ON APHASIA DUE TO ATROPHY OF THE CEREBRAL CONVOLUTIONS.

BY DR. G. MINGAZZINI.

Professor of Neuropathology at the Royal University of Rome.

SCARCELY ten years have elapsed since attention was drawn, first by Pick and subsequently by Dejerine and myself, to certain cases of aphasia which, as was proved by autopsy, were not caused by tumours or other diseases of the speech-zone, but were due to more or less severe atrophy of the respective convolutions.

Although the preceding observations have since been partially confirmed by other writers, nevertheless the literature dealing with this subject is still very scanty, and often consists of accounts of incomplete researches. For this reason, I have decided to publish the details of the following case, which I studied both during life and *post mortem* at the lunatic asylum in Rome.

Pio Marini, aged 59, a gilder. The patient, from his condition, is unable to give any information. He has never suffered from any infectious disease, or from syphilis, and has been moderate in the use of wine. About two years previously (1907), the first symptoms of the disease began to show themselves rapidly. These consisted in a tendency to impulsive actions, as well as in a difficulty in speaking and in understanding what was said. Those around the patient noticed that he gave the most curious names to the commonest objects (paraphasia); and that when his intimates did not understand him, and persevered in trying to ascertain his wishes, Marini could not comprehend their questions and broke out into threats and accesses of rage. The symptoms continued slowly and gradually to increase, without any signs of a stroke.

This state of affairs obliged the patient's relatives to send him to the lunatic asylum on March 1, 1909.

The patient's condition, March 10, 1909.—The eye movements, as far as they can be tested, are normal. In showing the teeth, the right upper lip becomes tremulous; the tongue can be fully protruded, and is slightly turned towards the left. The upper and lower limbs are able to perform all the usual active and passive movements, but, in producing the latter, the amount of resistance encountered is somewhat greater than the normal. The tendons and periosteal

reflexes of the upper limbs, and especially those of the lower limbs (patellar and Achilles tendons) are exaggerated. There is sometimes very evident foot clonus of the left side; the plantar reflexes give a flexor response. The abdominal reflexes are greatly exaggerated. A general hypalgesia is present. Speedy dermographia is evident.

Condition, March 12, 1909.—The patient remains quiet. At first he used to rave somewhat, and often called for a certain Don Antonio, who, according to him, ought to take him home; then he became calm, and showed himself indifferent. Echolalia is often noticed.

Examination of the patient's speech.—When questioned, the patient replies correctly only when asked his christian name and his surname. If other simple questions are put to him, he either repeats them (echolalia, or perseveration), or says: "Don Antonio"; "I do not know." He carries out verbal commands, provided they involve more or less common movements, with exactitude and rapidity; when, however, it is a question of obeying orders which are a little more complicated, he always replies "Don Antonio," and does not execute (does not understand) the order. He repeats words exactly. On being shown objects, and asked to name them, he succeeds at first (spoon, glass) and then replies as follows:—

Q.		A.
What is this called?	(a bottle).	Don Antonio.
,, ,,	(an inkstand).	Marini Pio.
,, ,,	(a pen).	Marini Pio, gilder; then, *I do not know;* then, *a paint brush.*
,, ,,	(nose).	Nose (after touching it).
,, ,,	(ear).	Ear (*idem*).
,, ,,	(lips).	Chin.
,, ,,	(a watch).	I do not know.

When invited to read, he often repeats: "Marini Pio," or "Don Antonio." Sometimes, on beginning to read, the patient reads two or three words correctly, and afterwards reads some words in a quite mutilated manner, and then stops, puts down the book, and tries to induce the examiner to allow him to do something else.

Written to dictation:—

Marini Pio.	Marini Pio.
Manicomio.	The patient always writes Marini Pio.
Roma.	
Roma.	

Copied.—The sentence to be copied ran as follows: "Se avete dei dubbi sulla campagna elettorale del 'Messaggero' (if you have any doubts as to the electoral campaign of the 'Messaggero')." He wrote "Marini Pio."

Condition, August 5, 1909:—

Examination of Speech. Q.	*Perception of Commands.* A.
What is your name?	Pio Marini.
Shut your eyes.	He obeys.
Open your mouth.	The patient does not open his mouth, but closes his eyes.
Stand up.	He obeys.
Sit down.	He obeys.
Raise your right arm.	The patient gets up and raises his left arm.
Get up, and go to the door.	The patient gets up, but does not go to the door.
Take the inkstand.	The patient does not obey the order.

The patient thus understands part only of the commands. He often repeats the last words of the orders without understanding them (echolalia automatica), e.g., Stand up, he repeats: "Stand up" (and does not do so), Go to the window; he says: "Go to the window" (but does not obey). At other times he persists in using the same words; thus when shown different objects such as a pencil, a glass, spectacles, and asked their names, he always says: "Don Antonio."

Condition, February 12, 1910:—

Examination in the Comprehension of Speech. Q.	*Perception of Commands.* A.
Shut your eyes.	He obeys.
Put out your tongue.	He obeys.
Stand up.	The patient does not obey, but says: "Don Antonio."
Give me your hand.	The patient raises both hands, but does not obey.
Shut the door.	The patient shuts his eyes.
Touch your ear.	The patient does not obey, and exclaims "Don Antonio."
Put on your cap.	He repeats: "Put on your cap," but does not do so.

Repetition of Words.

Q.	A.
Roma.	Roma.
Papa.	Papa.
Angelo (angel).	Angelo.
Repubblica (republic).	Repubblica.

Q.	A.
Uccello (bird).	Uccello.
Napoli (Naples).	Napoli.
Il mare è immenso (the sea is vast).	Immenso.
Il cielo è bello (the sky is beautiful).	The patient first repeats "il cielo," and, on the command being repeated, replies: "è bello."
La campagna è bella (the country is beautiful).	E bella.
Repubblica popolare (popular republic)	Popolare.
La casa è grande (the house is large).	This the patient does not repeat.
Circoscrizione (circumscription).	The patient repeats the word with some clear dysarthric disturbances.
Circondariale.	Circondariale.
Artiglieria (artillery).	Artiglieria? I do not know the word.
Circoncisione (circumcision).	He does not repeat the word.

When several objects are placed before the patient, and he is requested to carry out an order, the results are as follows:—

Q.	A.
Take the watch.	The patient obeys.
Take two soldi (two five centesimi pieces).	He does not do so.
Take the keys.	The patient does not obey, but exclaims "Don Antonio."
Take the scissors.	Idem.
Take the book.	Idem.
Take the inkstand.	Idem.
Take the bottle.	Idem.
Take the hat.	Idem.
Take the handkerchief.	He takes a ten-centisimi piece.

When different objects are shown to the patient, and he is asked to name them separately, the results are as follows:—

Object shown.	A.
A watch.	A watch.
A pair of scissors.	A pair of spectacles.
On making him take them in his hand.	I do not know.
A soldo (making him feel it).	I do not know. Don Antonio.
A pair of spectacles.	Don Antonio.
A bottle.	Don Antonio

The patient thus showed signs of partial sensory aphasia, amnesia verborum, verbal paraphasia, paralexia, and almost complete agraphia.

Condition, October 18, 1910.—The patient is always taciturn; he spends the whole day in walking aimlessly about in a state of complete apathy, and takes no interest in anything around him. He cannot find his bed, the water-closet, does not know the hours of the meals, and at meal-time has to be put in his right place. He often eats with his hands, but when convenient he also uses a spoon. If a plate of soup is placed before him, the patient begins to eat with his hands, then he takes up his spoon properly, but nevertheless he eats now with his spoon, and now with his hands. He is not able to dress or undress himself properly; in dressing himself he puts on his trousers, and also his vest, and draws his shirt over the latter and buttons them correctly; then he takes his socks, and goes in this condition out of the dormitory to the first door he comes across. Sometimes when dressing himself he tries instead to take off his vest.

His spontaneous speech is reduced to the monotonous repetition of the words, "Don Antonio." When asked to read the sentence, "Senza pieta" (without pity), he reads "Aprile 30." When the request is repeated, he reads: "Piri piri, pi." When asked to read "Concorso ippico," he reads "Don Antonio." The patient is unable to read any easy written sentence, however short. He is also incapable of reading or recognizing single letters and numbers, or even a single figure. When asked to write out of his head, or from dictation, or to copy, he merely makes the following hieroglyphic, "m m."

Comprehension of Questions.

Q.	A.
How old are you?	Don Antonio.
What is your name?	No reply.
To what country do you belong?	No reply.
What is your father's name?	No reply.

Repetition: His spontaneous speech is reduced to pronouncing the words, "Don Antonio"; he retains the capacity of repeating these words, although with decided dysarthric troubles; word-blindness and agraphia are complete, and word deafness is almost complete.

Voluntary movements of the face, the tongue and limbs, together with the tendon reflexes, were in the same condition as at the time of the preceding examinations.

