A REMARKABLE CASE OF APHASIA.

Acute and Complete Destruction by Embolic Softening of the Left Motor-Vocal Speech Centre (Broca's Convolution), in a Right-Handed Man: Transient Motor Aphasia, Marked Inability to Name Objects and Especially Persons, Considerable Agraphia and Slight Word-Blindness.

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It is, I think, no exaggeration to say that the case which I am about to relate is one of the most remarkable cases of aphasia which have ever been recorded, and that it is of very great importance both from a physiological and a pathological point of view. The details are as follows:—

A. B., aged 70, was seen with Dr. Menzies on March 4, 1898, at 11 a.m.

Previous History.

The patient has been a strong robust man, in the habit of attending business with great regularity. Some years ago, he suffered from symptoms suggestive of angina pectoris; at that time Dr. Menzies suspected the presence of fatty heart. After a few weeks, he completely recovered from this attack, and has been perfectly well ever since. Of late years, he has had a good deal of business worry.

History of the Present Illness.

His wife gave me in writing the following account of the commencement of the present attack:—

"On February 28, I left Mr. B. at his office at half-past four o'clock, in good health and spirits. He came home by the train which arrives at —— Station at a few minutes after six o'clock. I understand that as he was coming up the steps leading from the station his umbrella dropped from his right hand, but he was able to pick it up and to gain the landing. He then entered an omnibus which was waiting and which passes our door. On alighting from the omnibus, he opened the door with his key and came into the dining room. He then took off his boots and put on his slippers, but did not speak a word. I was surprised at this. I asked him what was the matter, but he only shook his head. I, at first, thought that he must have had some heavy business loss, but soon saw that he was ill. I became much alarmed and gave him a little whisky-and-water; this seemed to give him some relief. We then had tea; he was not able to eat anything, but drank a little tea. He then left the room and went upstairs to the bath-room; I followed shortly afterwards and found him smoking. I asked him if he felt any better, and he said, 'Yes.' I waited with him until he came down-stairs. After coming down to the dining room he took up the newspaper and tried to read, but he soon tired of it. I then asked him to lie down on the sofa and rest, but he would not do so; *he said he would take a game at bezique;* we had a game, but he made mistakes of which I took no notice. I thought he had had a shock of some kind, but he rather resented the idea of being ill and would not allow me to get a doctor. During the evening he spoke very little, but he said some words—for example, he said that he 'would have a game at bezique.'

"*Next morning* (*March* 1), he would not be persuaded to lie in bed, but rose as usual at six o'clock. His speech was better than on the previous night. We had breakfast at seven, and I prepared myself without his knowledge to accompany him to town. He was not very well pleased when he saw me dressed, and asked me where I was going; I said I wished to go with him. I went to the station, but he forbade me to come further, and I saw that if I persisted it would only make him angry. I was, therefore, forced very unwillingly to go home again. In the forenoon, I went to his office and saw him; he looked very well and said he felt better. I, however, without his knowledge, went along and saw Dr. Menzies, who advised me to persuade Mr. B. to go home as soon as possible, and promised to come out and see him. I expected to have some difficulty in getting him to go home with me, but he went quite willingly."

Dr. Menzies, who saw the patient on the afternoon of March 1 (the day after the onset), gave me the following account of his condition :—

"Mr. B. was in the drawing-room. When I went into the room he rose up and shook hands with me, and said, 'How do you do? How do you do?' He then spoke about the weather. At first, while he was speaking about ordinary things, I noticed nothing wrong; there was no hesitation and no thickness of speech. After some conversation I asked him, 'Do you know me?' and he said, 'Yes, yes, perfectly, of course.' I then said, 'Well, what is my name?' but he could not tell me. He understood everything that was said to him, and correctly answered the questions which I put to him. He seemed at a loss for some words, but the chief defect in speech, which I was able to make out, was that he could not name persons and objects. I noticed no paralysis of the face, but I thought he dragged the right toe slightly in walking. The pulse was slow —50 per minute. I then tested his writing and found that he could write very little. I advised that he should keep indoors and not go to business the next day. He did not, however, accept this advice."

The next day (*March* 2), he insisted upon going to business. While at business he was unable to write; he spoiled three cheques in trying to sign them.

On Thursday, March 3, he kept his bed. He had been annoyed about the mistakes he made in drawing the cheques the previous day, and realised that he was ill and unable to do his usual work. The temperature was 100°. He was speaking better than the day before, saying more words. I tested his power of writing and found he was able to sign his name fairly well.

PRESENT CONDITION (FRIDAY, MARCH 4, 1898).

When I saw him at 11 a.m. he was in bed (he had been kept in bed for the purpose of my examination). He was a fresh, robust-looking man, and looked younger than his age.

I entered into conversation with him; he seemed to understand everything that was said to him, spoke quite rationally and properly, and did not seem to be at a loss for words. I could detect no defect in speech until I came to ask him to name persons and objects. Dr. Menzies had previously told me that he was unable to name persons or objects. When I asked him what his own name was he could not say it;

he could not name Dr. Menzies or his own wife. When I asked him, "What is your wife's name?" he said, "Sugar and spice" four times over. (His wife was not in the room when the examination was made.) I then showed him various objects—a knife, a button-hook, a tuning-fork, a paper-knife—but he was unable to name any of them. A key he said was "a lock" (paraphasia); this was the only attempt which he made to name any object shown to him. He was able to repeat his own name and the names of objects which he was unable to name spontaneously.

There appeared to be a slight degree of word-blindness.

He could read aloud fairly well. He was asked to read aloud the following heading to a prospectus which was lying on the table:—*The Edison-Bell Consolidated Phonographic Company, Limited.* He read it correctly except the word *Phonographic*, which he read as "Photographic." He was then asked to read the following sentence:—*List of some of the Users of the Commercial Phonograph—Continued.* The word *Users* he read as "Liners," the word *Commercial* as "Farewell" and the word *Phonograph* as "Photograph." He read the word *Dog*, which I had written down, correctly.

His writing was then tested. He was first asked to sign his name, and did so correctly, the signature being not at all bad. He was then asked to write his wife's name; he again wrote his own name. He was next asked to write Dr. Menzies' name; he again wrote his own name. Asked to write the letter *o*; wrote *o* followed by two words which looked like *of zinc*. The numbers 20 and 100 were written in the same way—20 correctly, but it was followed by *of zinc* or *of June*. He was able to read his own name, which I had written. He was then asked to write his wife's name, and he wrote "Dear Sir," "Dear Surgises," several times over—in fact, he wrote this or his own name for almost everything he was asked to write. When asked to write the words "one hundred," he did so correctly. Asked to write a thousand, wrote "one forager." He read several words which he could not write. Was again asked to write the number 20; wrote "twevry flory" followed by "20" in figures. When asked to write down the individual letters *a*, *s*, *o*, he failed to do so.

Summary of the Speech Defects.

The chief defect was inability to name persons and objects. There was also a considerable degree of agraphia and paragraphia; a certain, though slight, degree of word-blindness, and some diffi-

culty in reading aloud, which was perhaps a motor defect. Except for naming objects and persons (if this was a motor defect), there appeared to be no motor-vocal aphasia. There was no word-deafness.

The patient was a right-handed man and had always been so. After the *post-mortem* examination I again made particular enquiries as to this point. His wife assured me that her husband had always been right-handed and had never used his left hand more than any ordinary right-handed person is in the habit of doing. His sister stated that as a child the patient was right-handed and that none of the family (father, mother, two brothers and two sisters) were left-handed.

