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A STUDY OF

THE EFFECT OF CORTICAL EXCISION ON SPEECH IN PATIENTS WITH PREVIOUS CEREBRAL INJURIES

by

J. Preston Robb, B.Sc., M.D., C.M.

PREFACE

This study has been made possible by the work of Doctor Wilder Penfield, the Director of the Montreal Neurological Institute. The cases studied were those on whom he had operated for the treatment of focal epilepsy. For the opportunity of working under his direction, for the use of his material, and for the help he has provided, I would like to express my deep appreciation.

I would like to express my sincere thanks to Miss Anne Dawson who helped with the proof reading and to Mr. Charles Hodges for his help in preparing the photographs. I am greatly indebted to Miss Ethel Fanning for typing and checking the thesis so carefully, and would like to express my deep appreciation.

It is hoped that with the passing of time and the accumulation of more material, more definite conclusions may be drawn for some of the questions which here remain unsettled.

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I. INTRODUCTION

The present study was undertaken with the hope that something further could be added to the question of cerebral localization of speech. To know the anatomical location of a lesion in a patient with a specified type of aphasia is of interest to the neurologist but in the majority of cases it is only a matter of interest, and does little to help the patient. To the neurosurgeon, in planning and executing an excision of cortex, it becomes a matter of paramount importance. He wants to know what areas can be removed without affecting speech, and if known speech areas have to be removed, what the chances of recovery will be.

Dr. Wilder Penfield has accumulated, since his arrival at McGill University, a unique series of cases in which he has performed cortical excisions for the treatment of focal epilepsy. These cases form the basis of this study. Most of the information has been obtained from the hospital records of the patients. Eleven cases reported here were seen and followed by the author.

The cases were reviewed to see what areas could be removed from the major hemisphere without producing any aphasia and what areas, when excised, did produce an aphasia.

In the latter group the recovery was studied from two points of view. First, if a speech area was excised and the patient became aphasic could he recover, and secondly, if he did recover, was it due to contiguous areas taking over, or was it due to the corresponding area of the opposite hemisphere assuming the function of the excised or damaged cortex.

The evolution of our knowledge of the function of the cerebrum in regard to speech has developed slowly and there is still a great deal of work to be done. Weisenburg and McBride (1935) have made the most recent and comprehensive study from the psychological point of view. Nielsen (to be published) has summarized and added to the knowledge of the various areas of cortex related to specific types of aphasia. There remains still a great deal to be done from a physiological point of view. It is well known and recognized that Broca's area, the angular gyrus, and the first temporal convolution are important cortical areas for speech and that damage to any one of them in the major hemisphere will cause a disturbance of speech. However, the part which deeper structures play in speech is not as clear. The recent physiological and anatomical studies on the projection fibers from the various thalamic nuclei to the cortex, and the study of cortico-cortical connections by the strychninization method of Dusser de Barenne gives

one a more dynamic conception of the actual functioning of the cerebrum in the production of speech.

The electrical stimulation of the human cortex under local anaesthesia has been of great value in localizing functional units of the motor and sensory cortex. It is hoped that in the future a similar method may be developed in which the speech areas may be outlined by arresting their function. Preliminary work has been done during operations by Dr. Penfield with the cooperation of the author, which is here reported. It is hoped that with further understanding of the physiology of the cortex in relation to speech this method may be enlarged and become of practical value.

In order to reduce the body of the thesis, the case reports have been summarized and added as an appendix. As this thesis is to provide a basis for further work on the subject, some things that could ordinarily have been omitted are included to complete the work to date.

II. REVIEW OF LITERATURE

A) History of Aphasia with Special Reference to the Localization of Speech and the Function of the Major and Minor Hemispheres.

Little was known of the cerebral localization of speech before the nineteenth century. About this time, Flourens and others taught that all parts of the brain served the same function, and that any one portion could functionally replace another. In 1819, Gall, reporting on two cases, attempted to localize speech function to the anterior portion of the brain.

Later, Bouillaud (1825) reviewed a large number of post-mortems, and published the fact that he thought the faculty of language was situated in the frontal lobes. Marc Dax (1836) collected material over a 36 year period, and became convinced that the anatomical structure on which speech was based was located in the left side of the brain.

In 1861, Broca published his two famous cases and claimed that the faculty of language was at the base of the third left frontal convolution. Although Broca's work aroused considerable opposition, and he was unable to explain all cases on the basis of a lesion of the third left frontal convolution, it was on his series of cases that general recognition of the importance of the left hemisphere was established.

In 1862, Trousseau expanded the concept of Broca when he found several cases with lesions of the temporal and parietal lobes. He also suggested the use of the word "aphasia" meaning speechless, rather than "aphemia" which had been used by Broca.

In 1869, Bastian localized auditory and visual centers, as well as tongue and hand centers. He believed that all aphasic disorders resulted from the destruction of auditory and visual centers, and the fibers connecting these and the lower effector centers of the tongue, lips or hand. He was one of the so-called "diagram makers" who felt that in any case of speech defect it was possible to foretell the situation of the lesion with perfect assurance. They listed the clinical manifestations and the site and nature of the lesion which produced them.

In 1874, Wernicke, with the same point of view (Nielsen, 1936), assigned the auditory center to the first temporal convolution and the conceptive basis for articulate speech to Broca's area. He made a scientific presentation of anatomical material on sensory aphasia, and "Wernicke's Aphasia" is well known to-day. It consists of auditory verbal agnosia, visual verbal agnosia, alexia and agraphia. However, due to preconceived ideas he was unable to justify his theories by clinical observations. From his diagram he deduced four types of

speech disorder, motor aphasia, equivalent to the aphemia of Broca; conductive aphasia, due to interruption of the paths between his two centers; sensory aphasia, produced by the destruction of the auditory center in the first temporal convolution; and total aphasia, involving expression and comprehension, as a result of a lesion of both centers.

Perhaps the most outstanding worker on aphasia of that time, when viewed in the light of modern work, was Hughlings Jackson. His work was at first acclaimed, but due to the obscure way in which he wrote, and the difficulty in comprehension, his work was neglected in favor of those who stressed the anatomical approach. It was not until Pick, and later Head, brought attention to his work that its true greatness was appreciated.