The patient has a good perception of light when it comes from the right or left, but shows a certain fear if the light approaches too near his eyes.

Condition, October 23, 1910.—In the afternoon the patient suddenly began to run of his own accord, and fell down, producing abrasions of the skin on the right side of his face. As a rule, he only walks aimlessly about the garden, he does not recognize the refectory, the water-closet, or the dormitory, and always needs assistance in finding his own bed. In dressing, he is always

able to put on his vest and shirt properly, but is unable to get on his coat, unless the nurse puts it on his shoulders, when he manages quite well. Often, after putting on one shoe and sock, he goes out of the room and begins to walk about, carrying the other sock in his hand. As a rule, he does not

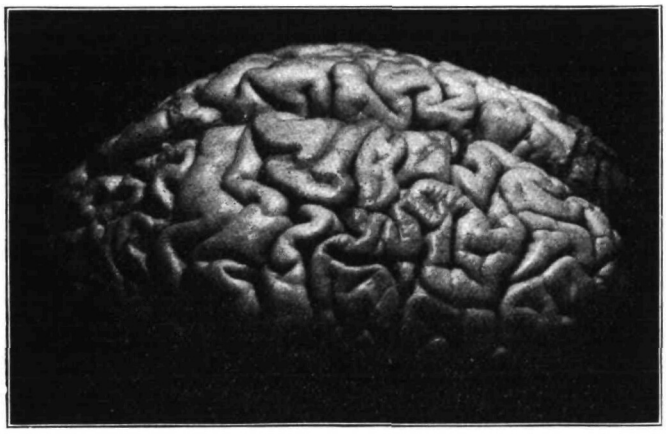

FIG. 1.—Photograph of the brain seen obliquely from above. Note on the right, the contrast between the enormous atrophy of the convolutions of the prefrontal lobe, and the normal volume of the rolandic convolutions.

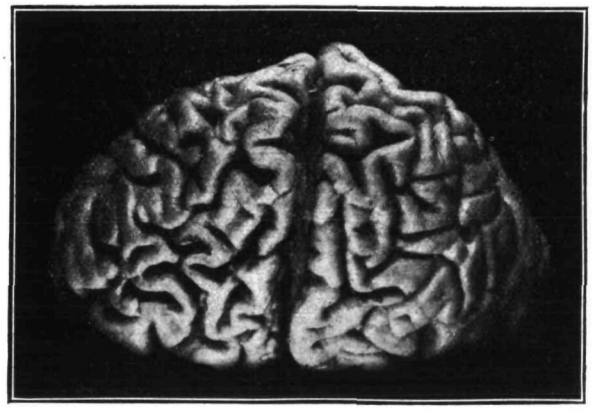

FIG. 2.—Photograph of the prefrontal lobes of the brain, seen from above. Note the enormous extent of the atrophy of the corresponding convolutions.

succeed in putting on both socks without the assistance of the nurse. Almost every morning, while sitting on his bed, he draws his socks over his hands, and then puts his hands into his shoes, then, however, following the suggestions and indications of the nurse, he finishes by putting on his socks and shoes properly. He eats, for the most part, with his hands, but when necessary he

manages his spoon well. At night he undresses himself properly and folds up his clothes neatly.

The late appearance and slow progress of the symptoms of aphasia, the absence of strokes or any focal symptoms, together with the ever-increasing

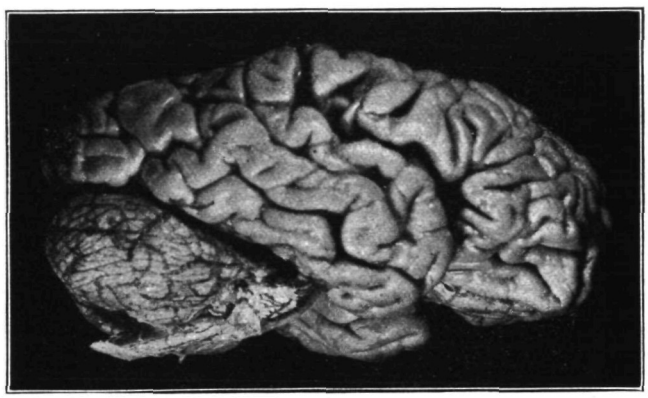

FIG. 3.—Photograph of the right cerebral hemisphere of the brain of the same patient. Note the conspicuous atrophy of the pars opercularis of F^3 and of the middle and posterior portion of T^1.

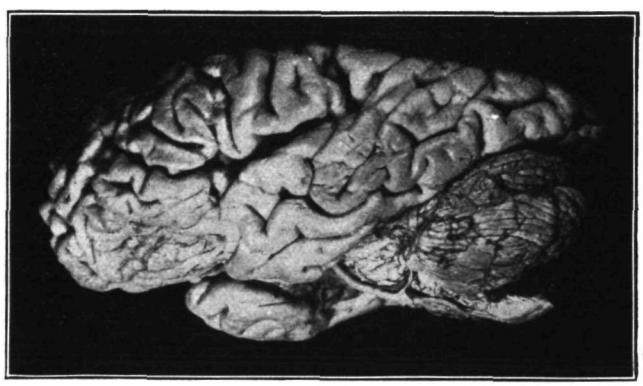

FIG. 4.—Photograph of the left cerebral hemisphere of the same patient. Note the serious atrophy of the pars opercularis of F^3 and of the middle portion of T^1.

dementia, led me to conclude that the patient was developing progressive atrophy of the cerebral convolutions, to which must be attributed the increasing speech defects. This diagnosis was confirmed by his death, which occurred on December 15, 1910.

Autopsy.—There is nothing amiss with the dura mater. The pia mater is normal and easily removed from the cerebral convolutions.

In the left hemisphere the frontal sagittal convolutions, especially the first and

second, the pars orbitalis and triangularis of the third frontal convolution have noticeably decreased in volume (fig. 1), and scarcely attain half of their normal size; they seem of an almost fibrous consistency to the touch. The median and posterior parts of T^1 and T^2, and especially the gyrus angularis, are somewhat diminished (figs. 1 and 4). The corresponding sulci are enormously enlarged. In the right hemisphere the præ-frontal (fig. 2) and temporal convolutions are

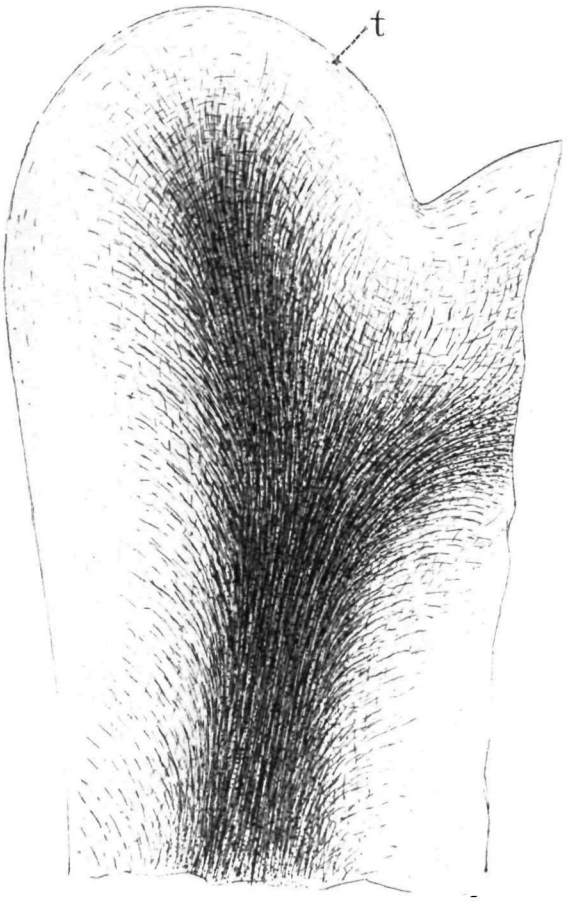

FIG. 5.—Section of the cortex of the left T^1. Stained with Pal's hæmatoxylin; *t*, tangential fibres. A very slight rarefaction of the fibres of the medullary axis is seen, the transverse fibres of the supraradial plexus are here and there slightly rarefied.

much reduced, although to a considerably less degree than is the case with those on the left (figs. 2 and 3). The median portion of T^1 is almost as much reduced as that of the corresponding convolution on the left side. In the zones where the convolutions are atrophied, the pia mater is raised here and there into little cysts containing a clear, serous liquid.

For the purpose of studying the condition of the cells, and the nerve-fibres of the cortex of the atrophied convolutions, and especially of those belonging to the acoustic and motor word spheres, I examined at measured points on the right and left, the pars opercularis of F^3 and the passage zone of the latter with the lower extremity of gyrus præcentralis and the median point of T^1. All these were stained either with Weigert-Pal's hæmatoxylin, or with hæmatoxylin-eosin.