Except the aphasic defects described above and a slight degree of tremor of the right hand, apparently due to commencing paralysis agitans, there was absolutely no indication of any nervous lesion. I could detect no paralysis either in the face, tongue, arm, or leg. The knee-jerks were equal and normal. There seemed to be no disturbance of sensation; in particular there was no hemianopsia.

His wife stated that for the first two or three days after the attack the patient had fed himself with his left hand. She further said that he had been shaky in his right hand for some considerable time, apparently the result of slight paralysis agitans; she did not think he was more shaky since the attack came on than he was before.

The heart seemed somewhat dilated, the aortic second sound accentuated, the first sound in the mitral area impure. The urine was free from albumen.

Diagnosis.

The diagnosis was embolism of one of the branches of the left middle cerebral artery, with softening in some part of the speech area. The symptoms seemed to me to suggest that the lesion was situated in the neighbourhood of the visual speech centre, and that the impulses passing from this centre (and perhaps also from the auditory speech centre) to the motor speech centre (Broca's convolution) and to the motor centre for writing were probably, in part at least, interrupted.

There was no evidence of any lesion in the auditory speech centre.

The fact that motor speech was not affected, except for naming objects and persons (if that was a motor defect) seemed

to show that the motor-vocal speech centre (Broca's convolution) was not directly affected by the lesion.

The patient's inability to name persons and objects was the most striking speech defect, and at the time of the consultation Dr. Menzies said: "If there is a naming centre, that is the seat of the lesion in this case." The patient was not only unable to spontaneously name persons (such as his wife—who was not present in the room when he was asked to name her), but was unable to name objects which were shown to him.

Subsequent Progress of the Case.

I did not see the patient again during life, but Dr. Menzies has kindly given me the following account of the subsequent progress of the case:—

"After our consultation the patient improved slowly, but the aphasic symptoms still persisted in some degree. The great difficulty was in recalling the names of persons. There was no other defect in motor speech; for example, I would go in to see him and he would talk quite well, say 'How are you to-day, Doctor? It is a cold morning.' I would ask him, 'How do you do?' He would say, 'Quite well, thank you,' etc. Then I would ask him at what hour he got up this morning, and he would tell me he got up at seven o'clock. I would then ask him what he had for breakfast, and he would tell me correctly. He would talk in this way for about five minutes, and then I would ask him, 'Do you know me?' and he would say 'Yes, quite well, perfectly.' Then I would say, 'Well, what is my name?' and he could not name me. Then I would ask his wife's name, and he could not name her. He was annoyed that he could not do so. Except during the earlier stages of the case, I did not notice any other defect of vocal speech except his inability to name persons and objects.

"He continued to have considerable difficulty in writing for some time after our consultation. He usually could write his own name, but tended to introduce more letters than were necessary, and the same with numbers.

"*On March 9* he wrote my name correctly when asked to write 'Dr. Menzies;' after an interval, when asked to write it a second time, without the name being mentioned, he wrote 'Dr. Magnession.' He was then asked to write his own name and did so correctly; asked to write it a second time, wrote 'Dr Dr do do'; asked to write it a third time, wrote 'Dr Dr do do' (meant for ditto). Was asked to write a hundred, wrote it

correctly; then added, '£100-0-0' in figures and his own name; 'one hundred £100 0 0' and his own name. Wrote the letters A, C, D, Z, N correctly when asked to do so. Wrote 'One hundred pounds,' his own name and address, when asked to do so. When asked to write his wife's name (the name not being mentioned to him) wrote 'Mrs. Bagginny.' When asked to write Dr. Menzies' name, wrote 'Mr. Dr. Menzies.'

"He knew my name, and sometimes if I asked him to write my name, Dr. Menzies, he would write it; and if I gave him what he had written before—perhaps on some previous occasion he had written my name—he could read it, but only by spelling it out, letter by letter, and then he knew that that was my name (persistence of some word-blindness). He never could say spontaneously my name or his wife's name. Sometimes he could name objects which were shown to him; for example, on one occasion I picked up a cup from the table in the drawing-room and asked him what it was, and he named it correctly. Names of persons and objects which he could not himself spontaneously utter he could repeat. For example, on one occasion on which he could not mention his own name, after I named him he said, 'Yes, A—— B——, of course, A—— B——.' He could repeat everything (names, etc.), which he was asked to repeat.

"His power of reading aloud gradually improved. During the course of the illness he made his will; he was quite capable of making it. He asked me to arrange this for him, and I advised him to get a lawyer to draw it up for him. When the lawyer visited him for the purpose of making his will, he spoke quite well and explained what he wanted. The lawyer then read over the will, explained its general provisions and asked him if it was what he intended to do, and he said, 'Yes, perfectly.' He made no alterations or additions. The will was a very simple one; he left everything to his wife.

"A peculiar thing was that in his previous illnesses it was difficult to get him to remain at home or to lay up; but in this illness, after the first few days, he seemed to be indifferent about his business; he did not even care to go out, and rarely made an attempt to do so.

"*On Friday, April* 8, he went to business. He had been down to his place of business, but had not done much, on several previous occasions. (On this day his wife says that he was better than he had been since the attack commenced.) He remained at his business the whole day, visited his two business

places in Edinburgh and Leith, looked through the books, took an intelligent interest in what was going on, gave instructions, etc. His foreman did not notice that there was anything the matter with him. He made no mistakes. He drew and signed several cheques quite correctly. I have seen the counterfoils of these cheques, and I find that he had himself written the names of two or three firms and had spelt these names correctly. The cheques were drawn by his own hand.

"He never wrote any letters during the course of his illness. He read very little, but he never had been a great reader. He never complained of his sight.

"*On April* 9 he complained of pain in the region of the stomach. I saw him the next day (April 10). He did not look ill, but there was considerable tenderness over the abdomen. The pulse was slightly quickened—about 80 per minute—but there was no temperature. Up to this date, from the onset of the aphasic attack, the pulse had been slow (45 to 50 per minute).

"*On April* 11 the temperature went up to 100°, and the pulse to about 96. He complained of intense pain over the abdomen and great tenderness on pressure, and he vomited frequently. The abdomen was moderately distended. Peritonitis was obviously present. During the attack the bowels were more than once freely opened by medicine. There was no intestinal obstruction and no apparent cause for the peritonitis.

"*On April* 12 *and* 13 he remained much *in statu quo*.

"*On April* 14 he vomited a small quantity of blood.

"*On April* 15 he died. He remained mentally quite clear up to the end. On the day of his death he drew and signed a cheque quite correctly; the writing was good."

Post-Mortem Examination.

The *post-mortem* examination was made on April 16, 1898, at 3.30 p.m., by myself, Dr. Menzies and Dr. Steven being present.

The body was well nourished. The skull cap was thin, the dura mater very firmly adherent along the vertex. Several marked depressions in the bone were present, the result of the pressure of Pacchionian glands on the vertex, where the dura was adherent.

