernest] Auburtin. 'Tan' was regarded as egotistical, vindictive, mean, and his comrades, who hated him, accused him of being a thief. These traits may have been due to his cerebral lesion, anyhow, they were not pronounced enough to be called pathological; and although the patient was at Bicetre, we never thought to put the patient into the psychiatric division. He was regarded as a man who was perfectly responsible for his acts.

It is clear that the patient was suffering from a progressive cerebral lesion, which was very small at the beginning of the illness and did not affect organs of movement or sensitivity. In the first 10 years it had spread to one or more organs of movement but had left the organs of sensitivity intact; later general sensitivity had lessened at the same time as his vision diminished, especially in his left eye. The paralysis and loss of sensitivity being on the right, the lesion had to be in the left hemisphere.

It was now a question of determining accurately the original lesion and although the last session of the Société d'Anthropologie had thrown some doubt on the theories of Mr. Bouillaud, I wanted to argue the question as if his theories had been verified, for that is the best way to prove a point. [Mr. Auburtin, who had announced that he would change his theories if he saw one case of aphemia with involvement of the anterior lobes was called in and confirmed the diagnosis.]

In order to complete the diagnosis, I argued that the corpus striatum is the closest motor organ to the anterior lobes. Doubtlessly the lesion had spread to this area and caused the hemiplegia. The probable diagnosis was then: primary lesion of the left anterior lobe, spread to the corpus striatum on the ipsolateral side. All facts pointed toward a slowly progressing softening and in the absence of pressure, the diagnosis of intracranial tumor was excluded.

The patient died on the 17th of April at 11 o'clock in the morning. Within 24 hours an autopsy was performed. The temperature outside was not high and the corpse had not putrified. The brain was shown a few hours later to the Société d'Anthropologie and then put into alcohol. The organ had undergone such change that it was difficult to preserve it and it took several months and several changes of liquid until it began to become firmer. Today, in excellent condition, it is exhibit 55 in the 'Dupuytren Museum.'

[Some neurological findings are discussed.]

The anatomical inspection shows that the lesion was still progressing when the patient died. This lesion had progressed very slowly, since it took 21 years to destroy a small cerebral area. It may be assumed then, that at the beginning there was a long period during which it did not surpass the limits of the primary locus of infection. Also, we have seen that this locus is situated in the frontal lobe, probably in the third convolution. This led us to believe that from the point of view of pathology there were two periods: one in which a single convolution was affected (third frontal); the other in which the disease progressed to other areas.

If we examine now the succession of symptoms we also find two periods: a first period which lasted 10 years during which language was lost and all other functions of the brain were intact; and a second phase of 11 years during which at first partial, then complete, paralysis successively invaded the upper and lower extremity of the right side. It is impossible to overlook the fact that a relation exists between the two anatomical periods and the two symptomatic periods. The second clinical period corresponds to the second anatomical period, that is, that time when the softening had gained the insular lobe and corpus striatum. The first period of 10 years then, characterized clinically only by aphasia corresponds to the period when the lesions were still limited to the frontal lobes. Until now, in drawing a parallel between lesion and symptom, I have spoken neither of intellectual disturbances nor of their anatomical causes. We have seen that the intelligence of our patient, perfectly preserved for a long time, had notably declined. We find, in the autopsy, changes which were sufficient to explain this state. Three out of four frontal convolutions had been affected by the lesion. Almost the entire frontal lobe had more or less softened. Finally the entire mass of convolutions of the two hemispheres had atrophied and had become much softer than in their normal state. It is difficult to understand that the patient was able to preserve any intelligence at all, and it seems improbable that one could live for a long time with such a brain. I, for one, feel that the general softening of the left frontal lobe, the general atrophy of the two hemispheres, and the chronic general meningitis were lesions of standing. I feel that these short lesions had started a long time after the softening of the

corpus striatum, and that one could subdivide the second period into two secondary periods.

	Lesion	Symptom
Period 1 (10 years)	Softening of frontal convolution	Aphasia
Period 2	(a) Left corpus	(a) Paralysis striatum affected
	(b) Softening of entire frontal lobe—general cerebral atrophy	(b) Loss of intelligence

These facts which are questions of doctrine cannot be exposed with enough detail nor discussed carefully enough. I need to excuse myself for the dryness of the description and the length of the discussion. I have only a few words left to sum up the consequences of my observation.

(1) Aphemia, that is, loss of speech was the consequence of a lesion in one of the anterior lobes of the brain.
(2) Our observation confirms the opinion of Mr. Bouillaud who had assigned to these lobes the seat of spoken language.
(3) The observations, at least those that are accompanied by a clear and concise anatomical description are not sufficient to consider this localization of a particular faculty in a particular lobe as having been definitely demonstrated, but it may be considered as extremely probable.
(4) It is a much more doubtful question whether the faculty of spoken language depends on the entire anterior lobe or more specifically on one of its convolutions, in other words the question is whether the cerebral faculties are arranged by faculty and by convolution or only by groups of faculties and by groups of convolutions. Further observations should be made to solve this question. In order to do that, one must indicate exactly the name and number of the convolution affected, and, if the lesion is very extensive, to find as soon as possible by anatomical examination the point or rather the convolution where the illness seems to have started.
(5) In our patient the primary seat of the lesion was in the second or third frontal convolution, more probably in the latter. It is possible then, that the faculty of spoken language is located in one or the other of these convolutions. We cannot definitely establish which, since the localization by convolution does not rest on a certain basis as yet.
(6) In any case, it is sufficient to compare our observation with those which preceded it to discard the idea that the faculty of spoken language resides in a fixed point and may be located in any bump on the head. The lesions of aphasia have most often been found in the most anterior part of the frontal lobe, not far from the eyebrow and above the orbital arch; in my patient they were slightly posterior and closer to the coronal suture than to the rim of the brow. This difference of locus is incompatible with a system of phrenology. It would be perfectly reconcilable with a system of localization by convolution since each of the three large convolutions of the upper part of the frontal lobe cuts across all the regions where the lesions of aphasia have been found to date.