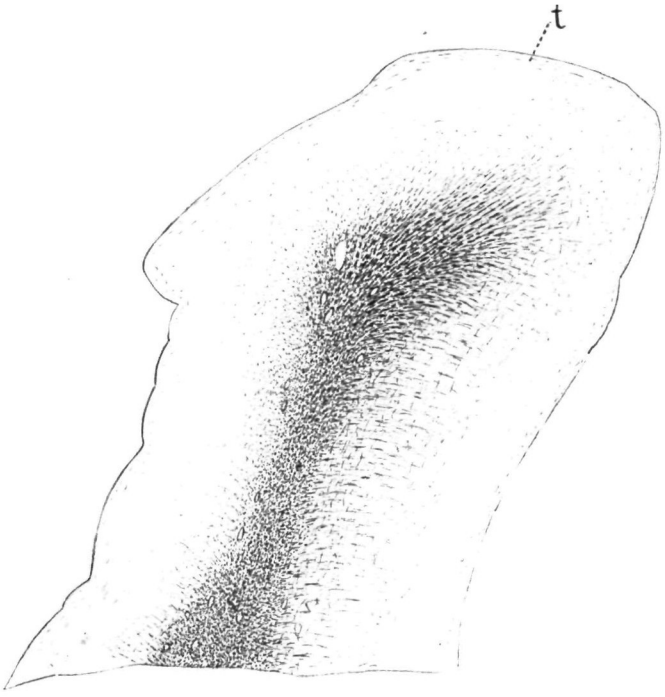

FIG. 6.—Section of the cortex of the middle portion of the pars opercularis of the left F^3 (staining as above). Note the almost complete disappearance of the tangential fibres (*t*) and of most of the fibres taking part in the formation of the infra- and supraradial plexuses. In one part of the section (on the left of the figure) the disappearance of the medullated fibres is so nearly complete, that scarcely any traces of them are visible. The fibres of the medullary axis are so rarefied and degenerate that even in the most successful preparations they are conspicuous from their very pale colour.

In sections cut at the level of the zone of passage between the pars opercularis of the left F^3 and the lower extremity of the gyrus præcentralis (fig. 11), the nerve-fibres composing the medullary axis are very scarce, many are very thin and dilated from time to time in such a manner as to have an actual moniliform structure. The radial bundles taking part in the formation of the infra- and supraradial plexuses are attenuated, and the nerve-fibres accompanying them are broken and dilated; some of the transverse fibres have disappeared, almost all that remain are broken and dilated; very rarely the tangential fibres are replaced by a few swollen fibrils.

In the sections of the same zone stained with hæmatoxylin-eosin, the nerve-cells of all the strata show, to a greater extent than in the other convolutions examined, the alterations already described, the disappearance of a considerable number of these elements being especially noticeable (fig. 12, and fig. 9, *a*, *b*, *c*, *d*).

FIG. 7.—Section of cortex of the pars opercul of the left F^3 (stained with hæmatoxylin-eosin. Enlargement objective 4, ocular 2 Leitz); p, pia mater. A good number of the nerve-cells are present, and to much larger extent than in T^1; further, also here, there are but few which have kept their pyramidal form.

In sections cut on the level of the median part of the left pars opercularis of F^3 (fig. 6), the alterations of the medullary nerve-fibres described in the preceding sections are much more conspicuous. The fibres constituting the medullary axis, especially towards the dorsal (apical) half, are almost all broken during their course in such a manner that they are replaced by a number of little

masses and fragments (of myelin) which are arranged without any order (fig. 8). In the same way, the transverse fibres, and also the radial fibres making up the two plexuses, have disappeared in much larger numbers. The tangential fibres are reduced to a few irregular fragments occurring here and there. Here we observe the same alterations of the nerve-cells as in T^1. The number of those preserved, however, is relatively larger (fig. 7, and fig. 9, *c*, *d*).

In frontal sections of the cortex of the median part of the left T^1 there are, in the medullary axis of the Pal preparations, numerous small bundles of well-preserved fibres (fig. 5), though here and there a fair number of degenerate medullated fibres occur, and small masses and drops of myelin. Also in the infra- and supraradial plexuses a good number of fibres have been preserved;

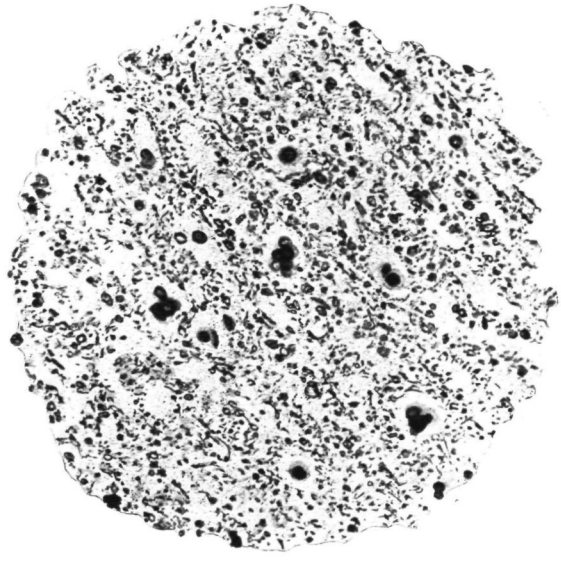

FIG. 8.—Segment of the medullary axis of the pars opercularis of the left F^1. Much enlarged. (Enlargement, objective 6·7, ocular 4 compens., Leitz.) In the place of the radial nerve-fibres running longitudinally are seen numerous little blocks of myelin, which are more or less dilated, and separated from one another by large clear spaces.

nearly all of these have, however, a moniliform structure owing to their having become dilated during their course. The tangential fibres are, to a great extent, preserved. In the hæmatoxylin-eosin preparations the following facts are observed; a considerable number of nerve-cells are seen to have disappeared, (fig. 10), and in almost a uniform manner in all the strata. In those which have still been preserved are clearly seen the nucleus with its network and nucleolus, in many the cytoplasm is almost completely filled with granules of a yellow pigment; in others the cytoplasm has in a great measure disappeared and only stains slightly (fig. 9, *e*, *f*). Thus, not a few of the pyramidal cells seem as if truncated, while in others the surrounding

protoplasm has completely disappeared, and it is not always easy to distinguish the nucleus of the nerve-cell from the nuclei of the glia. The fibres and the cells of the glia are normal in number, only on the periphery of the cortex of the said convolutions are the bundles similar in their thickness and shape to those described and figured by Franceschi. Thus there is not always a

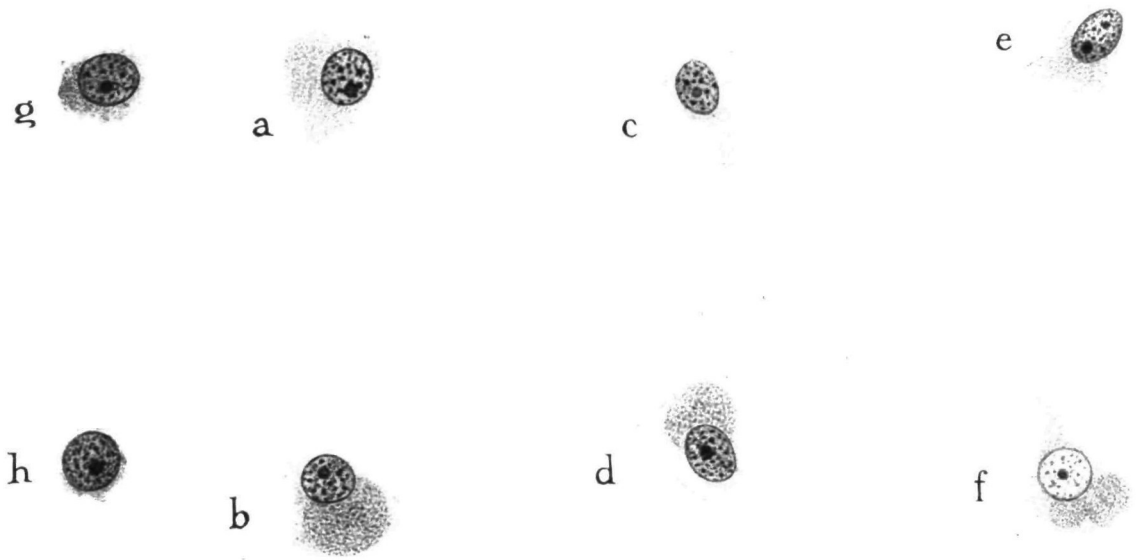

FIG. 9.—Types of nerve-cells of the cortex of the convolutions belonging to the motor word and to the auditory speech zones of the left side. Stained with hæmatoxylin eosin. (Enlargement, Leitz, ocular, compens. 4, objective 1½ min.). *a* and *b* are nerve-cells belonging to the cortex of the passage zone between the pars opercularis of left F^3 and the gyrus præcentralis, in which the cytoplasm has in part disappeared; *b*, nerve-cells (pyramidal), in which the cytoplasm is in a great measure invaded by granules of yellow pigment; *g* and *h* are nerve-cells belonging to the same convolution, the cytoplasm has almost completely disappeared; *c* and *d*, pyramidal nerve-cells from the cortex of the pars opercularis of the left F^3; in *c*, the cytoplasm is slightly coloured; in *d*, the yellow pigment occupies a great part of the latter: *e* and *f*, nerve-cells belonging to the cortex of the left T^1; in *e* the cytoplasm has in part disappeared, and the prolongations appear as if truncated; in *f* the cytoplasm is, to a large degree, invaded by the yellow pigment.

correspondence between the changes in the nerve-cells, and those which have taken place in the medullary fibres, and in fact, while in the pars opercularis of the left F^3, the changes in the fibres are very great, those in the nerve-cells are relatively little noticeable. On the other hand, in the passage zone between the left F^3 and the gyrus præcentralis, the nerve-cells present extremely noticeable alterations, while the medullary nerve-fibres, though certainly changed, are altered to a much slighter extent.