The convolutions of the *brain* were slightly atrophied. There was a marked depression in the position of Broca's convolution and the lower end of the left ascending frontal convolution. The exterior of the brain was otherwise normal. The brain was at once placed in formalin, and after it had become sufficiently

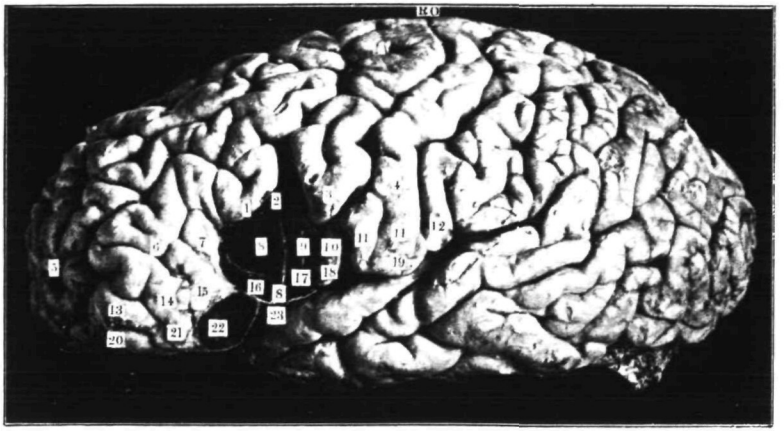

FIG. 1.

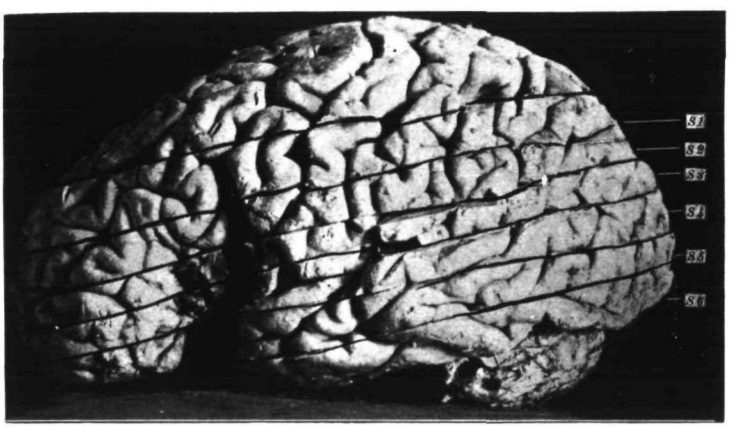

FIG. 2.

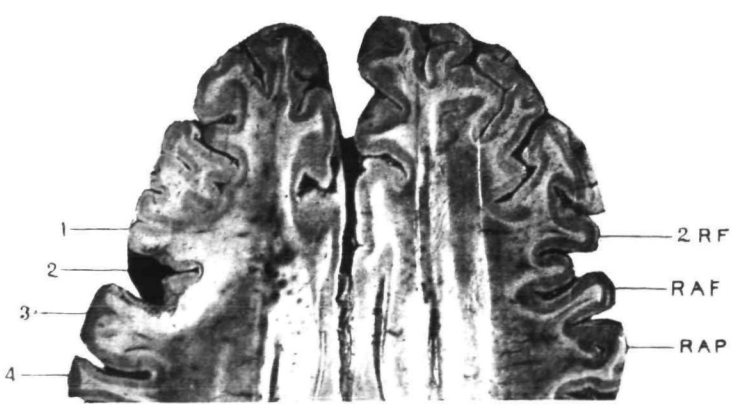

FIG. 3.

hardened was cut into a series of horizontal sections. A more minute description of these sections is given below.

On opening the abdomen several coils of the small intestine were found to be black and gangrenous, and there was general peritonitis. There was no constriction and no hernia, either externally or internally. The portion of mesentery going to the affected part of the small intestine was markedly swollen and œdematous. The gangrene of the intestine was obviously due to blocking of the mesenteric artery. The abdominal aorta was somewhat atheromatous.

The *heart* was moderately dilated, the valves healthy, the muscular substance markedly fatty and degenerated. The *thoracic aorta* was atheromatous. The *kidneys* were slightly enlarged and somewhat granular. (The urine was tested on several occasions during the course of the patient's illness, and was always free from albumen.) A few recent small petechial hæmorrhages were scattered over the mucous membrane of the *stomach*.

No other lesions were present in the body.

Subsequent Examination of the Brain.

After the brain was sufficiently hardened in formalin (10 per cent. solution), the left and right hemispheres were photographed with the membranes *in situ*.

The photograph of the left hemisphere shows a marked depression (shrinking due to softening) in the position of the posterior end of the lower (3rd) left frontal convolution (Broca's convolution).

The membranes were then carefully removed, and the left and right hemispheres were again photographed.

The depression (due to softening and atrophic shrinking) in the position of the posterior end of the lower (3rd) left frontal convolution was still more apparent.

The extent of the softening is exactly represented in fig. 1.

A small portion, the uppermost portion, of Broca's convolution (on which the figure 2 in fig. 1 is placed) appeared, at first sight, to have escaped; but although it was not shrivelled and completely destroyed, it was markedly affected (softened). This is not brought out in the photograph, but is distinctly seen in the brain itself.

The part of the lower (3rd) left frontal convolution on which the figure 22 in fig. 1 is placed, and a part of the convolution on which the figure 7 is placed, were also markedly softened; this

is well seen in the actual preparation and in the longitudinal (horizontal) sections reproduced in figs. 3, 4, 5 and 6, and in fig. 7.

When I first examined the specimen I was disposed to think that the lower end of the left ascending frontal convolution was affected (softened and atrophied), but subsequent examination showed that this was not the case. I may here state that I have had the advantage of Sir William Turner's opinion, and that he agrees with the description which I have given of the locality of the lesion and the arrangement of the convolutions in this case.

In this particular brain the lower end of the left ascending frontal convolution (on which the figures 10 and 18 in fig. 1 are placed) is relatively narrow (small), and the "foot" and ascending portion of Broca's convolution (on which the figures 9 and 17 are placed) is relatively broad (large). At first sight I was consequently disposed to think that the posterior end of the lower (3rd) left frontal convolution was the lower end of the left ascending frontal convolution.

The figures in fig. 1 and in figs. 3, 4, 5 and 6 point to identical parts of the convolutions. By this procedure the exact localisation of the softening and of the different convolutions in the longitudinal (horizontal) sections can be determined at a glance.

The brain was then cut into a series of longitudinal (horizontal) sections, and *the upper surface* of each of these sections was then photographed.

Finally, the sections were placed in accurate apposition, and the brain (left side) was again photographed (see fig. 2) so as to show the exact level of the various sections (1, 2, 3, 4, 5 and 6).

The *first section* (to which S. 1 in fig. 2 points) was absolutely normal, and has not therefore been reproduced.

The *second section* (see fig. 3) to which S. 2 in fig. 2 points, shows a small area of softening, which corresponds to the uppermost point of Broca's convolution, the point on which the figure 2 in fig. 1 is placed.

The *third section* see fig. 4) to which S. 3 in fig. 2 points, shows a marked area of softening involving the posterior end of the lower (3rd) left frontal convolution and the anterior end of the island of Reil.

The *fourth section* (see fig. 5) to which S. 4 in fig. 2 points, shows a still more extensive area of softening, which involves Broca's convolution, and the grey matter of the anterior end of the left island of Reil.

