

**Case with a lesion involving Broca's convolution without Broca's aphasia /
by J. Batty Tuke, M.D. and John Fraser, M.B.**

Contributors

Tuke, J. Batty, Sir 1835-1913.
Fraser, John, M.B.
Medico-Chirurgical Society of Edinburgh.
University of Glasgow. Library

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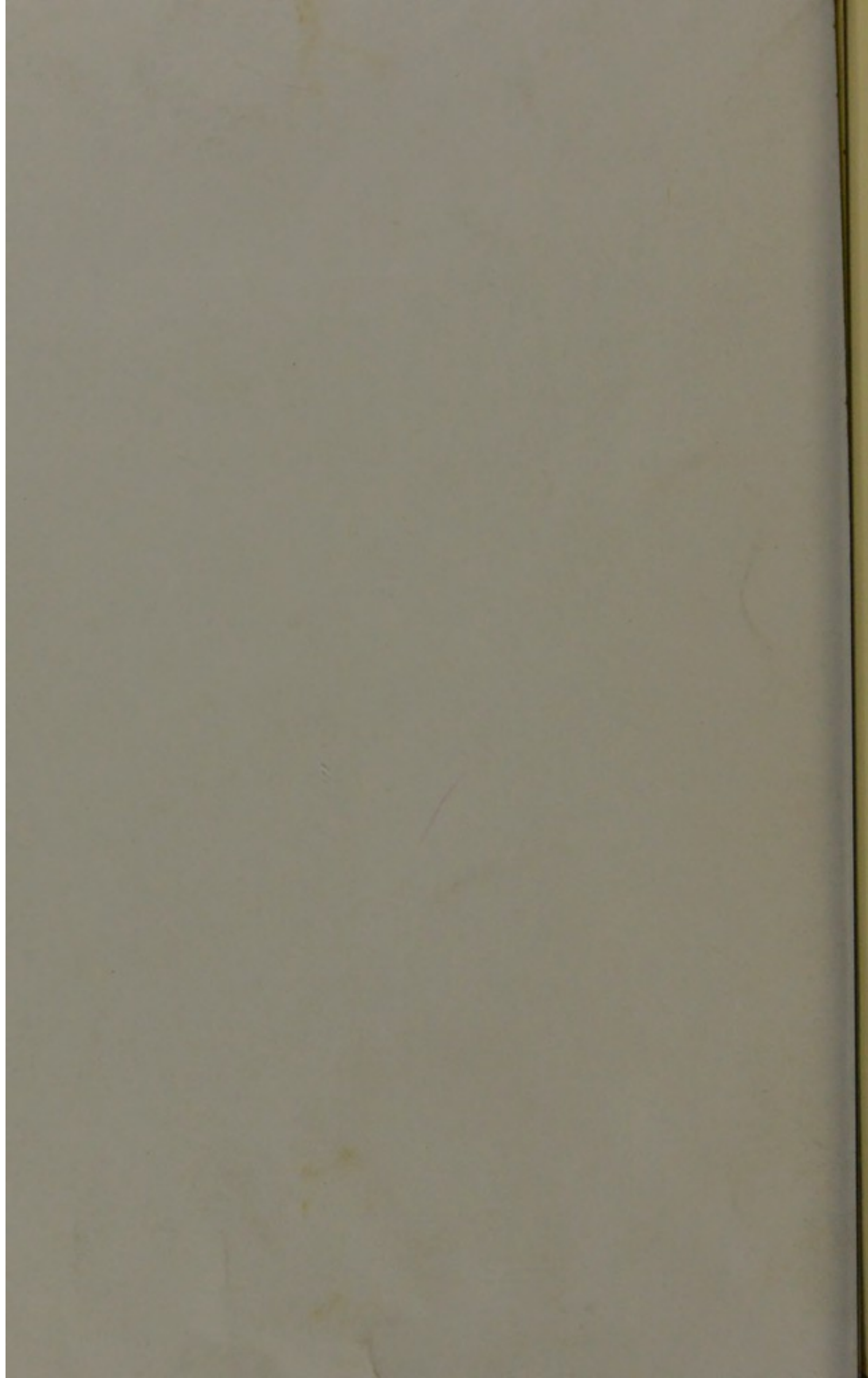
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CASE WITH A LESION

INVOLVING

BROCA'S CONVOLUTION WITHOUT BROCA'S APHASIA.