In the right T^1 there is no appreciable reduction of the nerve-fibres of the medullary axis, nor in those of the supra- and infraradial plexuses; the tangential fibres are also but little reduced, although fairly numerous varicose dilatations of the latter occur occasionally.

In sections at the level of the passage zone of the pars opercularis of the right F^3 to the adjacent part of the gyrus præcentralis, the medullary nerve-

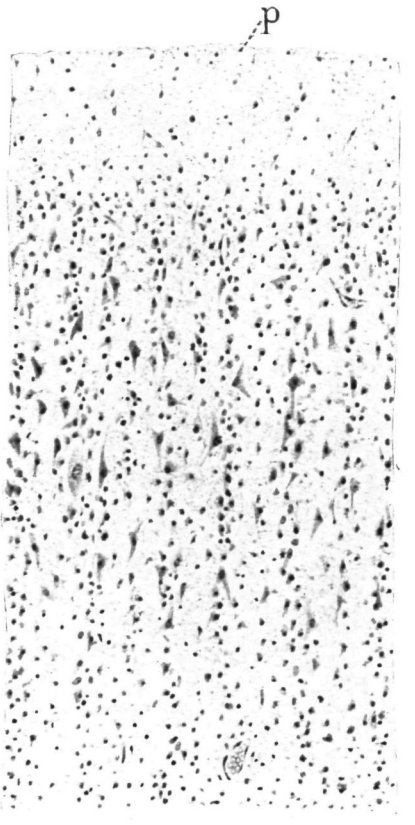

FIG. 10.—Section of the cortex of the left T^1 (stained with hæmatoxylin-eosin, enlargement objective 4, ocular 2, Leitz). Note the disappearance of many nerve-cells of the different strata of the cortex; the nerve-cells, especially the pyramidal ones, have to some extent lost their characteristic shape, owing to the loss of a considerable portion of the cytoplasm.

fibres appear to be well preserved, only some rare varicose swellings are seen in the tangential fibres, and in some of the vertical fibres of the infra- and supraradial plexuses.

In the pars opercularis of the right F^3 (fig. 13), the changes are greater than in the other two zones, many of the tangential fibres have disappeared, while in these, and also in the transverse fibres, numerous varicose swellings

occur. In the fibres of the medullary axis the disappearance of some fibres is noticeable.

In the nerve-cells of the right T^1 most of the cytoplasm has disappeared, and a few only of the cells are missing, although a larger number have disappeared in the sections of the passage zone from the right F^8 to the gyrus præcentralis, and still more in the pars opercularis of the right F^3.

FIG. 11.—Section of the cortex at the point of origin of the left F^3 from the lower extremity of gyrus præcentralis (stained with Pal's hæmatoxylin). The tangential fibres, and those forming the infra- and supraradial plexus, are much rarefied; the same is seen in the medullary axis, and especially in the basal portion of the latter.

SUMMARY.

In the period between 1907 and 1909, the patient showed disturbances in his speech, which gradually increased, and consisted in the use of unusual words in ordinary conversation (paraphasia and echolalia). He also experienced difficulty in understanding many questions. After

two years (March to August, 1909), the examination of the patient revealed true partial sensory aphasia (with automatic echolalia and perseveration), associated with paralexia and almost complete agraphia.

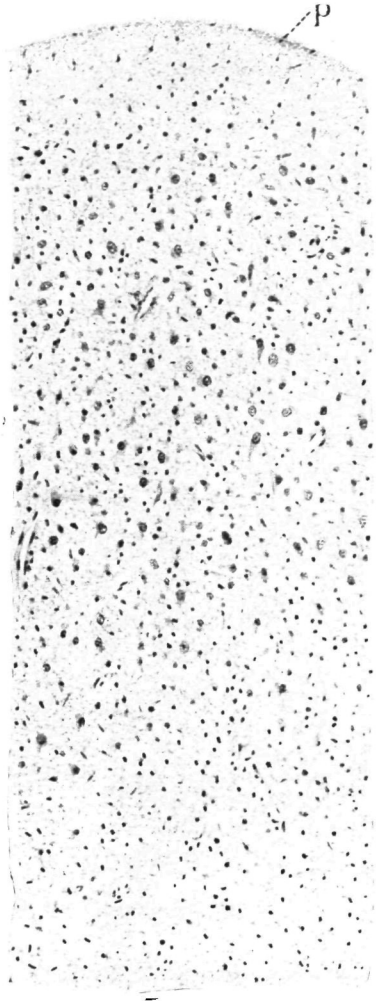

FIG. 12.—Section of cortex at the point of the passage from the pars opercularis of the left F^3 to the adjacent portion of the gyrus præcentralis. (Enlargement and staining the same as in figs. 7 and 10). The disappearance of the nerve-cells is much more noticeable than in the two sections 7 and 10, the pyramidal nerve-cells preserved being very few.

In February, 1910, the sensory aphasia had become almost complete and some dysarthria was noticed in his repetition of words. The understanding of words was extremely limited; further, a condition of advanced dementia showed itself, accompanied by symptoms of agnosia.

In October, 1910, the speech disturbances had become still more serious; the word deafness, the alexia and the agraphia were complete, while voluntary speech was almost entirely absent. Parallel with these

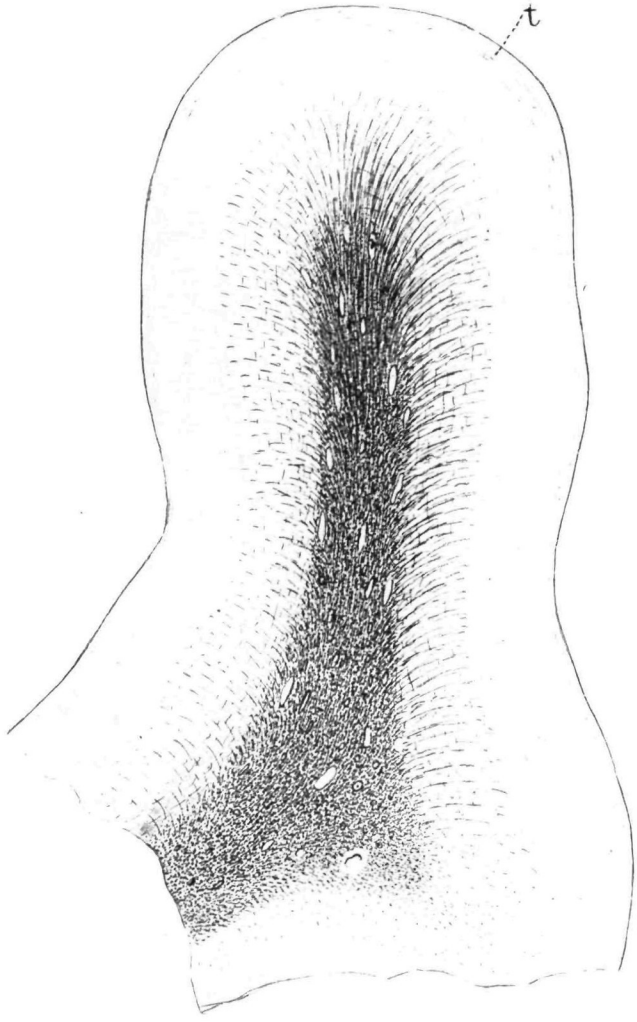

FIG. 13.—Section of the cortex of the pars opercularis of the right F^3 (stained with Pal's hæmatoxylin). The medullary fibres are well preserved, except for a slight rarefaction of the fibres of the supra- and infraradial plexus, and on one side only of the section.

progressive disturbances of speech a condition of mental weakness gradually developed, which at last became a true dementia and was attended with symptoms of agnostic apraxia, and psychical blindness.

Two months later (four years after the beginning of the disease) the patient died.

The *post-mortem* revealed severe atrophy of the prefrontal and temporal convolutions, the left side being the most affected, while the histological examination showed severe alterations in the nerve-cells and medullary fibres of the convolutions belonging to the Broca region (*in sensu lato*) and to T^1, these changes being greatest on the left side.