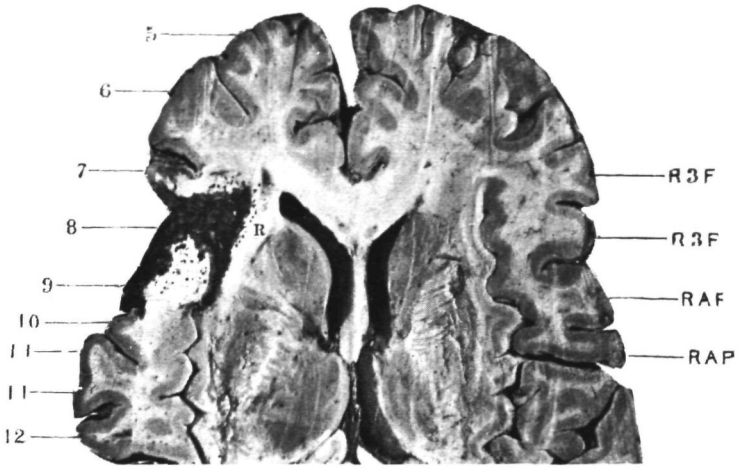

FIG. 4.

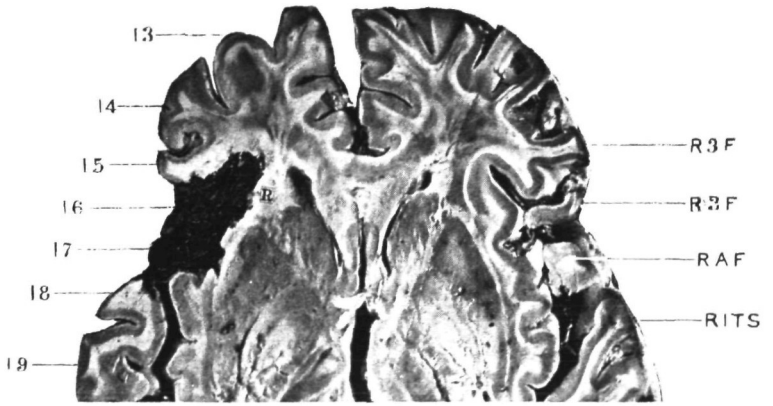

FIG. 5.

FIG. 6.

FIG. 7.

The *fifth section* (see fig. 6) to which S. 5 in fig. 2 points, shows a small area of softening corresponding to the orbital part of the lower (3rd) left frontal convolution.

It should be noted that this section was not quite horizontal, the right frontal lobe is cut through at a lower level than the left; hence the portion of the left frontal lobe which is seen in the section is considerably larger than the portion of the right frontal lobe seen in the section.

Finally, the inferior surface of the frontal lobes was photographed; in other words, section 5, fig. 2, was reversed and its under surface photographed.

The small part (anterior end of the left inferior frontal convolution) to which the figure 24 points and which is shaded black, was distinctly affected (yellowish-brown in colour and softened). This is not apparent in the photograph, but it is very apparent in the actual preparation.

I took the opportunity of showing both the sections themselves and the photographs to Professors Ferrier and Joseph Collins at the recent Edinburgh Meeting of the British Medical Association, and these authorities spontaneously remarked that the area of softening was much better seen (owing to the difference in colour) in the actual sections than in the photographs, and was more extensive than one would have supposed from an examination of the photographs.

Remarks.

The point in this case which I wish to specially emphasise, for it seems to me to be of supreme interest and importance, is that in this patient, who was a right-handed man and in no sense more left-handed than the great majority of right-handed persons, acute and complete destruction of the left motor-vocal speech centre (Broca's convolution) and of the anterior end of the left island of Reil merely produced a very temporary motor aphasia, and did not produce, as one would have expected it to produce, complete and persisting motor-vocal aphasia.[1]

All authorities on aphasia are agreed:—(1) that in right-handed persons the motor-vocal speech centre is

[1] I use the term *persisting* in contradistinction to *temporary*, but it must be remembered that the term "persisting" is not synonymous with *permanent*, for in some cases in which aphasic symptoms persist for some time they are not permanent.

situated in the posterior end of the lower (third) left frontal convolution (Broca's convolution); in other words, that Broca's convolution is "*the way out*" for spoken (vocal) speech; and (2) that complete destruction of the left motor speech centre (Broca's convolution), if acutely produced in a right-handed person, should produce complete and persistent (though not, of course, necessarily permanent) motor aphasia, or, as I term it, motor-vocal aphasia.

Up to the present time almost all authorities on aphasia have emphatically maintained:—(1) that, in right-handed persons, the speech centres are located in the left hemisphere of the brain; and (2) that the corresponding cortical centres in the right hemisphere are not concerned with speech. Though almost every one will, I presume, allow that the corresponding centres in the right hemisphere can, in some cases at all events (more especially in young subjects), be trained to take up and carry on the function of the speech centres in the left hemisphere, when the speech centres in the left hemisphere are destroyed; and that under such circumstances the way out for spoken speech is through the posterior end of the third *right* frontal convolution.

Consequently, according to the usually accepted view, complete destruction of Broca's convolution, if acutely produced in a right-handed person, will *necessarily* (*i.e.*, always) cause complete and persisting motor-vocal aphasia; and the aphasic defects produced in this way can only be recovered from (and in the adult this recovery is usually very imperfect) by a slow and gradual process of education and training of the corresponding (motor-vocal speech) centre in the right hemisphere of the brain.

The case which I have related above seems to me to show that these statements do not invariably hold good; in other words, that exceptions to the usually accepted theory do occasionally, though, of course, I admit *very rarely*, occur. It proves, in my opinion:—(1) That acute and complete destruction of Broca's convolution, in a right-handed person, does not necessarily produce complete and persisting motor-vocal aphasia, but that, in exceptional

cases, such destruction may merely cause a very temporary and slight degree of motor-vocal aphasia.

(2) That in right-handed persons "the way out" for speech is not necessarily and invariably through Broca's convolution (the posterior end of the lower *left* frontal convolution).

It seems obvious that in this right-handed man, after the occurrence of the lesion, motor-speech impulses did not pass out through Broca's convolution, for almost immediately after the occurrence of the lesion, *i.e.*, almost immediately after Broca's convolution was completely destroyed, motor-speech impulses continued to pass out in what was to all intents and purposes a normal manner.

The question therefore occurs, through what part of the brain did they pass out?

There can only, I think, be one satisfactory answer to this question, viz., through the posterior end of the lower (3rd) frontal convolution *on the right side of the brain*, unless the terms "Broca's convolution" and the "motor-vocal speech centre" are allowed to include the inferior end of the left ascending frontal convolution, which was not implicated by the lesion in this case.[1]

The only theory which seems to me to afford a satisfactory explanation of the facts of this exceptional case is that, in this patient, the cortical centre in the right hemisphere corresponding to Broca's convolution, or the right motor-vocal speech centre, as I term it, was more highly educated than it is in the great majority of right-handed people—in other words, that, in this particular patient, the motor-vocal speech centre in the right hemisphere was

[1] Recent observations would seem to show that the lower end of the ascending frontal convolution (which is separated from the posterior end of the 3rd left frontal convolution by the inferior end of the præ-central sulcus) is the ordinary psycho-motor centre for the lower part of the face, tongue and larynx, and does not form a part of the motor-speech centre properly so called. Further, it is usually supposed that the motor-speech centre does not include the whole of Broca's convolution, but is limited to that portion of the 3rd left frontal convolution which is bounded behind by the præ-central sulcus and in front by the ascending limit of the fissure of Sylvius (*i.e.*, the "foot" and ascending portions of Broca's convolution). According to this view, the portion of Broca's convolution (the "cap") which is situated in front of the ascending limit of the fissure of Sylvius does not form part of the motor-speech centre.

sufficiently educated and active to *immediately* take up and carry on the function of the left motor-vocal speech centre when that centre was *acutely* destroyed.