Weisenburg and McBride (1935), in their critical review of aphasia, when commenting on Jackson, state - "The usual approach to the problems of aphasia in that day was structural and Jackson's analysis required a dynamic conception of behavior. It is hardly enough to say that he was ahead of his time. Even now, fifty years later, with the far greater knowledge of cerebral functioning and of the abnormal changes in language and behavior, there still appears every year work that has not caught up to Jackson".

Jackson insisted on the necessity of studying and

classifying the phenomena before any attempt was made to correlate them with morphological changes. He laid down the principle that a destructive lesion may never be responsible for a positive symptom. Pure destruction produces negative effects and any positive symptoms are the consequence of the released activity of lower centers. This general law he applied rigidly to the phenomena of affections of speech. He never failed to recognize that the phenomena of aphasia did not stand alone but illustrated the same principles and were governed by the same laws as other functions of the nervous system. Jackson is generally credited as being the first to realize the value of the psychological study of aphasia and the primary importance of exact clinical findings. He pointed out the difference between propositional and emotional speech and how the higher and voluntary aspects of speech tend to suffer more than the lower or automatic. Writing was affected not as a separate "faculty", but as part of the failure to propositionalize in words. Affections of speech he felt were caused, on the emissive side, by inability to form or express a proposition in words, and on the receptive side by failure of those mental processes which underlie perceptual recognition.

For further details on his work one is referred to the excellent review of Head (1926) in which he brings out the important observations and views of Jackson that are of help to-day.

In his lectures on "Affections of Speech", Gowers (1885) clearly stated his position as regards the subject and brought out many things that to-day are considered fundamental. He agreed with Charcot that no two cases of speech defect were alike, and that identical cerebral lesions would not necessarily produce the same clinical syndrome. He stressed the importance of time, pointing out that some weeks must elapse from the time of onset before one could infer, from the character of the symptoms, the location of the lesion. In acute lesions one would get a temporary derangement of the connecting fibers between the damaged and the intact centers concerned in speech. The action of the speech centers might also be interfered with, for a time, by disease that is near them but does not actually involve them or the fibers from them. He summarized his beliefs regarding cerebral localization as follows:

"In each hemisphere the lower part of the ascending frontal convolution contains the centers for the movements of the muscles concerned in articulation. From these centers conducting fibers pass down to the lower mechanism. Hence, motor processes for words must leave the cortex at this part. The adjacent posterior part of the third frontal convolution also contains structures that subserve speech, perhaps somewhat higher processes than those of the motor centers in the ascending frontal, and this region is usually regarded as the chief

speech center. Whether the island of Reil contains similar structures is still uncertain. The first temporo-sphenoidal convolution contains the structures that subserve the auditory perception of words. Those for the visual perception of words are probably contained in or near the angular gyrus. But there is an important difference in the functions of the two hemispheres. Voluntary speech processes go on chiefly in the left hemisphere in right-handed persons, in the right hemisphere in left-handed persons. The sensory word processes, perhaps influenced by the motor, also go on chiefly in the left hemisphere. Disease of the left motor speech region causes loss of the power of uttering words voluntarily; and that of the first temporal convolution, loss of the power of understanding spoken words - "word deafness"; whereas disease of the corresponding regions of the right hemisphere produces no such effect. The power of understanding words that are seen is also localized in the left hemisphere. Thus, although the sensory centers for hearing and sight are double, one in each hemisphere, it is only in the left that they subserve the recognition of words."

Gowers felt that the left hemisphere had by no means a monopoly of speech function. The right hemisphere contained structures of similar position and connections and that these structures could supplement those in the left hemisphere.

He pointed out that loss of speech, due to permanent destruction of the speech region in the left hemisphere, had been recovered from and that this recovery, he felt, was due to the supplemental action of the corresponding region of the right hemisphere and was proved by the fact that, in some of these cases, speech had been again lost when a fresh lesion occurred in this part of the right hemisphere. This supplemental action occurred in the sensory, as well as in the motor, functions and occurred far more readily in children than in adults. In his experience permanent aphasia in children, from disease of the left hemisphere, was almost unknown, the loss of speech rarely lasting longer than a week and then the child spoke almost as well as ever. Hence he felt it was probable that speech processes go on more equally in the two hemispheres in childhood than they do in adult life. He also felt it was highly probable that there were individual differences in this respect among adults. Certainly, with a lasting lesion, speech was recovered more readily by some than by others.

He agreed with Jackson that both hemispheres played a part in emotional expression and the automatic use of words, whereas the left side was used in proportion as the expression was voluntary.

Of the modern workers, probably the strongest protagonist of the strictly anatomical approach was Henschen. He reviewed