BY

J. BATTY TUKE, M.D., F.R.C.P.

AND

JOHN FRASER, M.B., C.M.

*(Read at a Meeting of the Medico-Chirurgical Society of Edinburgh, February 7th,
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CASE WITH A LESION INVOLVING BROCA'S CONVOLUTION WITHOUT BROCA'S APHASIA.

The chief object in bringing this case before you is its important bearing upon the present pathology of aphasia. It is one in which the posterior half of the third left frontal convolution was completely destroyed, both as regards the grey and white matter, and the only defect in language was partial verbal amnesia. The other points of interest are the laying bare of the extra-ventricular nucleus of the corpus striatum, without its being morbidly affected, and the absence of right hemiplegia.

M. R., æt. 54, single. Admitted to Fife and Kinross Asylum on December 14th, 1868.

The history of this case is that of an apoplectic seizure eleven years previous to the above date. While working in the field at the time stated, she suddenly fell down and became unconscious. Her friends affirm that she was insensible "for some weeks." No amount of cross-examination of her sister, with whom she was residing at this time, could elicit any evidence of paralysis; there was no distortion of the face, no lameness or want of power in either hand, but complete speechlessness existed for some time. From this, however, she must have totally recovered, for, previous to her admission, she seems to have been rather talkative. She is reported to have been weak-minded since this attack.

This patient had an hereditary predisposition to insanity. Her cousin was insane and an inmate of the Dundee Asylum.

According to the schedules, M. R. became insane only four weeks previous to her admission, December 14th, 1868. The medical certificates stated that she was the subject of various delusions and hallucinations, such as the belief that strong men were trying to kill her, that she heard guns and cannons constantly going off on the roof of the house, which prevented her sleeping. Consequent upon these were acts which re-

quired her detention in an asylum, such as great restlessness at night and constant attempts to get out of the window. She was often found on the roof of her cottage in a state of nudity.

Soon after her admission these delusions and hallucinations apparently left her, for she never spoke of them, and became a quiet and industrious patient. She, however, became possessed of a peculiar idea, viz., that Thursday was Sunday, and on that day she would not work, whereas on the Sunday she would readily have done so if allowed.

During the whole period of her residence two peculiarities in her speech were observed—a thickness of articulation resembling that of general paralysis, and a hesitancy when about to name anything, the latter increasing very much some months previous to her death.

The thickness seemed apparently due to slight immobility of the upper lip when speaking, but there was no paralysis when the lip was voluntarily compressed against its fellow. The inaction of the upper lip was observed by all.

The hesitancy was most marked when she came to a noun, the hiatus varying in duration according to the uncommonness of the word. Latterly, she could not recall even the commonest terms, and periphrases or gestures were used to indicate her meaning. She was always relieved and pleased if the words were given her, when she invariably repeated them. For example, she would say, "Give me a glass of—." If asked if it was "water," she said, "No." "Wine?" "No." "Whisky?" "Yes, whisky." *Never did she hesitate to articulate the word when she heard it.*

Her friends said she could read very well before the apoplectic attack, but not afterwards. This is incorrect, for she was constantly reading in her spare time, and that this was not a pretence at reading could be gathered from her comments on the book.

She was never observed to write, yet her friends state she could do so.

Her memory generally was slightly impaired, more in regard to recent than to remote events.

She became paraplegic two months before death. There had been a peculiar stoop in her walk for many years, and a constant pain, increased on pressure, at the third and fourth dorsal spines. She died 26th Dec., 1871.

The following is the result of the autopsy:—

Head—calvarium very dense and thick, tables thickened,

diploe thin, membranes healthy, except some old adhesions on the left side, at the anterior part.

Brain appeared to be slightly shrunken, but the amount of serum which escaped ($1\frac{1}{2}$ ounces) was not sufficient to indicate any great general atrophy.