For several years past I have been in the habit of teaching:—(1) that the commonly-accepted view that the left hemisphere is alone concerned with speech in right-handed persons (and the right hemisphere in left-handed persons) is erroneous; (2) that, in right-handed persons, the cortical centres in the right hemisphere (corresponding to the speech centres in the left hemisphere) must be possessed of some sort of speech function (and the reverse in left-handed persons); (3) that, in right-handed persons, the relative activity of the different speech centres (the auditory speech centre, the visual speech centre and the motor-vocal speech centre) in the right hemisphere is probably different in the same individual, the right auditory speech centre, for example, being, I think, in most individuals, probably more highly educated and more active than the right motor-vocal speech centre; and (4) that, in right-handed persons, the relative activity of the speech centres on the left and right sides of the brain, or perhaps it would be more correct to say, the relative degree of activity which the different speech centres in the right hemisphere of the brain possess (for, so far as our present information enables us to judge, in right-handed persons the speech centres in the left hemisphere are always the "active" or "driving" centres), probably varies considerably in different individuals.[1]

The experience of all observers goes to show that the motor-vocal speech centre in the right hemisphere (though, in my opinion, it probably possesses in every right-handed individual some sort of active speech function, which, I presume, is carried on in conjunction with, *and in subordination to*, the function of the active or "driving" motor-vocal speech centre in the left hemisphere) is in the great

[1] In my Lectures on "Aphasia" (published in the *Edinburgh Medical Journal*, vol. ii., 1897) I have thrown out the suggestion that in some right-handed persons whose ancestors were left-handed the active or driving speech-centres may *possibly* in *rare instances* be situated in the right hemisphere; but while this is, I think, theoretically probable, no case in which it actually occurred has, so far as I know, been recorded.

majority of right-handed persons not sufficiently educated and active to immediately take up and carry on (*i.e.*, to independently carry on) the motor speech-function when the active or driving centre (Broca's convolution) in the left hemisphere is acutely and completely destroyed.

But that, in some persons, more especially in young subjects, the motor-vocal speech centre in the right hemisphere may, in the course of a short time, become so perfectly trained and educated as to be able to actively carry on the speech function without any obvious defect, after Broca's convolution (the active or driving motor-vocal speech centre) has been acutely and completely destroyed, is *conclusively proved* by Barlow's well-known case.

Now, the present case seems to me to show that, in very rare and quite exceptional cases, the right motor-vocal speech centre in right-handed adults is sufficiently educated and active to *immediately* take up and carry on (*i.e.*, to independently carry on) the speech function (the emission of motor speech-impulses) when Broca's convolution is acutely and completely destroyed.

And if this be allowed, it is reasonable, I hold, to maintain (as I have long argued) that the degree of functional activity and educational endowment which the right motor-vocal speech centre (and indeed all the speech centres) possesses in different right-handed persons is probably variable. I presume :—

(1) That in the vast majority of right-handed persons the functional activity of the right motor-speech centre is probably slight—so slight as to be insufficient to enable it to *immediately* take up and carry on (*i.e.*, to independently carry on) the motor-vocal speech function in any obvious or in any considerable degree when the driving motor-vocal speech centre (Broca's convolution) is destroyed.

(2) That in very rare instances, of which the present case is, I suggest, an illustration, its functional activity and educational endowment are so perfect that the speech function can be *immediately* taken up and, practically speaking, almost perfectly carried on when the left motor-vocal speech centre is acutely and completely destroyed.

And (3) that between these two extremes there are probably all degrees of difference; though, as I have already remarked, the experience of all observers goes to show that the cases in which the right motor-vocal speech centre is sufficiently educated and active to carry on the speech function under such circumstances (*immediately* after acute and complete destruction of Broca's convolution), with any degree of completeness, are altogether exceptional.

I maintain, then, that this case seems to me to corroborate and strongly support the view which I have for a long time advocated, that the speech centres and speech functions are bilaterally represented in the brain—the view which supposes that, in right-handed persons, the cortical centres in the right hemisphere which correspond to the speech centres in the left hemisphere must be possessed of some sort of speech function. Though, as I have already stated, I fully admit that, in the great majority of right-handed persons, the left hemisphere is, so far as the speech functions are concerned, the active or "driving" side; and that the degree of educational endowment and functional activity of the speech centres in the right hemisphere are usually quite insufficient to enable the speech function to be actively carried on when (immediately after) the speech centres in the left or active hemisphere are acutely and completely destroyed.

This view I prominently advocated in a series of papers and lectures which were published in the *Lancet*[1] in 1897 (March 20, *et seq.*); and in the *Edinburgh Medical Journal*, vol. ii., 1897, I have discussed the question in considerable detail.

Additional Points of Interest.

In addition to the point which has just been emphasised, the case presents several other points of considerable interest. Some of them are as follows:—

[1] The papers in the *Lancet* (which appeared simultaneously with Dr. Charlton Bastian's lectures, in which a similar view as to the bilateral representation of the speech centres was advocated) had been in the hands of the Editor of the *Lancet* for six months before they appeared.

(1) *Absence of Paralysis of the Face, Tongue, &c.*

So far as I could detect, there was no paralysis of the face, lips, or tongue on the opposite (right) side five days after the occurrence of the lesion; and Dr. Menzies assures me that, when he saw the patient twenty-four hours after the onset of the attack, he did not observe any paralysis in these parts.

It seems certain, however, that the occurrence of the lesion was followed by some (temporary) motor weakness on the opposite (right) side of the body. This is shown by the following facts:—At the moment when, it is reasonable to suppose, the embolism occurred, the patient dropped his umbrella which he was carrying in his right hand; the next day Dr. Menzies thought that he dragged the right toe slightly; and for some days after the onset of the attack he did not feed himself with the right hand, as he had previously been in the habit of doing.

And since the onset of the lesion was attended with some (temporary) loss of motor power in the right arm, and apparently also in the right leg, it is reasonable to suppose that the onset of the lesion was probably attended with some motor weakness (paralysis, or paresis, as some writers would term it) in the muscles of the face, lips, tongue, &c., on the opposite (right) side of the body. (The temporary paralysis in the right arm, in (?) the right leg, and in (?) the right side of the face, tongue, &c., if there was temporary paralysis of the face, tongue, &c., was obviously due either to shock and inhibition or to temporary vascular changes produced by the lesion.)

But if such paralysis existed, it must have been very slight and it must have rapidly passed off. The very slight and very temporary character of this facial paralysis (if it did occur) is quite satisfactorily explained by the fact that the lower end of the left ascending frontal convolution (the ordinary psycho-motor centre for the lower part of the face, the lips, tongue and larynx) was not affected. That in man the lower end of the ascending frontal convolution is the ordinary psycho-motor centre for the lower face, lips, tongue

and larynx, and that it does not form part of the motor-vocal speech centre seems shown by a case recorded by Elder in which the lower end of the left ascending frontal convolution was acutely destroyed with the result that there was paralysis of the lower facial muscles, tongue, &c., *but no motor aphasia*, Broca's convolution not being implicated by the lesion. Further, it must be remembered that many of the facial movements are bilaterally represented in the cerebral cortex and can be put into action from either hemisphere.