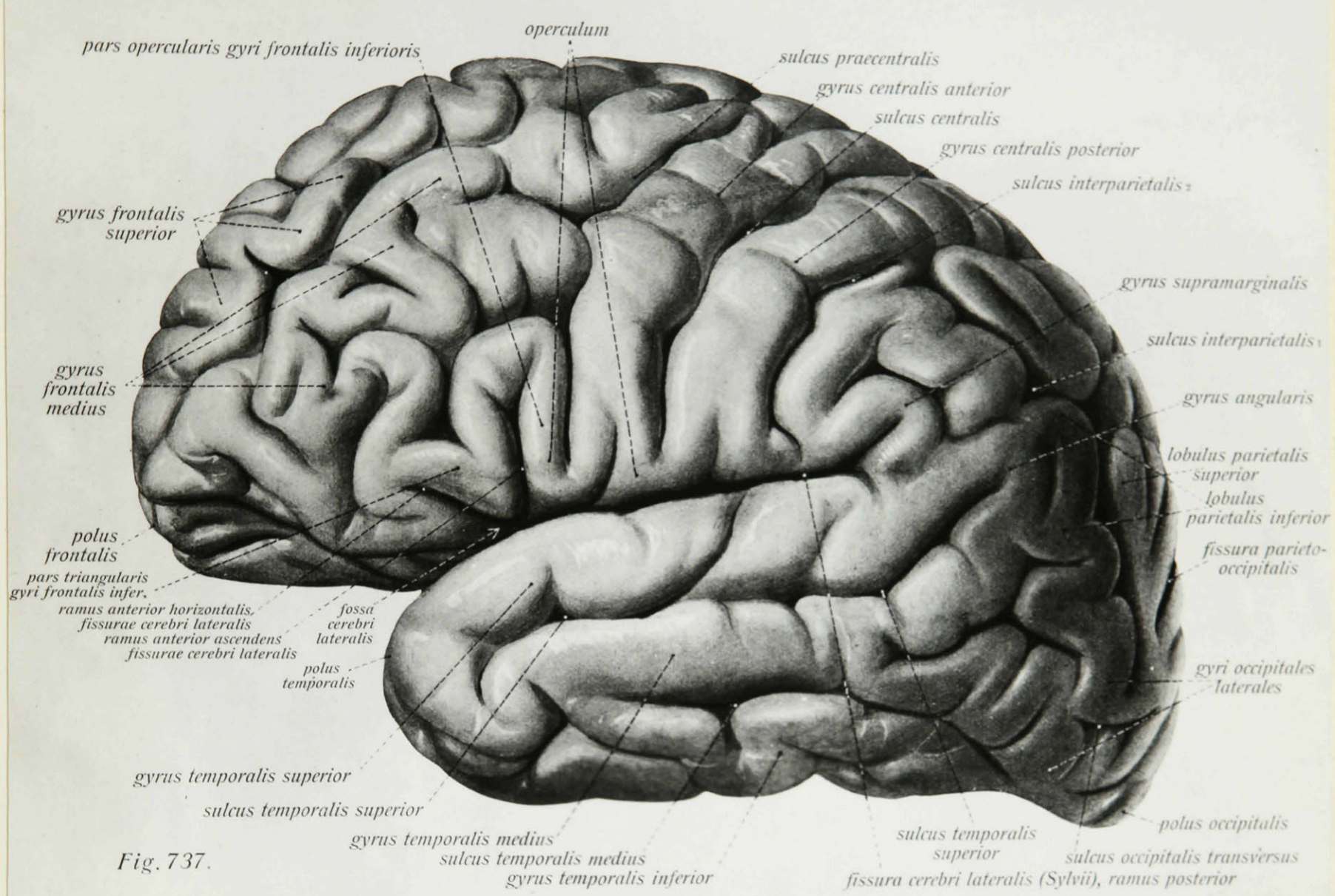

Fig. 737.

Fig. 1. Lateral view of left hemisphere.
(from Sabotta and McMurrich)

and tabulated all the cases that had appeared in the literature in which a fairly adequate amount of anatomical and clinical material had been presented. The difficulty with his work, as Weisenburg and McBride point out is that it did not allow for the finer clinical analysis which modern work emphasizes and which is, of course, impossible without comprehensive reports of the disturbances in their functional relationships.

Henschen (1926) was of the opinion that there were definite centers for speech, agraphia, word deafness and word blindness on the left side. The centers for motor forms of amusia and sensory amusia, for the most part, were on the left side, although they could be on the right, especially with instruments requiring two hands. He felt calculation was predominantly left-sided. Motor aphasia or "aphemia", as he preferred to call it, he localized in the left third frontal convolution. He felt, in cases where speech returned or was spared in lesions of this region, that this was due to the right hemisphere acting as a substitute. He pointed out that in cases where recovery had taken place and then a second lesion destroyed the right third frontal convolution, speech was lost completely and there was no recovery. This was contrary to von Monakow's belief that preservation of speech, or recovery of speech, was due to the cortex in the immediate vicinity of the third frontal convolution taking over.

Henschen felt that the transverse temporal gyrus was the hearing center, and the first temporal convolution the center for the hearing and understanding of speech. With bilateral destruction of the first temporal convolution there results permanent word deafness. With destruction only on the left, in the right-handed, there may be partial recovery through the activity of the right.

Word blindness was due to a lesion of the angular gyrus. In this case the right angular gyrus, he felt, was a poor substitute for the left. Destruction in two different occipital areas might lead to different forms of word blindness, associated with varying defects in writing and copying.

Although lesions of the angular gyrus, temporal lobe or parietal lobe might produce a secondary form of agraphia, Henschen was of the opinion that there was a specific graphic center at the foot of the second frontal convolution. A lesion here was usually associated with a lesion of the third left frontal convolution producing a motor aphasia, and the capacity for substitution by the right side was very poorly developed.

The center for calculation he placed in the left occipital region but it was poorly developed. The right side probably could take over simple forms of calculation.

The center for singing he placed in the left third frontal convolution, but the right side might suffice if the