On stripping off the dura mater on the left side, some slight adhesions were found between the layers of the arachnoid. These were easily detached, and exposed an excavation of the brain substance at the postero-infero-external part of the left frontal lobe. Its outline was irregular, its cavity filled with serum, and narrow white bands sprang from its sides. The serum was opalescent, but otherwise normal, and it was held in by the visceral arachnoid. On emptying the cavity, its dimensions were found to be as follows:—In its long axis, from before backwards, parallel to the fissure of Sylvius, two and a quarter inches obliquely, vertically one and three-sixteenths of an inch; in its deepest part it was three-quarters of an inch, but generally only half an inch. This lesion had destroyed posteriorly the inferior fourth of the ascending parietal convolution, leaving a small posterior portion of the knuckle, in which this gyrus ends, the inferior third of the ascending frontal, the inferior margin of the second frontal, and the *posterior half of the third frontal convolution** (Turner's inferior frontal), (Broca's). At the inferior margin there was a narrow ridge of slight eminence, which might have been the remains of the inferior border of the third. With these exceptions, the destruction of the posterior half of this convolution was complete, both as regards its grey and white matter. Its inferior boundary was the superior marginal convolution. The bottom seemed to be an anatomical limitation, as it was smooth, rounded, and presented no evidence of morbid action. Incision proved it to be the extra-ventricular nucleus of the corpus striatum. The edges of this lesion implicating the convolutions were ragged, which was suggestive of erosion, but there was no indication, by induration, softening, or thickening of the membranes, of inflammatory action.

Microscopical examination of the grey matter taken from the orbital lobule, and from the various superior gyri, showed few nerve cells, and these few had undergone almost complete fatty or fuscous degeneration. The nerve fibres were

* We submitted the specimen to Mr. Turner, Professor of Anatomy in the University of Edinburgh, who authorizes us to state that in his opinion the above description is correct. He would only add that he considers that rather more than one half of Broca's convolution was implicated.

found moniliform, and were so distinctly defined as to suggest that they were thickened. The vessels had, attached to their external coat, a finely granular material of a yellow colour and slightly refracting. This was in greater quantities at their bifurcations. On one large vessel was found great proliferation of nuclei.

Amyloid bodies were seen in large quantities in the deeper layers of the convolutions, but not very markedly present in the outer. In the former situation they were seen with a double contour, and in some cases with a granular centre. In size they varied from that of one to two blood corpuscles.

There was slight dilatation of the aorta and a few atheromatous patches on the valves, which were thin, and the mitral orifice was small.

It is needless to enter into the pathological conditions elsewhere in this autopsy. Suffice it to say that the cause of death was caries of the vertebræ, resulting in atrophy of the cord, which accounted for the paraplegia.

In order that this society might see the specimen in its entirety, no microscopic sections have been made; but this will be done within a few weeks, and the result reported. Whatever it may be, it cannot alter the conclusion to be drawn from the naked eye appearances.

Commentary.—Perhaps the most convenient method of studying the bearings of this case on the question of the localization of language will be to take up *seriatim* the three following conditions in cerebral symptomatology and pathology. There may be—

1st—Aphasia with a lesion.

2nd—Aphasia without a lesion.

3rd—A lesion without aphasia.

The first condition, aphasia with any brain lesion, may be a mere coincidence of circumstances; that is to say, this affection may be but a simple concomitant of a lesion, not the direct result of it.

Lesions occurring with aphasia are various in seat, extent, and kind. As regards seat, they have been found in the right hemisphere and in the left, in the anterior, the middle and posterior portions of each hemisphere, in the medullary as well as in the cortical substance of the brain, and in the medulla oblongata. Their extent has also been variable, some involving both frontal lobes, or only one, others the middle lobe of either side, while many have been circumscribed and confined even to portions of particular convolutions.

Their kind is generally threefold—softening, localized atrophy, and excavation.

That such different lesions should occur with the affection of speech, and this affection oftentimes the only indication of the mischief within, is, to say the least, strange and mysterious. The method of procedure which may in some degree tend towards the solution of the mystery is to calculate the frequency of the speech-perversion occurring with lesion of different portions of the brain, and to see if the result warrants the throwing aside of the coincident theory, and the adoption of a belief in their relations as causes and effects. It is from the frequency of certain lesions having aphasia as one of their concomitants out of which have sprung the many theories as to the seat of the organ in which resides the so-called faculty of articulate language. Each theorist has arrayed his morbid facts, and demanded credence in his belief; but a lesion occurring at the chosen spots without aphasia militates against this demand.