Critical Considerations.

Having thus described this new case, it will be well to compare its clinical and anatomical features with the results obtained by other writers. The complex of symptoms is almost uniform, and consists of a double set of phenomena, of which one belongs to speech and the other to the mental processes.

In the case described by Bischoff, the power of understanding words was extraordinarily impaired, but that of the repetition of words was quite intact. At the autopsy, atrophy of both the temporal lobes was revealed, the left one being the most affected.

Liepmann's patient was able to speak spontaneously, though intercalating clear signs of paraphasia. He understood questions, but he first repeated them in an interrogatory form, and then replied. As the disease progressed, the patient no longer understood the sense of the words, which he still repeated, though without giving any reply (automatic echolalia) and his command of spontaneous words became much reduced. Macroscopic investigation revealed atrophy of the frontal and temporal convolutions, and especially of the apex of the left temporal lobe; the smallest of all were the left T^1 and T^2. Upon the pia were vesicles containing a clear liquid.

In their first case of aphasia, P. Marie and Léri did not record any appreciable alterations in the horizontal sections of the right hemisphere, the lateral ventricle was found to be dilated, and especially the upper portion of the occipital pole. In the subependymal region it seemed to be surrounded by a sclerotic zone, and the white matter of the hemisphere appeared much atrophied. In the cortex of the brain there were no lesions whatever.

In the second case of the same writers, where the patient was affected with typical sensory aphasia, abnormal dilatation of the left occipital cornu was also found.

In a case of Ascher's, which was not typical, inasmuch as motor

aphasia, first temporary and afterwards permanent, was associated with paralytic dementia, there were found the usual lesions of this psychosis, further in the left T^1, the degeneration of the nerve-cells and of the white matter had reached a more advanced stage than in the rest of the brain.

In Shaw's case, the aphasic symptoms, consisting of word-blindness and word-deafness, suddenly manifested themselves; the patient was suddenly seized with paralysis of the right arm. The affection afterwards disappeared, but thenceforward the patient was afflicted with a slight incoherence of speech, and in addition showed signs of word-blindness and word-deafness. At the autopsy, atrophy was revealed of both the gyri angulares and of both the T^1 and T^2. This case is, however, exceptional, and it is very doubtful whether these disturbances of the speech were present before the occurrence of the stroke.

In Mills's case, where we have to do with a man who had suffered from complete word-deafness for thirty years, without any improvement in his condition, it was found at the *post-mortem* that there was considerable atrophy of T^1 and T^2 on both sides, but chiefly on the left.

In Edlich's patient, a change was first noticed in her character and manners, together with a reduction in her command of words; afterwards real motor aphasia gradually developed, associated with sensory aphasia, which was incomplete at first, but afterwards became complete, and was accompanied by agraphia and total alexia. The patient only showed signs of sensory apraxia. In the last stage of the disease, the patient's conduct was increasingly insane; she no longer recognized persons and rooms, wandered about aimlessly, rose at night, and displayed a marked tendency to laughter and tears. At the autopsy, the weight of the brain was found to be reduced to 893 grm. This decrease in weight was due to advanced atrophy of both the frontal lobes, while in the corresponding sulci the pia formed cysts as large as a hazel-nut. The parts most atrophied were the posterior portion of F^3 and the left insula.

In my case, No. 1 (published in 1902), which was that of a woman (illiterate), aged 67, the first symptoms appeared after a slight stroke; they consisted in in slight dysarthric troubles (the patient pronounced words badly and scanned them), in a reduction in her command of words, and in a slowly progressive incapacity for understanding the meaning of words. These symptoms continued to increase slowly; thus, a few months before her death, the patient did not understand a single word, and could only pronounce certain syllables (be, be, po, po, pa).

During this period, she performed foolish acts due probably to agnostic apraxia. She put her dress over her jacket, exchanged her spoon for her fork or knife, burst into laughter or tears without any reason. Examination revealed increased resistance in the limbs to passive movements. Reflex iridoparesis to the light, mostly on the left. Macroscopic and microscopic examination showed that the convolutions were reduced in volume, and that their external surfaces were shrivelled and dark in colour, especially on the left. The cerebral hemispheres were full of liquid. The grey matter of the cerebral cortex and of the cerebral ganglia were of a darker colour than usual. The weight of the encephalon was 1,000 grm. Histological examination (Nissl's technique and method) revealed a considerable amount of alteration in the nerve-cells of all the strata of the cortex, and especially in those of pyramidal shape. In the pars opercularis of the left F^3, a fair number of the nerve-cells were affected by a diffusion of pigment; in many, a good deal of the cytoplasm had disappeared, while some cells showed the characteristics of chronic alteration. In the lip of the calcarine sulcus and in the left T^1 alterations in the nerve-cells were relatively rare.

In the patient studied by Alzheimer there gradually developed, first motor aphasia, then partial sensory aphasia and lastly word-deafness. At the *post-mortem*, T^1 and the lobulus parietalis inferior were found to be darker than is normally the case. Microscopic examination revealed in these, degenerated convolutions, sclerosis and calcification of the nerve-cells, containing both large and small nuclei poor in chromatin. The medullary fibres of the corresponding cortex had disappeared. At the same time, in some of the brown spots of the above-mentioned convolutions, a large number of enormous pyramidal cells had made their appearance.

Veraguth's patient manifested, for a short time, symptoms referable to the subcortical form of sensory aphasia. Words sounded to him like a murmur, while his spontaneous speech was fluent and free from mistakes. At the *post-mortem*, there was discovered to be a considerable degree of atrophy of the posterior portion of the pars opercularis of the left F^3, and also of the median portion of the left T^1; these zones were reabsorbed and their places taken by a serous liquid. No microscopic alterations were met with in the injured part.

In Franceschi's patient a relatively short period of depression was succeeded by sufficiently clear symptoms of sensory aphasia, together with signs of extreme dementia. The latter revealed itself both in serious lapses in behaviour, and in the fact that the woman was unable

to answer questions coherently. Neurological investigation disclosed cutaneous hyperalgia, loss of the plantar reflexes and exaggeration of the patellar and Achillis reflexes (without clonus). There was noticeable atrophy of the three left temporal convolutions. In the cerebral cortex corresponding, many nerve-cells had either disappeared, or undergone alteration, this being especially the case with the pyramidal cells. In the zones whence the nerve-cells had disappeared, there were many glia elements, both in the grey and the white matter. A slight rarefaction of the radial and of the transverse nerve-fibres was also recorded, together with an absence of the tangential fibres. These alterations were much less evident in the right temporal convolutions.

Pick studied three cases of the kind. The first was a woman, aged 58. The disease had begun two or three years before with symptoms of dementia in the form of loss of memory and serious loss of the power of identification; she believed that her son was one of her nephews, that the house she inhabited was her own property, &c. Upon this, followed an actual loss of all capacity of orientation; the patient no longer knew the time, and could not find her way to church. An examination, made at this time, revealed difficulty in finding words and in understanding their meaning. Thus, the patient was incapable of understanding the sense of words, but she repeated them correctly and in the form of a question. Two years later, the dementia had made rapid progress, and the patient no longer comprehended any questions, while her command of words was reduced to the use of a few stereotyped phrases.

Pick's second case was that of a woman, aged 75, who had already been ill for three years when she came under his observation. She began to show signs of want of power of orientation, pulled up the vegetables in the garden and went out of her house for no purpose. Her comprehension of questions was most limited, so that she gave one the impression that she had become almost deaf. She could write well if she copied (servile writing). Some months later, the disturbance of her speech became more serious; a jargon-aphasia prevailed, both in her spontaneous speech and when she named objects. Her command of words had continued to become more limited, with a tendency to the repetition of the same phrases.

The third case was that of a woman aged 38, suffering from progressive paralysis, in which numerous oculo-motor disturbances were accompanied by hypokinesis of the lower branch of the seventh right nerve and noticeable dysarthria and exaggeration in the patellar reflexes.

The investigation of her speech revealed the presence of paraphasia (of the verbal and syllabic type), less evident during emotion. But the dominant symptom was amnesia verborum; the patient when asked to name an object always replied "I do not know what it is."

At the *post-mortem* of the first case was found a considerable atrophy of the convolutions, and especially of the left frontal and temporal gyri. In the second case, the atrophy of the encephalon was so great that its weight was reduced to 967 grm. The affection extended to all the convolutions, but was most marked in the left temporal lobe. The condition of the third case was almost identical.

In my present case (No. 2), the patient began first to show signs of partial word-deafness, which were very soon followed by symptoms of agnostic apraxia, amnesia verborum, paralexic troubles, and almost complete agraphia. Later, the sensory aphasia became complete, and was associated with dysarthria in repetition; while the patient's command of words became increasingly limited, and the dementia increased. At last, the agnostic disturbances became extreme, the pre-existing symptoms of aphasia became aggravated and more evident, while the patient's spontaneous speech became reduced to the repetition of the stereotyped words "Don Antonio," and complete loss of comprehension of words and total agraphia followed.