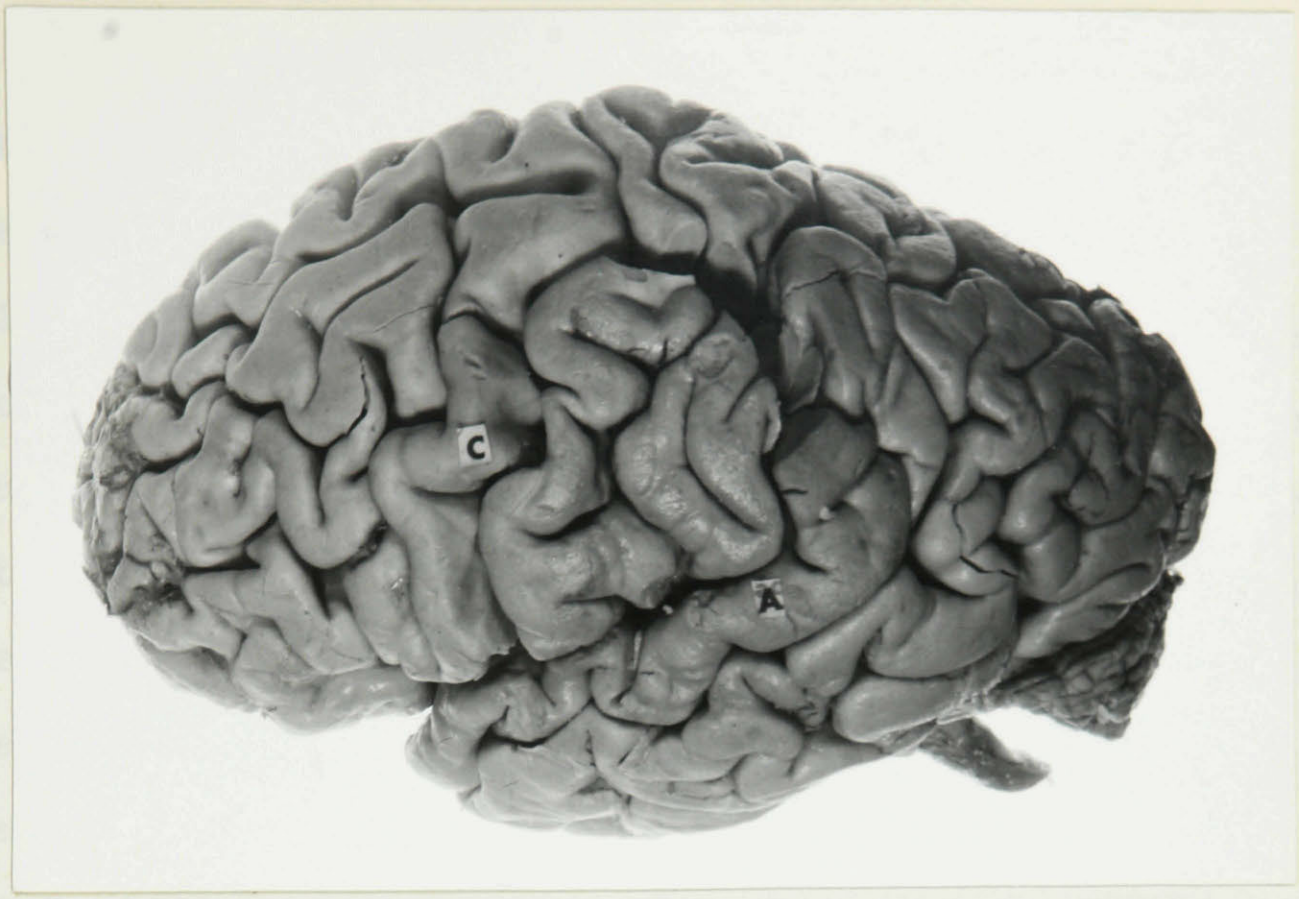

Fig. 2a. Dissection to illustrate the transverse temporal gyrus (of Heschl). A) first temporal gyrus, B) precentral gyrus.

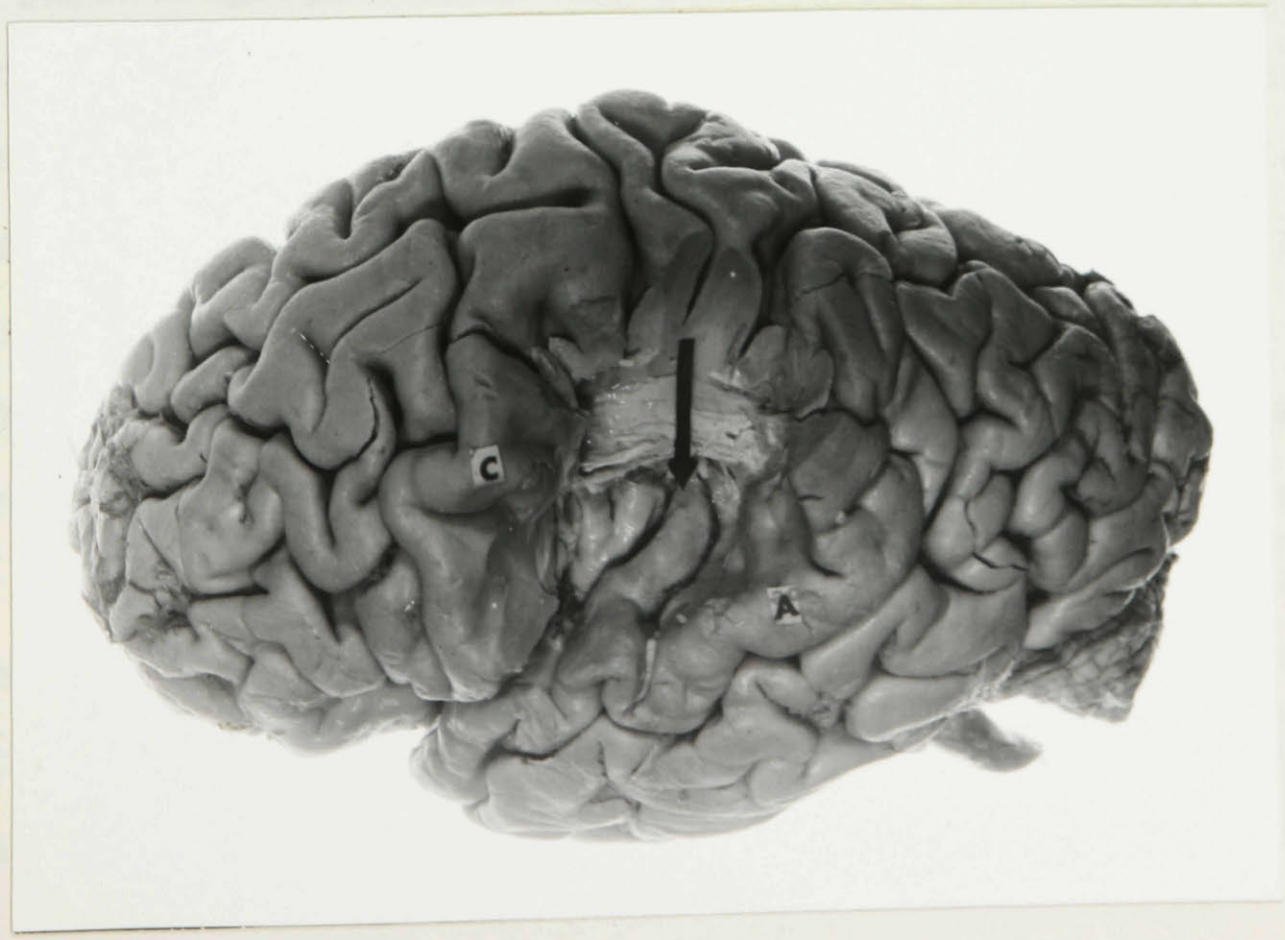

Fig. 2b. Same as 2a. Block of brain removed. Arrow points to transverse temporal gyrus.

left was injured. The left temporal pole was probably the important area for hearing and comprehension of music, but again the right could often compensate when the left was destroyed. The musical faculty he felt, was phylogenetically as well as ontogenetically older than speech, and its representation was more uniformally distributed over both hemispheres. Consequently, the right hemisphere could take over more fully the musical function of the left, when it was damaged, than in the case of speech.