Explanatory theories have been advanced of the fact that aphasia occurs more frequently with lesions of the left hemisphere than with those of the right—according to some twenty times, according to others only three times more often. We allude to the education theory of Broca, Moxon, and its modification by Baillarger and Hughlings Jackson, that founded by Gratiolet on his observations on the priority of development, and that of Bateman on the direct blood supply. These failing, we must, with Dr. Bastian, confess “that we stand before a mystery, of which at present no adequate solution has been offered.”

That lesions are as common on the right side of the brain, taken as a whole, as on the left, is a statement which is daily borne out by facts, for hemiplegia, a frequent result of unilateral injury, is indeed slightly more common on the left than on the right side of the body. This is all the more curious, as the greater liability of the left side to embolus of the middle cerebral artery and consequent right hemiplegia throws the onus of the causation of the greater number of cases of left hemiplegia on the posterior and middle lobes of the right hemisphere.

The frontal lobes stand first in order of frequency as to the coincidence of lesion of their substance with aphasia, as compared with the other lobes. This may be probably explained by the fact that the anterior portion of the brain is much more tolerant of injury than the middle and posterior, where

such morbid conditions as we often find in the frontal lobes, would prove fatal within a period too short for the development of psychical symptoms. They are also, we believe, more frequently the seat of the kinds of disease which have been especially coincident with aphasia, and further, that the left frontal lobe, as contrasted with the right, stands in the same relation in order of frequency of lesion of its substance with aphasia, as do the frontal lobes to the rest of the cerebrum. The reason of this is that, from physical causes, emboli are peculiarly prone to resort to the left middle cerebral artery, and the consequence is the greater liability of the adjacent tissues to atrophy or softening, which, gradually implicating a vessel or vessels, cause an apoplectic clot in the region best suited physically for its presence, and where it is least likely to cause sudden death. Having, therefore, lesions in the left frontal lobe more frequently than in the right, it is not to be wondered at that they are found more often coincident with an affection of language common to many other lesions in other parts of the encephalon.

The question now arises, does the narrowing of the limits of these lesions, that is to say, their localization in a particular convolution, or part thereof, concurring with aphasia, lessen the probability of their being mere coincidences? Our answer is in the negative, and in so answering we take up the position that nothing less than the never failing association of aphasia with the lesion of the asserted seat, and never the lesion without the aphasia, can establish as a positive physiological fact that there is a locus for the faculty of language. Whether this co-relation has uniformly been present will be discussed when considering the third condition.

The lesion most commonly met with in the left frontal lobe is found in its postero-infero-external region, and being accompanied very frequently, although by no means invariably, by aphasia, it has been concluded that it involves the organ or locus whence emanates the faculty of language. Lesions are rare in this precise spot on the left side, for reasons indicated above, but certain of those which have been recorded have been noted as associated with an impairment of language. The question is, whether the proportion of these circumscribed lesions occurring on the left side, with aphasia, is not in equal ratio to the lesser frequency of those in the right side, also with aphasia, and whether this affection is not as common to one as to the other must be left to further pathological research. Unluckily we have comparatively few accurately re-

ported cases. We have a mass of reports of patients exhibiting the symptoms of aphasia, most of which are unsupported by pathological demonstration, and rest solely on symptomatology. The autopsies in which atrophy of this convolution on either side has been observed and correctly noted, are, considering the frequency of the symptom, extremely rare, and so contradictory as to throw doubts on the conclusions which have been deduced.