The *post-mortem* revealed a considerable amount of atrophy of the prefrontal lobes, especially of the pars opercularis of the F^3, and of the upper temporal convolutions, the left being more markedly affected than the right, and particularly in the middle portion. Microscopic examination of the cortex of the more affected convolutions showed the presence of a true process of degeneration affecting the nerve-fibres of the medullary axes, the superior and inferior plexuses and the tangential fibres; the nerve-cells of the cell strata of the cortex were severely altered; the nerve-cells of the motor word area had suffered more than those of the auditory word area; and those on the left side were more affected than those on the right.

The symptoms of the Dejerine and Sérieux case coincided (Liepmann) with the complex of transcortical sensory aphasia, all the more since difficulty in reading and writing were uncommon. The Dejerine-Sérieux patient, in fact, was affected by a slighter degree of echolalia, namely, in the form of asking questions, and did not get as far as subcortical sensory aphasia (Pick). Further, the essential feature of this last type of aphasia is that speech is not apprehended as such, and therefore is neither understood nor repeated, while the other speech

functions are intact. In Dejerine's case, on the other hand, the patient repeated the questions, although, as a rule, he did not understand the sense. In writing from dictation, he was faithful to the sounds of the words, and the chief mistakes were those which anyone (writing) would make, through failing to understand the sense of what was dictated. At the *post-mortem* was found symmetrical atrophy of the temporal lobes, which above and before were reduced to half their normal volume, so that the insula was exposed. Their cortex had more consistency than that of the rest of the hemispheres; the pia was adherent, microscopic examination revealed the disappearance of the tangential fibres of the nerve-cells of the molecular strata, of the fibres of the radii and the short association fibres, as well as a decrease in the number of the pyramidal cells, especially in that of the small ones.

Rosenfeld's first patient (a man, aged 62) was sufficiently correct in behaviour during the early stage of the disease, but he was less active and played chess all day. His powers of reading, writing spontaneously, of copying, and of understanding and speaking spontaneously were perfect. With the passage of time (eight years) his power of criticism weakened, as did also his capacity for association of ideas. The neurological examination carried out one year before his death (i.e., when he was received into the hospital) showed that the motion and reflexes, &c., were normal; verbal amnesia was very noticeable; he was able to read and to write from dictation. On the occasion of a second examination it was observed that the conditions of speech were unchanged, though he uttered few words spontaneously. The speech disturbances remained the same until the patient's death, which took place one year later. At the *post-mortem* the left temporal lobe was found to be much reduced, the atrophy having chiefly affected T^2 and T^3. T^1 was relatively larger than either of the latter. The convolutions (including the apex) of the right temporal lobe were smaller than normal. Microscopic investigation showed that in the medullary zones, which were much reduced, the perivascular lymphatic spaces were notably dilated, and that an enormous number of nerve-fibres had disappeared from the cerebral cortex. The number of the ganglion cells were greatly diminished, but they had mostly preserved their fibrillary structure; the nucleus was, in general, well preserved; it was surrounded by a granular substance.

In Stransky's case it was noted that the patient could not understand the sense of many words, and showed a tendency to reply, repeating first the question addressed to him (not automatic echolalia),

but evinced little inclination to speak spontaneously. He could repeat correctly what he heard, and also some songs, but nothing else at will; he also showed, in general, a tendency to word-perseveration. He used to read aloud, but with evident paralexia, and he did not clearly understand what he read. He was incapable of writing, and even of copying. Flexibilitas cerea was evident. The patient was unable to recognize the use and significance of the commonest objects. He manifested a tendency to echopraxia, servile imitation of what he saw others doing. At the *post-mortem* atrophy was found to be present, especially on the left side, some special parts of the cerebral convolutions being affected, i.e., the middle part of gyrus calloso-marginalis, the cuneus, the præcuneus, and, above all, gyrus supra-marginalis; the atrophy of the said zones on the right was much less noticeable.

As a general conclusion we find that aphasic disturbances are not always uniform. In some cases the prevalent symptom is perceptible even to lay minds, as it consists in an incapacity to comprehend the meaning of words and of a considerable number of questions, and it manifests itself early. Gradually, the disturbance becomes more and more noticeable, and finally the patient becomes indifferent to all questions. In the development of aphasic troubles, occasionally the intervention of true paraphasic troubles, of the verbal and syllabic type, has been recorded, sometimes amounting to jargon-aphasia, or perseveration. Dysarthric disturbances are of rare occurrence (in Pick's first case and in my patients No. 1 and No. 2).

Sometimes the patients, though understanding the questions, repeated them before replying (simple echolalia); with the progress of the disease, this echolalia always assumes the automatic form, for the patients repeat the questions, almost as if obliged, though they do not understand their signification. With sensory aphasic disturbances are often associated, from the commencement, difficulties in recalling and pronouncing words. Occasionally these symptoms precede the sensory aphasia. The first signs are an incapacity for finding words—amnesia verborum—which later on gives place to a progressive reduction in the command of words, and also to an incapacity for repeating pronounced words. This trouble always increases with time, so that finally every effort of the patient to speak merely results in the utterance of one or two words, or of some syllables (in my first case the patient said be, be, po, po, pa, pa) or at most a few stereotyped phrases (Pick's cases 1 and 2, my case No. 2). In a few cases it is almost impossible to judge whether the progressive aphasia proceeded

parallel with the loss of the memory of the words, or if it was only consequent on an inability to recall the motor images of an always greater number of words.

Many of the patients examined were illiterates, which accounts for the lack, in the greater number of cases, of notes respecting their capacity for reading and writing. In rare cases, the power of reading and writing remained unimpaired throughout the course of the disease. As a rule, the patients at first understood what they read, but made mistakes when reading aloud (paralexia), but they always finished by being unable to pronounce the words they read, much less could they understand them (word-blindness). Sometimes the capacity for writing was partially retained; the patients could write to dictation and could copy. Sometimes difficulties in writing (servile writing) showed themselves, either at the beginning or during the progress of the disease, and generally increased almost parallel with the development of the sensory aphasic troubles, so that in the end the patient was unable to write at all.

Parallel to the aphasic disturbances, disorders of the other mental processes developed. At the beginning of the disease the patients neglected their business, became less efficient, and showed less capacity for work, and above all, their critical faculty became weakened. With the progress of time they forgot things to an enormous extent, seemed to have no idea of time or of place; they were unable to distinguish persons, and no longer recognized the faces of their friends or the aspect of rooms, but wandered about aimlessly, arose during the night, pulled up plants in the garden, and no longer knew how to dress themselves, sometimes putting on only one shoe or one sock. Sometimes they showed actual signs of dyspraxia, usually of the agnostic type, putting a dress over a jacket, using a spoon instead of a fork, putting socks over their hands, perhaps in consequence of partial psychical blindness. This symptom-complex is always completed by exaggerated emotion which culminates in spasmodic laughter or tears (Edlich's case) usually without adequate cause (my case No. 1.)

Paralytic symptoms rarely manifested themselves during the course of the disease. In my case, No. 2, there was slight facio-lingual-brachial hypokinesis with an exaggeration of the tendon reflexes of both sides and sometimes with clonus of one foot. In Shaw's case partial paralysis of the right arm is recorded; this, however, afterwards disappeared. In Liepmann's patient there was, in addition to a slowness in pupillary reaction, a tremor of the hands and tongue. The patient walked insecurely, and with his legs far apart.

The age at which the disease declares itself varies from 38 to 67; it attacks men and women about equally. Its course is rather slow and progressive, the exact date of its beginning is difficult to determine. In general the disease lasts from two to eight years, with an average duration of four years. Mills alone states that in the case of his patient the malady had lasted for thirty years.

As for the macroscopic and microscopic data recorded by the various writers in studying their cases, it can be stated that the fundamental change consists in an atrophy of the cerebral convolutions, and that the atrophy is not uniform, either as regards the different lobes affected or as regards the right and left halves of the brain. The alterations are generally more pronounced on the left side. In some cases there occurred a dilatation of the lateral ventricle, especially of the occipital cornu (Marie's second case). Occasionally this dilatation is associated with atrophy of the medullary matter of the hemispheres, and with sclerosis of the subependymal region (first case of P. Marie and Léri). At other times it is a question of an atrophy of all the cerebral cortex (my case No. 1), but chiefly of the left T^1 (Ascher's case, Pick's second case), or else the atrophy is limited to one part of the parietal and temporal lobes, i.e., to the gyri angulares and to T^1 and T^2 (Shaw), to both T^1 and T^2 (Mills), to the supramarginal gyrus, to the cuneus and to the præcuneus (Stransky), to the upper and anterior portions of the temporal lobes (Dejerine and Sérieux), or to the three temporal convolutions, especially the left (Franceschi). Sometimes the atrophy affects the frontal convolutions as well as the temporal (my case No. 2 and Pick's cases 1 and 3), or the frontal convolutions of both sides, and the left insula (Edlich).