(2) *The Alterations in the Condition (functional activity) of the Visual Speech Centre, of the Motor Writing Centre, and perhaps of the Auditory Speech Centre, which were present in this case.*

The lesion, as we have seen, was strictly limited to the motor-vocal speech centre (Broca's convolution) and to the anterior end of the left island of Reil. But when I saw the patient (five days after the onset) and for some time afterwards, there was a certain (though slight) degree of word-blindness, considerable agraphia and paragraphia, and almost complete inability to name objects and persons. These symptoms were so marked and the motor aphasia was so slight (when I saw the patient there was, practically speaking, no motor aphasia, unless the inability to name persons and objects was a motor defect) that I was disposed to think that the lesion was situated in the neighbourhood of the visual speech centre, and that it probably more particularly implicated the fibres passing from the visual speech centre to the writing centre. I never for one moment suspected that the motor-vocal speech centre (Broca's convolution) was completely destroyed, as the autopsy subsequently demonstrated.

The case shows how difficult it is, in some cases, to predict the exact locality of aphasia-producing lesions; though I admit, of course, that this case is altogether an exceptional one, and I allow that, in the vast majority of cases in which one of the speech centres is acutely destroyed and in which the lesion is limited to that centre, the

aphasic symptoms which result are sufficiently characteristic to enable one to localise the lesion with certainty and accuracy.

The slight degree of word-blindness which was present in this case cannot, so far as I see, be explained in any other way than by supposing that the destructive lesion of the motor-vocal speech centre produced temporary disturbance in the action of the visual speech centre. And if this is the correct explanation of the mode of production of the slight word-blindness, the case corroborates the view of those authorities (such as Déjérine, Mirallié and Joseph Collins) who maintain that the action of the different speech centres (auditory, visual and motor), which are comprised in what Freund terms the "*zone of language*," is very intimately connected; and that a lesion in one centre (say the motor-vocal speech centre) is apt to produce (these authorities would, I suppose, say will necessarily produce) some disturbance in the action of the others (say the visual speech centre).

But here I would remark that it is impossible to argue on hard and fast lines, and to affirm that the same lesion will necessarily produce exactly the same symptoms or results in every case. It is of the utmost importance, I think, to recognise the fact that individual differences in the relative degree of the functional activity of the individual speech centres, *and of the closeness and intimacy of their functional activity and connections*, as it may be termed, occur in different cases; and that in one case in which this connection is close, a lesion (say of the motor-vocal speech centre) will produce considerable disturbance (say in the action of the visual speech centre)—as it seemed to do in this particular case; while in another case, in which the connection is less intimate, the same lesion will produce little or no disturbance. There is every reason, I think, to suppose that in some persons (those who are accustomed to read and write much) the action of the visual speech centre becomes so highly specialised and independent that the act of reading and writing can be largely (possibly entirely) carried on independently of the action of the

motor-vocal speech centre; while in other persons (who are accustomed to read and write little) the action of the visual speech centre is much less highly specialised and independent of the action of the motor-vocal speech centre.

In this particular case, in which, I presume, the functional connection between the motor-vocal speech centre and the visual speech centre was intimate and close (and with reference to this point it may be of importance, as it certainly is of interest, to note that the patient had never been a great reader), destruction of the motor-vocal speech centre (Broca's convolution) produced a certain, though slight, degree of word-blindness and considerable agraphia.

But complete destruction of Broca's convolution does not always produce this result. In the *Lancet*, May 22, 1897, I have recorded a case in which complete and acute destruction of Broca's convolution produced complete motor-vocal aphasia, but no word-blindness and comparatively little agraphia.[1] In that case, I presume that the connection between the motor-vocal speech centre (Broca's convolution) and the visual speech centre (the left angular gyrus) had become, comparatively speaking, a loose one; in other words, in that case the action of the visual speech centre had become so differentiated, so highly specialised, that it was in the habit of acting much more independently of the motor-vocal speech centre than is often (? usually) the case.

And here I may note a very remarkable coincidence, viz., that both of these patients, who were not related in any way, had the same surname, which for obvious reasons I suppress, but which I may term "Brown."

Another point of interest between these two cases, in both of which the motor-vocal speech centre (Broca's convolution) was completely and acutely destroyed, was the condition of writing.

[1] In this case there were two lesions. One, which occurred on November 21, 1892, was attended with complete motor-vocal aphasia, no word-blindness and comparatively little agraphia; the second, which occurred four months afterwards, was followed by some word-blindness and marked agraphia. The case in some respects forms a remarkable contrast to the one which I am at present recording.

In the present case, in which there was little or no motor-vocal aphasia (unless the inability to name objects and persons was a motor defect), but a certain though slight degree of word-blindness, there was considerable agraphia and paragraphia. In the former case, in which there was complete motor-vocal aphasia, there was no word-blindness and very little agraphia. The absence of any marked agraphia was forcibly illustrated by the fact that the patient, who was unable to answer questions in vocal speech, carried a notebook about with him for the purpose of answering questions and carrying on a conversation in writing.

The former case (recorded in the *Lancet*, May 22nd, 1897) seemed totally opposed to the view which the great majority of authorities on aphasia hold, that destruction of Broca's convolution (the motor-vocal speech centre) necessarily produces agraphia, and that, as Gairdner long ago argued, the degree of agraphia is usually proportionate to the degree of motor-vocal aphasia. It seems to support the view advocated by Bastian, which I had also independently arrived at, viz., that the nervous impulses which set the motor writing centre into action do not necessarily (and presumably, therefore, do not usually) pass through the motor-vocal speech centre in order to reach the writing centre, and that destruction of the motor-vocal speech centre does not necessarily (and presumably, therefore, does not usually) give rise to agraphia, although I, of course, admit the clinical fact that motor aphasia due to destruction of Broca's convolution is usually associated with agraphia and that the agraphia is usually (roughly speaking) proportionate in degree to the motor aphasia.

The present case seems, at first sight, to support the usually accepted theory. It shows that a lesion (complete destruction) of Broca's convolution (the motor-vocal speech centre) and of the anterior end of the left island of Reil which does not produce motor-vocal aphasia (unless the inability to name objects and persons was a motor defect) may produce considerable agraphia and paragraphia.

But while this must be admitted, it does not necessarily follow that the agraphia in this case was due to the interruption of nervous impulses passing through the motor-speech centre on their way to the writing centre; in other words, it does not necessarily prove that the nervous impulses which put the motor writing-centre into action *pass through* the motor-vocal speech centre.

The agraphia which was present in this case and which, it must be allowed, resulted either from the destructive lesion of Broca's convolution or from the destruction of the anterior end of the left island of Reil, for the lesion was sharply limited to these parts, may have been produced in other ways. Two theories may be advanced to account for the agraphia, viz. :—

(1) That the lesion of the anterior end of the island of Reil partially interrupted the conduction through the fibres which convey writing-producing impulses, as they pass from the visual speech centre to the motor centre for writing (which is, I think, probably situated in the ordinary psycho-motor centre for the movements of the right hand, but which Exner and some authorities think is perhaps situated in the posterior end of the second left frontal convolution).

(2) That the agraphia was an *indirect* result of the destruction of Broca's convolution, and was due to derangement of the action of the visual speech centre. In other words, the agraphia may have been due to the fact that destruction of Broca's convolution produced temporary functional disturbance in the action of the visual speech centre, and this functional disturbance in the action of the visual speech centre (in which, every one admits, writing impulses arise,[1] whether they afterwards pass through Broca's convolution or not, in order to reach the writing centre) was the cause of the agraphia. But, if this theory were correct, one would have expected the word-blindness to have been at least as great as the agraphia; and this was not the case.

[1] In order not to complicate the argument too much, I omit any reference to the auditory speech centre in which the nervous impulses concerned in the production of writing probably originate.