In 1921, and later in 1925, S.A. Kinnear Wilson briefly reviewed the subject. Although there was little that was new, his work was important as it stressed the need, so often done since, for more careful work along anatomical, physiological and psychological lines. He pointed out how in the past the approaches had often been mixed and had rendered the subject most confusing. He doubted whether a pure case of motor or sensory aphasia ever existed, feeling that the so-called pure cases were probably the result of an unconscious trend towards schematization, and to an inadequate examination. A given case might be largely limited to the expressive side, but a receptive element could always be demonstrated if the examination was adequate. As the clinical studies should be complete, so also should the postmortem anatomical examination, including a careful microscopic study of the exact

extent of the lesion. Further, one should not neglect the fact that one is dealing with a limited neurone system, and the distal effects, due to a specific lesion, must not be overlooked.

Regardless of whether one could demonstrate pure cases or not, Wilson felt there was justification in seeking limited lesions for small aphasic disturbances although no correspondence between the anatomical size of the lesion and the physiological amount of impaired process could be expected in any regular way. He felt that no advance could be made in the study of aphasia as a cerebral symptom or psychical disturbance if we did not take a stand on the clinical certainty that varieties of speech impairment occur, are recognizable, can be differentiated and are of practical and localizing significance. For the clinician the problems of aphasia have to be faced in the same way as those of disorders of function of the non-speech parts of the brain.

He felt that there was considerable advantage in considering aphasia as part of a wider cerebral syndrome - viz., that of agnosia and apraxia*. Unless some explanation,

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*Wilson's outline (1921) of the relationship of aphasia to agnosia and apraxia is so clearly stated that it is here quoted:

"Word-deafness and word-blindness are defects of recognition; the patient sees and hears but does not understand. Now there are other defects of recognition than those concerned with words. He may see objects, but may not recognize them by sight; he may taste, smell, touch objects, but fail to recognize

them by these sense avenues. He may fail by one avenue but succeed by another. The general name given to this symptom of failure to recognize is agnosia. Thus we may have visual, tactile, auditory, gustatory, olfactory, agnosia. From this point of view word-deafness is a variety of auditory agnosia; it is agnosia for the sounds of words. 'Transcortical sensory aphasia', as we have seen, is agnosia for the meanings of words. Exactly analogous defects can be demonstrated in the case of word-blindness and in the case of the musical functions.

"On the expressive or executive side, the patient with expressive aphasia has no paralysis of the musculature of the lips, tongue, palate, etc., but he cannot speak. In agraphia, there is no paralysis of the small muscles of the hand but he cannot write. The generic term now employed for inability to perform certain acts, etc., where no paralysis prevents their execution, is apraxia. Looked at in this way, motor or expressive aphasia is apraxia of the lips, tongue, palate, etc., for the movements of speech. Agraphia is apraxia of the arm and hand for the movements of writing. Aphasia and agraphia may thus be, and not-infrequently are, part of a larger apraxia - viz., apraxia for other movements than those implicated in speech. Sensory or receptive aphasia may be, and often is, part of a larger agnosia - i.e., an agnosia for other things than words."

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such as that provided by the conception of apraxia and agnosia was offered, the limits of aphasia proper would be stretched unjustifiably. A patient who dresses himself wrongly or makes mistakes or "short circuits" in performing any act, such as lighting his pipe, shows a disturbance which cannot by any legitimate means be regarded as aphasia, but the disorder is certainly one of agnosia or apraxia. With these modern conceptions he felt the way was open for a more comprehensive insight into cerebral activities, both on the receptive, and the executive side.

Other modern workers have added a great deal to the problem, particularly along psychological lines. In 1926 Head published, "Aphasia and Kindred Disorders of Speech". He carefully reviewed the history of aphasia up to the time of publication, and presented his own views and cases. To Head must be given the credit of having been the first to direct general attention to the development of test methods exclusively for aphasia. They consisted of the so-called "serial tests" supplemented by various suggested studies of a less formal nature but requiring equally systematic procedures. The tests were designed to trace the central disorder of symbolic formulation and expression through its various manifestations in speaking, reading, writing and so forth. Weisenburg and McBride (1935), in commenting on these tests, point out that Head did not have a knowledge of how the normal person would respond to the tests. When they tried them they found that many normal people showed "aphasia" responses with the more difficult tests, and in this respect the tests were not adequate.

Head emphasized, as others had, that the function of the brain was so integrated that an isolated lesion of the speech areas would affect the use of language as a whole, and not a single phase of speech alone. Defects of verbalization, if sufficiently severe, would disturb the full appreciation of meaning and lack of recognition of meaning would prevent the

normal formulation of words and phrases. He also emphasized, as Jackson had many years before, that when some act or process was disturbed in consequence of an organic or functional lesion, the abnormal response was a fresh integration carried out by all available parts of the central nervous system. It was a total reaction to the new situation, in which conscious processes played their part as a mode of response.

This point of view of considering the part of the brain substance still functioning, rather than that which has been destroyed, has been shown to be particularly important in modern work on cerebral cortical function.

Weisenburg and McBride's work (1935) was another important step forward in the psychological study of aphasia. Their chief object was to make a survey of the mental functioning in aphasia. They developed a battery of tests in order to record accurately and completely the patients' responses.

In their series they included 60 clear-cut cases of aphasia, 22 control patients with right-sided lesions without aphasia, and 85 normal adults as a control group. As their work progressed and as more careful examinations were made, it became increasingly clear that there were no true cases of pure agnosia or pure apraxia, nor were there sharp steps as one passed from the receptive to the expressive group. For this reason they felt that the older classifications, based on inadequate examinations, became obsolete.