The histological examination of the cortex of the atrophied convolutions, which has hitherto seldom been made, reveals varicose swellings of the fibres and almost always the disappearance of many of the transverse fibres of the infra- and supraradial plexuses. Almost without exception a greater or lesser number of the nerve-cells have disappeared in an almost regular manner from all the strata. Nearly all the nerve-cells which have been preserved show signs of pronounced modification, which consists in the pigmentation and reabsorption of the cytoplasm. Occasionally an increase was observed in the cells of the glia of part of the atrophied convolutions. A parallelism does not always exist between the alterations of the medullary fibres and those occurring in the nerve-cells (my case No. 2).

In Alzheimer's case the nerve-cells had undergone degeneration

and were sclerotic and calcified; the nuclei were poor in chromatin and there were giant pyramidal cells at the same level in some of the convolutions, which were of a darker colour than normal. It may be said, briefly, that the lesion causing the aphasic symptoms consisted of an atrophy, which rarely extended to the whole cerebral cortex, but was limited, although not exclusively, to the temporal convolution, or else to the frontal lobes, usually the left one. The corresponding histological alteration resolves itself principally into a primitive degeneration of the nerve-cells and medullated fibres of the grey matter of the cortex, and of the axis of the convolutions.

The reasons that atrophy at one time attacks all the cerebral cortex, and at another only affects certain parts, are at present quite unknown to us. It is impossible to conceive that this difference of behaviour can depend upon the method of the distribution of the arteries, for this hypothesis would not explain why the disease sometimes spares the gyrus præcentralis, or why the middle of the gyrus temporalis supremus is much more frequently attacked than the extremity. Sometimes an evident parallelism can be observed between some of the aphasic disturbances and the seat of the macroscopic cortical changes. In those cases where the sensory-aphasic symptoms are dominant (Mills, Bischoff, Dejerine, Liepmann, Franceschi, Pick's case No. 2), the only atrophied convolutions were exclusively, or generally, the gyri temporales 1 and 2. On the other hand, motor-aphasic symptoms were associated with sensory-aphasic disturbances (my cases 1 and 2, Pick's case 1, Edlich), atrophy also attacked the pars opercularis of F^3, together with the other convolutions of the prefrontal lobe. Nor should it be forgotten that in Shaw's case, and in my case 2, in which symptoms of word-blindness were also recorded, the left gyrus angularis was noticeably more atrophied than the other convolutions.

The relation between the cortical zone, which is the seat of the atrophy, and certain special aphasic symptoms ought, however, to be further investigated. It may here be called to mind that, according to some investigators, the fact that amnesic aphasia gradually produces loss of command of words is due to the ever-increasing affection of the temporal convolutions, while others hold that under these circumstances the process is not limited to the temporal lobe, but extends to the other zones of the hemispheres. This is the opinion of Pick; he points out that in the course of senile atrophy of the brain, even where the temporal lobes are also much atrophied, no noticeable loss in the command of words occurs, even if the patient no longer understands what he is

saying. According to Pick, the decrease in the command of words is a sign that F^3 is attacked. This idea is confirmed by my case where not only T^1 was affected, but the whole of the pars opercularis of F^3.

As for the word-deafness, Pick holds that, in his case, it was not a question of want of perception of words, because the atrophy had not begun in T^1. Rosenfeld is of the same opinion, since his patient, though he was ill for about eleven years, never lost his power of understanding words (nor did his command of language decrease). Macroscopic investigation showed that the T^1 was relatively little affected, and that the atrophy had chiefly attacked T^1 and T^2, together with the gyrus occipito-temporalis. This opinion is also confirmed by my present case, since the right T^1 and T^2, especially in the median zone, were much atrophied, together with those on the left side, and the histological alterations of the corresponding cortex were considerable, particularly on the left side, while my patient had almost completely lost the power of understanding the significance of words. This, on the other hand, tends further to confirm the belief of the bilateral nature of the seat of auditory word images.

Opinions are at variance concerning the seat of amnesia verborum. Rosenfeld is inclined to think that the centre for the recollection of names is to be found, on the left, in these three convolutions (T^2, T^3, gyrus occipito-temporalis), because these were the most affected in his patient. Quensel, on the contrary, holds that a morbid process going on in the gyrus angularis and in part of the gyrus supramarginalis (the so-called "via sensoriale anacentrale") should produce symptoms of amnesic aphasia. This opinion is not shared by Rosenfeld, who could not record any considerable reduction in these cerebral regions of the brain of his patient. Others, in conclusion (Wolff, Dejerine), hold that it is impossible to localize amnesic aphasia, since it forms a part of motor aphasia, and depends upon a diffused morbid process affecting the cerebral cortex. Rosenfeld, however, draws attention to the fact that this opinion cannot be unconditionally accepted, for in his patient's case there was certainly a diffused morbid process, but this extended chiefly to the said three convolutions (T^2, T^3, gyrus occipito-temporalis). From the data of my present case, it is impossible to come to a conclusion in favour of either view, for while it is true that the three convolutions just mentioned were atrophied, if only partially, nevertheless the left gyrus supramarginalis and angularis, as well as the convolutions of the prefrontal lobe, were also affected to a considerable degree. We could, therefore, only logically

deduce from this, that amnesia verborum is not due to a process diffused throughout the whole cerebral cortex. However, it would be illogical, since sufficient proof is lacking, to state that the rest of the cerebral cortex was sound, only because the volume of all the other convolutions was apparently normal. It will therefore be wise provisionally to look upon the most various zones, of the cerebral hemispheres, at least to some extent, as the anatomical ground of amnesic aphasia (v. Monakow and others).

Amongst the disphasic disturbances which sometimes occur during the course of aphasia due to atrophy of the convolutions, echolalia is mentioned. The seat of this symptom is also very far from determined. There are two kinds of echolalia: the one consists in the repetition, in the form of a question, of that which has been asked, and which has been understood (Pick), and the other in an automatic, forced repetition (echolalia automatica). Now, since the path of speech, by means of which the repetition is effected, is the oldest in ontogeny (as is shown by the development of speech in young children), it is clear why the second kind of echolalia occurs in the case of senile atrophy which is confined to the left temporal lobe, only at a stage when the sense of words is no longer understood (Liepmann) and, in truth, if the stimulation of the sensory centre of speech no longer awakens any response in the other cortical regions, it finds a vent along the more ancient path of speech (motorial path).

It therefore follows that it is impossible for the complete physiological separation of the temporal lobe from the other speech zones to arise from the atrophy of the temporal lobe alone, and it is thus logical to think that other parts of the cerebral cortex are also involved, and, therefore, that forced automatic echolalia represents a very marked degree of sensory transcortical aphasia, and hence is only found in serious degradation of the psychic functions. Accordingly it is not to be wondered at if two of Pick's cases (Ruczica and Pal) in which automatic echolalia occurred, manifested in the highest degree general atrophy of the brain and of the left cerebral hemisphere in particular. In the case of my patient No. 2 also, where atrophy had not only extended to the temporal lobe but also to the frontal lobe, the echolalia had become automatic only three years after the beginning of the illness. In Dejerine's case, on the other hand, where the brain was normal, with the exception of the two temporal lobes, the symptom in question was entirely absent. The same may be said of Liepmann's case, in which there was atrophy of the left temporal lobe alone. Perhaps the

simultaneous atrophy of the right temporal lobe may contribute to automatic echolalia; that hypothesis is favoured by the case of my patient, where the cortex of the right gyrus temporalis supremus also presented histological alterations.

It is thought that the study of cases of partial atrophy of the brain may open the way for the acceptance of the belief in the localization of psychic functions, and therefore that these cases might form the point of departure for a topographical psychology. Unfortunately, the problem is far less simple. Here, it is true, we have a limited loss of large nerve elements which present a functional, and perhaps an anatomical unity. Nevertheless, in these cases, it is not permissible to establish a connexion between clinical symptoms and histological data, both because the intelligence of the patients who present themselves for examination is already so much enfeebled that it is impossible to investigate exactly the psychic symptoms, and because also they are sometimes suffering from word-deafness. Thus in the case of my patient it was impossible to undertake my researches in this direction, and we were obliged to content ourselves with the study of his behaviour and conduct.

The data reported in the preceding pages testify clearly to the importance of the frontal lobes for eupraxia. Thus, Pick noted that one of his patients, who was suffering from serious atrophy of the frontal lobe, experienced difficulty in managing objects in common use. He, however, believes that this motor apraxia (in truth somewhat mixed) depended upon the atrophy being limited to the frontal lobe. In my cases Nos. 1 and 2, in which the atrophy of the gyri præfontales was also very great, the apraxic symptoms had attributes which were entirely those of extreme visual agnosia. At any rate it is not necessary to conclude that the aphasic and asymbolic (apraxic) manifestations are due to dementia, since sometimes, as in Edlich's case, dementia may not have already declared itself clearly at the period when the said symptoms prevailed.