I merely suggest this as a possible mode of production of the agraphia *in this particular case*. I do not mean, of course, to suggest that it is the usual mode of production of agraphia, in cases of motor aphasia due to a lesion involving the motor-vocal speech centre (Broca's convolution). I believe, with Bastian, that in most cases of motor aphasia in which there is agraphia, the agraphia is probably produced in one or other of the following ways :—(*a*) by the lesion which destroys Broca's convolution (and so produces motor-vocal aphasia), also implicating and destroying the motor writing centre (ordinary motor centre for the fingers and thumb, wrist, &c., of the right hand) ; or (*b*) destroying or interrupting the conduction through the fibres which conduct impulses to that centre (the motor writing centre) from the visual speech centre (*i.e.*, the fibres which *directly* connect the visual speech centre with the motor centre for writing).

The production of the agraphia in either of these ways is compatible with the theory that the nervous impulses which put the writing centre into action pass directly from the visual speech centre to the motor centre for writing ; in other words, that they do not necessarily (and presumably, therefore, do not usually) pass through the motor-vocal speech centre on their way from the visual speech centre to the motor centre for writing, as most authorities suppose.

That in this case, *after the occurrence of the lesion*, the nervous impulses which were concerned in the act of writing did not pass through the left motor-vocal speech centre (Broca's convolution) in order to reach the writing centre is conclusively proved by the fact that the motor-vocal speech centre (Broca's convolution) was completely destroyed.

But it is, of course, quite possible that such impulses may have passed to the motor centre for writing through the *right* motor-vocal speech centre, (which I maintain took up and carried on the motor-speech function) after Broca's convolution was destroyed.[1]

[1] I should here note that the patient's power of writing with the left hand was unfortunately not tested.

(3) *The Inability to name Persons and Objects.*

One of the most interesting points in the case (though it is altogether subordinate in importance to the central fact of the case, viz., that acute and complete destruction of the motor-vocal speech centre—Broca's convolution—merely produced a very temporary and slight degree of motor-vocal aphasia) is the fact that the patient was unable to name objects and persons. For some days after the onset of the attack he could not name himself, and throughout the subsequent stages of the case he was unable to name his wife or his doctor. This inability to name persons and objects was such a striking feature of the case that at the time of our consultation Dr. Menzies said to me: "If there is a 'naming' centre, that is the seat of the lesion in this case."

As is well known to all who have given any attention to the complicated subject of aphasia, Broadbent has propounded the view, which is supported by Mills, that there exists, on the sensory side of the cerebral nerve apparatus, a special *naming* centre, and, on the motor side, a corresponding *propositioning* centre. Personally, I see no reason to adopt this view and to suppose that a "naming centre," other than the auditory speech centre, exists.

When this case first came under my notice, I was disposed to think that it was totally opposed to Broadbent's view. But more careful consideration has shown me that such is not necessarily the case; and although I see no reason whatever for believing in the existence of a "naming" centre (as distinct from the ordinary auditory speech centre), I do not now maintain, as I was at one time disposed to do, that the facts of this case entirely disprove the existence of a "naming" centre. For if a special "naming" centre exists, as Broadbent and Mills suppose, on the sensory side of the cerebral nerve mechanism, it is obvious that the nervous impulses which are generated in that centre must necessarily pass out through the motor-vocal speech centre in order that they may be vocalised; and consequently that an inability to name objects and persons may be due either to (*a*) destruc-

tion of the "naming" centre; (*b*) destruction of the nerve fibres which pass from that "naming" centre to the motor-vocal speech centre; or (*c*) destruction of the motor-vocal speech centre by which these impulses are emitted.

Now, in this case, in which the motor-vocal speech centre (Broca's convolution) was acutely and completely destroyed, there was, after the immediate effects of the lesion passed off (five days after the onset of the lesion), no motor-vocal aphasia, except the inability to name objects and persons (if that was, in this case, a motor defect).

But there is one difficulty in the way of accepting the view that the inability to name objects and persons was a motor defect, viz., that the motor-vocal speech centre was completely destroyed and yet there was no motor aphasia, except for the emission of names (if the inability to name objects and persons was a motor defect). In other words, if inability to name persons and objects was a motor defect and was due to the destruction of Broca's convolution, the destruction of that centre, since it was complete, should have produced inability to use *all* words (words other than names), *i.e.*, should have produced complete motor aphasia. Or, to put it another way, if the absence of motor aphasia (other than the inability to name objects and persons) was due (as I maintain) to the right motor-vocal speech centre taking up and carrying on the function of the motor-vocal speech centre which was destroyed (Broca's convolution), why was the right motor-vocal speech centre not also able to emit the names of objects and persons?

It may of course be said that the naming of objects and persons is a more elaborate (more highly specialised and less automatic) mental speech-process than the production of ordinary words (other than names); and that it is consequently conceivable that the right motor-vocal speech centre may be sufficiently educated and active to emit ordinary words (other than names), but not sufficiently educated and active to emit names. But such a supposition seems to me to be extremely difficult to accept, for I see no reason whatever why a motor speech-centre which can emit the impulses (*i.e.*, send to the ordinary psycho-motor centre

for the lips, tongue, larynx, etc., the nervous impulses) concerned in the production of ordinary words should not be able to emit the impulses concerned in the production of the names of objects and persons. Further, as everyone knows, in many cases of motor-aphasia in which the motor aphasia is complete, the patient still retains the power of spontaneously uttering his own name and his wife's name—the names which by long practice have become, as it were, automatic. In cases of complete motor aphasia due to destruction of Broca's convolution, the few words which the patient retains (such as his own name and certain emotional and automatic utterances) are probably, I think, emitted by the right motor-vocal speech centre. But in this particular case the patient, when I saw him, could not name himself, and during the subsequent progress after I saw him was unable to spontaneously name his wife or his doctor. Though there was practically no other motor aphasic defect, he was unable to utter the very words (such as his own name) which are usually retained by patients affected with motor aphasia. It is consequently difficult, I think, to accept the view that in the case which I am at present recording the inability to name objects and persons was a motor defect, more especially since, as I have remarked in recording the case, the patient could *repeat* his own name, his wife's name and his doctor's name when they were mentioned to him, although he could not spontaneously emit them. The fact that the patient could repeat names which he could not spontaneously utter seems to show that the inability to name objects and persons was due to a defect on the sensory and not on the motor side of the speech mechanism.

Whether it is possible that a name or a word, which cannot be *spontaneously* emitted in consequence of complete destruction of Broca's convolution, can be *repeated* and emitted by the ordinary psycho-motor centre for the lower face, lips, tongue, larynx (*per* the lower end of the left ascending frontal convolution, *i.e.*, without having previously passed through the motor-vocal speech centre—Broca's convolution), or not, I do not venture to give an opinion. But

this occurs to me as a possibility; the point deserves consideration and should be kept in view (with the object of having this possibility proved or disproved) in dealing with cases of motor aphasia.[1]

The most probable explanation of the inability to name objects and persons is, it seems to me, to suppose that the action of that part of the auditory speech-centre in which names (highly specialised parts of speech) are stored and recalled (or, as Broadbent and Mills would say, of a special centre other than the auditory speech-centre—the "naming" centre, as they term it) was deranged by the lesion in the motor-vocal speech centre, just as I have presumed that the action of the visual speech centre was temporarily deranged by that lesion. In other words, that the inability to name objects and persons was produced in the same *indirect* way in which I suppose the slight word-blindness was produced, and in which the more marked agraphia was, perhaps, produced. But here again there is a difficulty, for when I saw the patient he could write his own name, although he could not say it.