The division suggested by them was:

1) Predominantly expressive group.

2) Predominantly receptive group.

3) Expressive receptive group.

4) Amnesic group.

The first, the predominantly expressive group, showed the most serious disturbances in the expression of speech or writing. In the second, the receptive processes suffered more than the expressive and the changes in speaking and writing were mostly of a different nature from those of the expressive disorder. In the expressive receptive group all language processes were extremely limited. The last group, the amnesic, was created because psychologically it was so different from the predominantly expressive group, that it could not be classified with them, neither could it be classified with the receptive group as the understanding remained relatively superior. The term was one which was well established in the literature on aphasia, and gave a satisfactory description of the particular language difficulty.

As far as the actual localization of speech was concerned they have little to add. In the predominantly expressive group they placed the lesion largely in the anterior or motor part of the brain. In the receptive group they placed the lesion in the posterior or sensory part of the brain. In

the expressive receptive type both parts were involved. They were unable to state anything definite about the localization of the amnesic group.

In 1936 Nielsen published his excellent monograph, "Agnosia, Apraxia and Aphasia - their Value in Cerebral Localization". Since then he has accumulated further data, and a second edition is about to appear.* He agreed that in every case there was a certain amount of general loss which would correspond to the form named by Head as "semantic aphasia". However, there were innumerable cases on record to show that except for the specific loss in the various types of aphasia, as the great diminution of spontaneous speech in aphasia of Broca, the mutism in subcortical motor aphasia, the forgetting of names in amnesic aphasia, the loss of comprehension of spoken words in acoustic agnosia or the loss of recognition of written words in visual verbal agnosia, the remainder of the psyche was often comparatively little affected.

With this in mind he examined 240 cases clinically with 25 autopsies, 13 surgical verifications and two roentgenological confirmations.

He combined the results of his findings with those of other workers, and concluded that in some cases the symptomatology had a highly specific localizing value, and others had none.

*Through the kindness of Dr. Nielsen, the galley proofs for this publication have been made available to the author.

Hughlings Jackson first pointed out the importance of the minor side in emotional speech. His ideas were elaborated by Henschen who felt that in most instances when the major side was destroyed the minor side could take over. Nielsen (to be published) again emphasized the importance of the minor side. He felt that the major and minor hemispheres started in life almost equally incompetent. In some instances when the major hemisphere was destroyed the minor one would have developed its engrams so crudely that it would not function at all. In other instances it could formulate language slightly, understand a little immediately and improve rapidly with training. In a few instances it would have established its engrams so well that when the major hemisphere was suddenly destroyed, it would be able to assume the function of language at once.

Handedness, he felt appeared at about nine months, but the superiority did not become great. Thus, when a major hemisphere was destroyed at the age of four or five years the resultant aphasia was only transient. Guttmann (1942), in reporting thirty cases of aphasia in children, pointed out with what surprising rapidity they recovered from a motor type of aphasia.

According to Chesher's figures, ipsilaterality of the dominant hand and dominant hemisphere occurred in about 6 per cent of

all persons. Thus, in considering individual cases one must be careful not to assume that the major hemisphere is opposite to the major hand. Until one has specific evidence, such as aphasia associated with paresis, or following cortical excision, one cannot say which side speech is on; also the major side for different phases of speech (receptive and expressive) may be on opposite sides.

As a result of his research and that of others, Nielsen divided each hemisphere into eleven association areas, each with its counterpart on the opposite side. The degree to which the major was superior to the minor was not the same for all pairs.

1. Prefrontal lobes.
2. The frontal writing center (foot of the second frontal convolution).
3. Broca's convolution.
4. Pars triangularis of the third frontal convolution.
5. Anterior end of the superior temporal convolution.
6. Wernicke's area (posterior third of the superior temporal convolution).
7. Area 37 of Brodmann.
8. Angular gyrus.
9. Area 18 of Brodmann.
10. Area 19 of Brodmann.
11. Convolution of Gratiolet.

The prefrontal lobes, he felt, showed little, if any, unilateral superiority.

Area 18 of Brodmann (area parastriata), he felt, was concerned with the recognition of objects and pictures. During life one side or the other became dominant. This he thought was due purely to chance. Usually destruction of the major one led to a temporary disability only, and the minor side readily took over.

Area 19 of Brodmann, he felt, was concerned with revisualization of former images and followed the rules of lateral superiority described as governing area 18. These three areas were not greatly concerned with language and this fact probably underlay the weakness of unilateral superiority.

The pars triangularis of the third frontal convolution (area 45 of Brodmann) was concerned, he stated, with all the functions of vocal music and the playing of musical instruments. In rare instances only did injury to the major side cause any symptoms, as the minor side took over so readily.

The anterior extremity of the superior temporal convolution (area 38 of Brodmann), he said, was concerned with musical auditory recognition and interpretation. It received impulses from the transverse gyri of Heschl which acted as centers of primary auditory perception. To produce symptoms it was necessary to destroy both sides as one side assumed the function of the other so readily.

Wernicke's area (areas 41 and 42 of Brodmann), he felt, was concerned with the interpretation of spoken language. After destruction of this area, the minor side functioned but fatigued rapidly. The patient might understand a few sentences and then would not be able to comprehend anything without a period of rest. This was shown in the cases in which the major temporal lobe had been excised (Nielsen and Raney, 1939). After a year the patient could understand most things that were said to him, and in two years it would be difficult to demonstrate any defect.

Area 37 of Brodmann, he stated, was concerned with the formulation of language. In cases of even minor destruction the patient was unable to find his words or formulate his sentences. He thought it was an area in which knowledge was coordinated for significance of words and expressions and in which grammar, rhetoric and syntax were considered in the formulation of language Lesions of this area produced amnesic aphasia as their most constant symptom.

The minor side was usually capable of assuming, to some degree, the function of formulation of language, but until it had time for training, it functioned imperfectly and misuse of words, jargon aphasia or agrammatism would result.

Broca's convolution, he felt, contained the engrams representing the physiological basis of memory of how to use the vocal organs

for the production of words. After destruction, the minor side functioned as does the minor Wernicke's area. Destruction of it, in the adult left the patient in the great majority of cases only able to say a few words,and it might take several years to train the minor side.