Language, according to many prominent authorities, appears to have two chief factors, viz., the memory of words, and the faculty which presides over the co-ordination of the movements by which words are produced. They seem to be independent of each other, as one can be affected without detriment to the other. This, however, is doubted by Professor Sanders, but our experience does not entirely coincide with his opinion. Each of these factors is said to have a locus of its own, injuries of which result in aphasia or amnesia respectively. M. Broca confines the co-ordinating power to the posterior third of the left inferior or third frontal convolution. In this Dr. Ogle agrees with him, but affirms in addition a belief that the seat of verbal memory lies in some contiguous convolution. Dr. Bastian does not locate the lesion productive of impairment of speech, but holds that aphasia is the result of injury to the efferent fibres between the cortical substance and the corpus striatum, or of that body itself; and Dr. Robertson, of Glasgow, is inclined to believe that these efferent fibres are those connecting the external convolutions of the left frontal lobe with the motor centres. Professor Sanders leans to the opinion that the Island of Reil is intimately connected with either affection, and Dr. Maudsley, mainly on metaphysical grounds, ignores the theory of localization altogether. The pathological appearances in the specimen now before you do not stand opposed to Dr. Bastian's theory, so far as amnesia is concerned, for a considerable area has been taken away by the excavation; but they do support his idea that the motor tract is diseased in aphasia, for we have no history of permanent hemiplegia, no lesion of the motor centres has been detected, and we have no clinical reason for supposing it ever existed. It will be remembered that the excavation was anatomically arrested at the extra-ventricular nucleus of the corpus striatum, and no aphasic symptoms presented themselves. These facts present negative support to this author's opinions. Nor does this case, taken alone, militate against

Dr. Ogle's rider on Broca's proposition; how it utterly breaks up their combined theory of localization of the co-ordinating faculty will be discussed under the third condition, that of a lesion and no aphasia.

The second condition, aphasia without a visible lesion, is by no means uncommon.* Functional derangement of the whole or part of an organ causes symptoms and perversion of action as well as apparent pathological change. The term "functional" may be objected to; it is here used simply as implying undemonstrable morbid change. Such derangements, in nerve tissue most particularly, are probably due to some alteration in the chemical composition of parts, some irregularity in molecular movements, or some change in the electrical conditions. "These are the inscrutable recesses of nerve life." The transitory, periodic, and recurrent nature of many attacks of aphasia and amnesia, and their causation from emotional excitement, render the supposition highly probable. Loss of speech, especially of the amnesic form, often arises from such general conditions as over exhaustion from excessive mental strain, or even from undue physical exertion, or from "the occurrence of some febrile illness."

This condition of aphasia without a lesion does not logically damage either the "cause and effect" or the "coincident" theory, as the maintainer of the first may assert that the locus, although not pathologically changed, has been functionally out of order, whilst the holders of the second might reply that it is impossible to have such an aberration of faculty, if permanent, without some resultant, organic, and demonstrable change. If not permanent their power of reply would be paralysed, as functional derangements are generally transitory. Until our knowledge of microscopic cerebral pathology is more advanced this position cannot materially affect the general question.

We now come to the consideration of the third and most important condition; most important, for on the demonstration of the existence of a lesion of the third left frontal convolution, *without aphasia ataxica*, the theories of Broca and

* Dr. J. Batty Tuke attended, some years ago, in company with Drs. Skae, Begbie, senior and junior, Sanders, and P. M. Deas, the autopsy on a distinguished Edinburgh physician, who for many years before his death had been the subject of gradually increasing aphasia, and in whose brain the only pathological appearances which presented themselves were general atrophy, with compensatory serum and adhesions of the dura mater to the calvarium. If anything, the general atrophy of the right frontal lobe was slightly more than that of the left. There was no definite lesion of individual parts.

Ogle, as to their being the absolute seat of the co-ordinating function which regulates speech, must fall to the ground. If this particular gyrus is the organ, its destruction must result in loss of the function. We believe the demonstration is before you. You have all seen that in addition to portions of three other convolutions (the ascending, parietal, and frontal) and the lower margin of the second frontal, the posterior half of Broca's gyrus (he is content with the posterior third) has been eroded, absorbed, and utterly destroyed, both as regards the white and grey matter. And what was the speech symptom? Not aphasia ataxica, which must of necessity have ensued had the theories in question been correct. Not a loophole for escape is left, for this woman was right-handed, and so nothing can be founded on the probability of the left hemisphere having been neglected in its education.

The lesion and phenomena of this case also stand opposed to the opinion of Dr. Robertson, as the medullary substance, the efferent fibres of which are considered by him to be conveyers of will to the motor centres in regard to articulate language, is destroyed.