I see no reason to suppose that the inability to name objects and persons was due to a lesion of the conducting tract, which is supposed by some authorities to pass from a special centre, the "naming" centre, to the motor-vocal speech centre.

This seems to me sufficiently proved by the facts:—(*a*) the lesion was practically speaking confined to Broca's convolution and to the anterior end of the left island of Reil; (*b*) these parts were completely destroyed by the lesion; (*c*) the ordinary "way out" for names, and words other than names, was blocked in the emissive centre (Broca's convolution); it is therefore quite unnecessary and superfluous to suppose that a lesion existed in the tracts proceeding to that centre; (*d*) there was no evidence of a lesion in any tract proceeding from the supposed "naming"

[1] Since writing the above, I see that Joseph Collins makes a similar suggestion with regard to "echo" speech. He says:—"Whether such reflex or echo-like words cannot be produced by the action of sensory impulses directly on the central executive motor speech mechanism, I have often debated."—"The Faculty of Speech," by Professor Joseph Collins, page 170.

centre (or from the auditory speech-centre, in which I believe the so-called naming centre is situated and of which I believe it to be a component part) to the motor-vocal speech centre (unless that tract passes immediately beneath the anterior end of the island of Reil); and (*e*) that the emissive tract for names was obviously open (unblocked), for the patient could repeat a name (such as his own name or his doctor's name) which he could not spontaneously utter.

Since the left motor-vocal speech centre (Broca's convolution) was in this case completely destroyed, it seems to me that names as well as other words must in all probability (when pronounced in this way, *i.e.*, repeated) have been emitted through the opposite *right* motor-vocal speech centre.

From these statements it will be apparent that I have very great difficulty in giving a satisfactory explanation of the patient's inability to name persons and objects.

Conclusion.

It may of course be argued that the motor-vocal speech centre is not necessarily limited to the posterior end of the lower (third) left frontal convolution (Broca's convolution), or indeed to the adjacent parts of the lower (third) left frontal convolution (for in this case the softening involved not only the "foot," but also the "cap" and the "orbital portion" of the third left frontal convolution), but that it may, in exceptional cases, of which this was an example, be situated in the adjacent parts of the left hemisphere (the lower end of the left ascending frontal convolution, &c.). In other words, that the modern view, that the lower end of the left ascending frontal convolution is merely the ordinary psycho-motor centre for the lower face, tongue, larynx, &c., and that it is not, properly speaking, a part of the motor-vocal speech centre, is erroneous.

If this be granted, it must of course be allowed that it is unnecessary to suppose that the action of the left motor-vocal speech centre was in this case taken up and carried on by the corresponding centre in the right hemisphere.

But such a supposition seems to me, in the face of our modern knowledge (and especially in the light of Elder's case to which I have previously referred), altogether less likely than the theory which I have advanced. Further, Barlow's case *conclusively proves* that the right motor speech-centre *can* (after a short time) take up and perfectly carry on the motor-vocal speech function; and other arguments and facts can be adduced to show that the speech centres and speech faculties are, in some degree at all events, bilaterally represented in the cerebral hemispheres (see my lectures in the *Edinburgh Medical Journal*, and Bastian's "Lumleian Lectures on Aphasia").

In short, after the most careful and impartial consideration of the whole question, the most reasonable explanation of the absence of motor-vocal aphasia in this case, in which Broca's convolution was completely and acutely destroyed, seems to me to be that the function of the left motor-vocal speech centre was taken up and carried on by the motor-vocal speech centre in the right hemisphere of the brain.

DESCRIPTION OF FIGURES.

FIG. 1.

The left hemisphere of the brain in the case of aphasia described in the text, showing the exact extent of the lesion.

The degenerated areas are shaded in black.

The figures point to the parts (convolutions) which are correspondingly numbered in the horizontal sections (see figs. 3, 4, 5 and 6).

The letters RO. are placed immediately above the upper end of the fissure of Rolando.

The letter S. is placed at the commencement of the fissure of Sylvius, at the point at which the horizontal and ascending limbs (marked by white lines) are given off.

FIG. 2.

The left hemisphere of the brain in the case of aphasia described in the text, showing the exact position of the series of longitudinal (horizontal) sections which were made through it.

The figures 1 to 6 point to the different sections, the upper surfaces of which were photographed, and are reproduced in figs. 3, 4, 5 and 6. The under surface of section 5 is reproduced in fig. 7.

FIG. 3.

Longitudinal (horizontal) section of the brain in the case of aphasia described in the text (the upper surface of section 2 in fig. 2.)

A small area of softening, which corresponds to the uppermost point of Broca's convolution, is shaded black.

1 = Posterior end of the second left frontal convolution.
2 = The uppermost point of Broca's convolution, softened and degenerated.
3 = The left ascending frontal convolution.
4 = The left ascending parietal convolution.
2 R. F. = The second right frontal convolution.
R. A. F. = The right ascending frontal convolution.
R. A. P. = The right ascending parietal convolution.

FIG. 4.

Longitudinal (horizontal) section of the brain in the case of aphasia described in the text (the upper surface of section 3 in fig. 2). The area of softening, which is shaded in black, involves Broca's convolution and the anterior end of the left island of Reil. These parts were completely destroyed.

5 = First left frontal convolution.
6 = Second left frontal convolution.
7 = Anterior end of the "cap" of Broca's convolution.
8 = The "cap" of Broca's convolution.
9 = The "foot" and "ascending portion" of Broca's convolution.
10 = Left ascending frontal convolution.
11, 11 = Left ascending parietal convolution.
12 = The anterior end of the left supra-marginal convolution.

The letter R. is placed on the white matter just beneath the anterior end of the left island of Reil.

R. 3 F., R. 3 F. = Right third frontal convolution.
R. A. F. = Right ascending frontal convolution.
R. A. P. = Right ascending parietal convolution.

FIG. 5.

Longitudinal (horizontal) section through the brain in the case of aphasia described in the text (the upper surface of section 4 in fig. 2).

A large area of softening and destruction, which is shaded in black, is seen in the position of the posterior and middle parts of the third left frontal convolution, and of the anterior end of the left island of Reil.

13 & 14 = Second left frontal convolution.
15 = Third left frontal convolution; a small gyrus which was practically unaffected.
16 = "Cap" of Broca's convolution.
17 = "Foot" and "ascending portion" of Broca's convolution.
18 = Left ascending frontal convolution.
19 = Left ascending parietal convolution.

R. 3 F., *R. 3 F.* = Right third frontal convolution.
R. A. F. = Right ascending frontal convolution.
R. 1 T. S. = Right first temporo-sphenoidal convolution.

Note.—The section of the right hemisphere is at a slightly lower level than that of the left.

FIG. 6.

Longitudinal (horizontal) section through the brain in the case of aphasia described in the text (the upper surface of section 5 in fig. 2).

The area of softening and destruction, which is shaded in black, involves the anterior (orbital) portion of the lower (third) left frontal convolution.

20 & 21 = Second left frontal convolution.
22 = Orbital part of the third left frontal convolution.
23 = Anterior end of the first left temporo-sphenoidal convolution.

FIG. 7.

The inferior surface of the frontal lobes of the brain in the case of aphasia described in the text (the under-surface of section 5 in fig. 2).

The portion of the lower (third) left frontal convolution which is shaded black, and to which the figure 24 points, was softened and degenerated.