Convolutions of Gratiolet, located between the angular gyrus and the second occipital convolution, was concerned with the body scheme. The major side was superior to about the same degree as that for Wernicke's area.

The frontal writing center (foot of second frontal convolution) and the angular gyrus he connected with the most highly specialized of language function, emission and reception of written and printed language respectively. The writing center at the foot of the second frontal convolution was essential to the memory of handwriting movements and in the majority of cases functions in close association with the convolution of Broca. It could not function in writing unless the language formulation area, (area 37 of Brodmann) and the area of revisualization of the written word (angular gyrus) were intact and in communication with it. The major writing center was far superior to the minor, in most cases, and a long period of training was necessary for the minor one to function at all well. He showed that conversion of a left-handed person to right handedness for writing established a writing center on the minor side (1937 and 1938).

The angular gyrus had a double function, one for recognition and one for revisualization of the written and printed word. The superiority of the major side was great. Unless the major side was destroyed early in life, the minor side rarely attained the degree of perfection present when the major side was intact.

Nielsen felt that if a minor lesion damaged an area of cortex concerned with language, the area involved might attempt to carry on its function, though crippled, or "resign" immediately. Whether it resigned or continued to function depended on the facility with which the minor side could assume its function. He did not know of any way in which one could tell what had happened in a given case. He felt that the polemical discussion of the last century in the field of cerebral localization arose from the neglect of the principle that the brain contained a major and a minor hemisphere of extremely variable degrees of dominance and recession. He pointed out the importance of studying bilateral lesions as there has not been a single case in the literature of bilateral destruction of Broca's area without aphasia. Similarly, there has not been reported a case of bilateral destruction of Wernicke's area without acoustic verbal agnosia (Nielsen, 1941).

After reviewing critical cases, Nielsen and Fri dman (1942) concluded that the putamina had no function in language.

The minor cerebral hemisphere assumed the function of the major in language with great facility in some cases, with difficulty in others and not at all in some persons. The language function, if partly destroyed, did not transfer to the minor side in toto, but the visual, auditory or motor functions might be transferred separately.

An artificial writing mechanism might be formed on the minor side by training and when this happens the entire speech mechanism did not of necessity move to its minor side. The major temporal and major occipital lobes need not be ipsilaterally located. Finally, they felt that it is unsafe to lateralize a lesion on the basis of an aphasic manifestation alone.

In 1935 Chesher reported 157 cases and lesions in the language zone in which the ability to formulate and to express in language appeared to be dependent upon a factor which was operative in only one cerebral hemisphere. This hemisphere was the one opposite the universally preferred hand. There were also nine cases of "mixed motor preference", who were left-handed for most things but wrote with the right. Five of these had left-sided lesions with aphasia and four had right-sided lesions with aphasia. He advanced the view that in the last group of nine, language was unlateralized, so that a lesion on either side could produce an aphasia. The first five of this group would tend to prove that an "artificial" writing center can be created in the left hemisphere in a left-handed patient who learns to write with his right hand. The last

four cases do not disprove this. The work of others, particularly Nielsen and his group (1937 and 1938) and Kuttner (1930) would tend to bear this out. They have reported cases in which a transient aphasia developed with a lesion of the left hemisphere, and later following a lesion of the same area of the right hemisphere, became completely aphasic.

B) Review of Some of the Experimental and Clinical Studies on the Function of the Cerebral Cortex in Relation to Speech.

Hughlings Jackson never failed to recognize that the phenomena of speech and aphasia did not stand alone, but illustrated the same principles and was governed by the same laws as other functions of the nervous system. With this in mind some of the modern experimental physiological, and clinical studies on the functions of the cerebral cortex are here reviewed in an effort to better understand the processes involved in the production of, and recovery from, aphasia.

1. Cortical stimulation in man:

Vocalization as a response to cortical electrical stimulation was described first in 1938 by Penfield. He reported six cases and since that time has had many others. It was produced by stimulation of dominant and non-dominant hemispheres equally. The area was localized to a small portion of the precentral motor gyrus between the area for eyelid movement above and the mouth below. The vocalization consisted of a loud continuous cry which in no way resembled words.

At the same time he reported cases in which speech was arrested by stimulation. The points from which such an effect was produced, were scattered over the hemisphere, but occurred more frequently in the neighborhood of Broca's area.

In 1933 Penfield and Gage reported producing a "buzzing sound" and the "vibration" of a drum by electrical stimulation of the third temporal convolution on the left side. To my knowledge this had not previously been reported. Later, Penfield and Erickson (1941) reported a case (M.G.) in which extensive stimulation of the temporal lobe was carried out. The patient heard a "buzzing" sound and had what she called dreams which consisted of hearing of music, hearing of voices and other hallucinatory phenomena that seemed to be in the auditory field. All of the stimulations were placed in the posterior portion of the first temporal convolution in an area that would correspond roughly with area 41 or 42 of Brodmann.

Penfield and Gage (1933) also reported the production of various coloured lights by stimulation of the occipital pole. It was their impression that positive stimulation might be obtained when an area had been subjected to recurring epileptic discharges, whereas the same area might be inexcitable in the normal brain. In other words, the habitual local epileptic discharge might leave behind a sort of facilitating influence.

2. Experimental cortical excision:

Much work has been done on experimental animals to determine the function of the cortex. First, the excisions were made to study the loss of normal function. Then the animals were studied to see how much return of function there would be, and if so, to what could one attribute the return. Was it due to contiguous brain assuming the function, or was it due to the corresponding area in the opposite hemisphere? This question, particularly in aphasia, is still incompletely solved.

Lashley (1929) performed cortical excisions of varying amounts and from various locations on rats and studied their ability to learn (maze habits). He concluded that the ability of these animals to learn depended not on what cortical tissue was destroyed, but on the amount destroyed. The mere existence of specialized regions in the brain (Lashley, 1937) was not conclusive evidence that specialization was necessary or important for integrative functions. On anatomical grounds alone, there was no assurance that cerebral localization was anything but an accident of growth.