The occurrence of a modified amnesia in this case must not be overlooked. The lesion is very extensive and complete, but the speech impairment was only partial. More thorough amnesia might have been expected from the nature of the injury, not only because three other important gyri are implicated, but also, if the seat of memory of words is in this neighbourhood, from the highly probable supposition that such a lesion would have affected the functional activity of a "contiguous" convolution. What is contiguity as applied to the brain? Very little research would suffice to constructively* destroy (if the expression may be permitted) the whole of the frontal lobes without any symptoms of amnesia having presented themselves in the various patients, and therefore it may be concluded that the "contiguous convolution" of Ogle does not lie in the frontal lobes.

A great deal of stress has been laid on the possibility of the coincidence of the occurrence of speech symptoms with lesions of the cerebrum at large, and of the third left frontal convolution in particular. The frequency of the observation is such as to preclude the conclusion that they play no part in

* On the evening on which this paper was read we exhibited to the Medico-Chirurgical Society a specimen in which the medullary substance of the left frontal lobe had been destroyed, owing to the presence of a large apoplectic clot. There had been no speech affection.

the causation of *amnesia*. They are, we believe, the causes, but by no means the necessary or direct ones, of the symptoms, simply on account of the influence their presence must have on the nutrition and function of the hemispherical ganglia generally, not on account of the destruction of any single particular convolution. The presence of such morbid conditions in so delicate an organ could not be expected to remain long without some results. Why, in such a large proportion of cases, speech should be affected, it is impossible to say, unless there be some connection between the decay caused by irritation and that caused by the natural decay of old age. Amnesia is perhaps the first symptom of the decay of old age, and it is at least noteworthy that under morbid influences the loss of the memory of words is an early symptom. We see no reason to doubt but that a similar lesion, if it *could* have existed in another part of the brain, would have produced the same result as far as word-memory is concerned, for to the irritation caused by it is due the general brain degeneration evidenced by the atrophy of the nerve cells, and the presence of large numbers of amyloid bodies in the three outer layers of the grey matter. Dr. J. Batty Tuke, in his investigations into microscopic cerebral pathology, has frequently observed that, in cases of senile insanity, the cells of the second and third layers of the grey matter of the superior surface of all the convolutions have undergone or were undergoing degeneration and atrophy. To this extent such lesions as that now before you may be held to be the cause of the effect, but by no means necessarily so, as irritation of the hemispherical ganglia productive of the same symptoms can be induced from other very various influences.

It is impossible to dismiss this case without regarding its bearings on *Aphasia*. This speech-symptom is very generally associated with hemiplegia, whilst the motor powers of the lips, tongue, and face are unaffected, and articulation, a delicate and complicated motor act, is the sole function of these parts perverted and destroyed. Hemiplegia consequent upon lesion of the ventricular portion of the motor tract is not unfrequently unaccompanied by Aphasia. These two facts, taken together, would seem to show that the store-house of the faculty of articulation has a definite locus.

In this case we had no paralysis, no aphasia, and no implication of the extra-ventricular nucleus, the disease having been anatomically arrested at that body. In the study of future cases, in which lesions of any part of the motor centres

are accompanied or not accompanied by aphasia, it will be advisable to accurately note the extent and direction of the morbid tracts in relation to this nucleus. Judging from its abundant vascular supply, and from the large size and number of its cells, its function must be important. It is impossible, on existing records, to suggest an hypothesis as to how far this portion of the corpus striatum is associated with the faculty of articulate speech; we merely offer the suggestion in order that pathologists may carefully observe the extension of disease from within outwards, and from without inwards, in relation to this nucleus, and tabulate them, with clinical observations as to speech-affection. In its favour we may add the following probabilities:—First, that lesions of the frontal convolutions which have been associated with aphasia had extended inwards to this nucleus, and those which have been without aphasia had not (this case is an example); and, second, that those lesions which have caused hemiplegia *and* aphasia had extended forwards and outwards to this nucleus, and those without had not.

However, the only definite result we claim as deduced from this case at present is its complete testimony to the erroneous nature of Broca's convolutional localization.