I will remind the reader in this connexion of the phenomena of psychic blindness present in my case No. 2, and also in others. Psychic blindness and amnesic aphasia often proceed *pari passu*. To understand this, it is only necessary to remember that speech is a highly differentiated mental process, which has been acquired late in ontogeny, and is readily affected by any decrease in psychic power. This is shown by the difficulty in speaking experienced on waking, and when suffering from fatigue, or from any shock, although other functions remain intact.

In old age, the difficulty found in recalling words is due to an incapacity of raising the optic stimulus to the point of producing a response in parts which, in their turn, have become difficult to excite. Therefore, in the greater number of cases of pure cortical blindness, we find a certain amount of amnesic aphasia present; this shows that the stimuli conveyed to the visual cortex, and the revival of the conceptual complexes, are no longer sufficiently strong, if the zone of the sensory aphasia begins to atrophy, and that, further, mental visual stimuli will not succeed in evoking them. If, then, both the spheres are affected by incipient atrophy, but one more seriously than the other, as in many of our cases, it is clear why, even from the beginning of the disease, amnesic aphasia is a symptom often associated with visual agnosia. For this reason disturbances, similar to those which accompany psychic blindness, are often found in our cases. Let me remind the reader of the difficulty in recognizing objects, in recalling immediate images and names, the increased slowness of identification, the feeling of disorientation, the want of visual attention, and hence the difficulty of comprehending the whole together with the parts. All these troubles we find present, and a little accentuated, in the beginning of old age (Stauffenberg); and in fact to-day the pathology of the cerebral cortex has taught us that cortical plaques (a cytopathological sign of senility) are to be met with as much in incipient normal old age, as in eyes of old persons affected by conspicuous mental weakness (F. Constantini).

The results of the microscopic investigations of the circumvolutions when compared with the different clinical pictures reported in the foregoing pages, confirm us still more in the belief that the different forms of aphasia dependent upon the atrophy of the gyri are specially due to the degeneration or the disappearance of the nerve elements lying in defined cerebral regions. In fact, the researches have taught us that echolalia only becomes automatic when the atrophy extends beyond the left temporal lobe, whereas if it is confined to this lobe the disturbance consists only of a partial sensory aphasia. Further, symptoms of motor aphasia (reaching the point of dumbness) are associated with the preceding troubles only when F^3 and the insula participate in the atrophy, and finally, the wider the atrophy becomes diffused the more clearly do the sensory aphasic disturbances and the signs of dementia manifest themselves. This does not testify completely in favour of the idea that the whole cerebral cortex contributes to the comprehension of the signification of heard verbal symbols (v. Monakow). Certainly, the more difficult it is to evoke a representative image in consequence of

the extension of the atrophy over the various regions of the cortex, the more marked will be the difficulty in recalling words, and if the processes of mental synthesis become difficult owing to the same pathological cause, the comprehension of what is read, or heard, also is attended with greater trouble.

It would be very daring to deduce from such a small number of cases, some of which have been reported in a summary manner, criteria for determining during life whether a certain aphasic syndrome is due to progressive atrophy of the cerebral convolutions, or whether it depends upon a destructive focus situated in the speech region. But, in any case, a few considerations may be of some use in enabling us to proceed to a differential diagnosis. Points in favour of a cortical, or subcortical, focus of destruction are the sudden manifestation of the aphasia, or at any rate the existence of transitory prodromal symptoms only. On the other hand, in cases of aphasia due to atrophy of the convolutions, a stroke is nearly always absent, the speech troubles begin and increase with excessive slowness, so that it is difficult, even approximately, to determine the time of the appearance of the first symptoms. Nevertheless, sometimes, as in the case of Shaw's patient and of my No. 2, the first speech troubles were recorded as occurring immediately after a species of stroke, which was followed by transitory paresis of the limbs. A second criterion for differentiating between the various kinds of aphasia is the simultaneous presence of severe paralysis of the limbs, since facio-linguo-brachial monopareses (or monoplegias) are often associated with aphasia due to the destruction of the speech zone, especially in motor aphasia, or hemiparesis of the right side, which is sometimes flaccid and sometimes spastic, according to the extension, the seat, or the date of the beginning of the lesion. Motor troubles of such severity are never observed in cases due to atrophy of the convolutions. However, the presence of slight paresis of one side is not irreconcilable with the disease in question; it is only sufficient to call to mind my No. 2, in which the muscular strength of the right side was weaker than that of the left, and in which there also was present paresis of the right half of the tongue. Frequently also, in aphasia following the atrophy of convolutions, the action of the tendon reflexes is on both sides; this does not occur in aphasia due to the focal lesions. It may, however, happen that the Achilles reflexes are exaggerated on one side, even to the point of foot clonus (my case No. 2).

Hence, it follows that in some cases, and especially when a clinical history is absent, which could furnish us with precise data

as to the manner in which the aphasic troubles have developed, it will not be easy to give an opinion on the pathogenesis of the aphasic symptoms. In the present case, indeed, I succeeded in forming a correct judgment during life, basing my conclusions upon the fact that the sensory amnesic and aphasic troubles had developed slowly, that notable disturbances in the motor power, of the limbs at least, were wholly absent, and finally, that the patellar and the superior tendon reflexes were equal on both sides.

Sometimes it may be doubtful, especially where symptoms of word-deafness are dominant, whether the incapacity to understand the meaning of any question whatever is a sign of simple senile dementia. Nevertheless, analysis of the symptoms will always form some basis from which to arrive at a correct decision. Thus, for example, even in the most advanced stages of this psychosis, the patients always understand a certain number of questions, and do not reply like an echo, much less do they lose all command of language to such an extent as scarcely to be able to use one or two words. On the other hand, even in the total wreck of the intellect, such as occurs in advanced senile dementia, loss of the memory of graphic symbols has never been observed.

BIBLIOGRAPHY.

[1] ALZHEIMER. "Ueber perivas. gliose.," *Allg. Zeitschr. f. Psychiat.*, 1897, Bd. liii, S. 863.
[2] BEITRACH. "A Case of Traumatic Aphasia," *Arch. of Med.*, 1885, vol. xii, p. 233.
[3] BISCHOFF. "Beitr. z. Lehre d. sensor. Aphasie," *Arch. f. Psychiat.*, 1899, Bd. xxxii, S. 730.
[4] CLARK, P. "An Anomalous Case of Aphasia," *Journ. of Nerv. and Ment. Dis.*, 1894, vol. xxi, p. 703.
[5] DEJERINE and SÉRIEUX. "Un cas de surd. verb. pure," *Comptes. rendus. de la Soc. de Biol.*, 1897, t. xlix, 10me sér., iv, p. 1074.
[6] EDLICH. "Ein Beitrag. z. Kenntniss d. Aphasie," Greisswald, 1902, Inaug. Diss.
[7] FRANCESCHI. "Gliosi perivascolare in un caso di demenza afasica," *Annali di Neurol.*, 1908, vol. xxvi, pp. 281-290.
[8] LIEPMANN. "Ein Fall v. Echolalie," *Neurol. Centralbl.*, 1900, Bd. xix, S. 389.
[9] MARIE et LÉRI. Soc. Neurol. de Paris, Séance du 2 juin, 1904, in *Rev. Neurol.*, 1904, t. xii, p. 636.
[10] MINGAZZINI. "Klin. u. anatom-pathol. Beitr. über Aphasien," *Deutsch. Zeitschr. f. Nervenheilk.*, 1902, Bd. xxi, S. 386.
[11] PICK, A. "Zur Symptomatol. d. linken Schläfenlappenatrophie," *Monatschr. f. Psychiat. u. Neurol.*, 1904, Bd. xvi, S. 378.
[12] ROSENFELD. "Die part. Atrophie der Gehirnwindung," *Journ. f. Psych.*, xiv, H. 3 u. 4.
[13] SHAW. *Brit. Med. Journ.*, 1892, vol. i, p. 438, cit. in MILLS, *Brain*, 1891, vol. xiv, p. 467.
[14] SOREL. "Aphasie, epileps. jackson.," *Rev. de Méd.*, 1881, t. i, p. 1011; *Neurol. Centralbl.*, 1882, Bd. i, S. 39 (Abstract).
[15] STRANSKY. "Zur Lehre von den aphasischen," &c., *Monatschr. f. Psychiat. u. Neurol.*, 1903, Bd. xiii, Erganzshft., S. 499.
[16] VERAGUTH. "Ueber einen Fall von trans. reiner Worttaubheit," *Deutsch. Zeitschr. f. Nervenheilk.*, 1900, Bd. xvii, S. 177.
[17] WESTPHAL. "Klinischer u. anatomischer Beitrag zur Aphasie-Lehre," *Allg. Zeitschr. f. Psychiat.*, 1907, lxiv, S. 169.