Kennard (1940, 1942), did very extensive experimental work comparing the motor deficits which appeared after removal of known cortical areas in the infant and adult monkeys. She showed that the central nervous system of the sub-human primate,

during infancy, exhibited a greater capacity for re-organization, and compensation than did that of the adult. If,in the infant, areas 4 and 6 were removed, motion was not affected at once,but as the animal developed, one found fine movements of the hands were missing. There was in these infants re-organization of cortical function, and areas 3, 1, 2, 5 and 7 of Brodmann helped. If these areas were later removed the deficit would become greater. The spasticity and paresis were immediately and ultimately much less severe if the ablation was made in infancy. The greatest re-organization occurred during the first six months and the greatest loss of capacity to re-organize occurred at the end of the first year of life, the same time as which spasticity began to appear. The same she felt seemed to be true for man, but the evidence was not as good, neither paresis nor spasticity was as great when acquired early as when acquired late in childhood.

The frog, after cortical extirpation, behaves in an almost normal manner. The dog and cat can have their entire cerebral cortex removed, and still be able to stand and walk almost as well as normal animals. In primates this does not follow (Bucy, 1944). The process of encephalization has progressed to the point where the precentral cortex has assumed most of the control over the skeletal musculature, and when the precentral motor cortex is removed from both cerebral

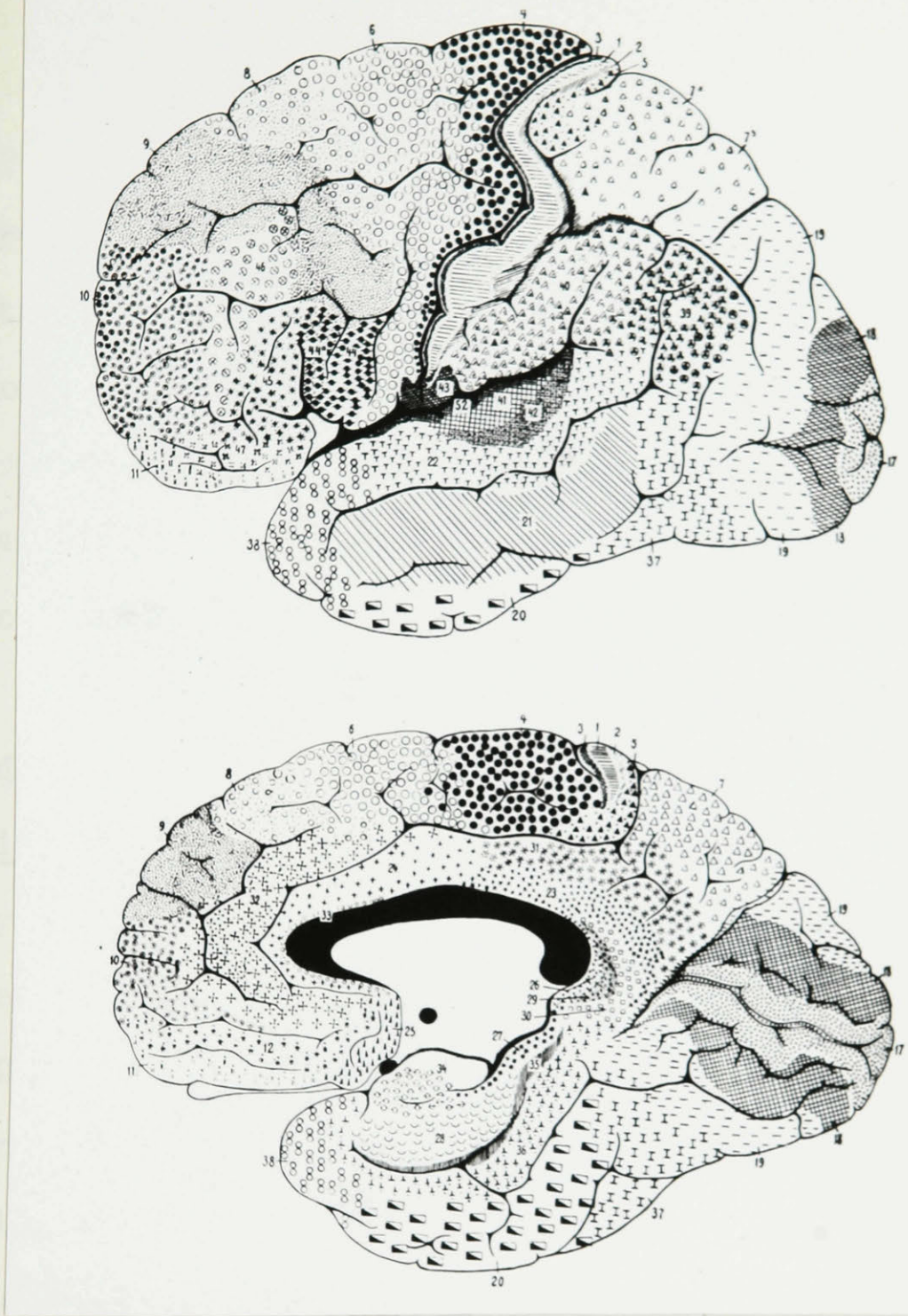

Fig. 3. Photograph of Brodmann's illustrations of his architectonic areas of the brain.

hemispheres, the animals become almost totally paralyzed and remain so even after 48 days. Bucy and Fulton (1933) and Bieber and Fulton (1938) showed that in primates practically all voluntary movement resulted from the activity of the precentral cortex. Any recovery of movement that occurred after removal of one part of this cortex must have resulted from the activity of some remaining part of the precentral motor cortex on the same, or opposite side, but not from the activity of some other cortical area outside the precentral region or from the activity of the basal ganglia as had so often been assumed.

3. Excision of complete hemispheres in man:

Bucy (1944), in reviewing the cases of complete removal of the right hemisphere in man (Dandy, 1928, 1933; Gardner , 1933; Rowe, 1937) was of the opinion that if all voluntary movement was dependent on the precentral motor cortex then all movement that persisted or returned in these patients must have resulted from activity in the ipsilateral precentral motor cortex.

In Gardner's case, after twenty months, the left masseter and temporal muscles contracted somewhat less forcibly than the right, but the jaw did not deviate on opening. There was very slight weakness of the left orbicularis oculi and of the left side of the face about the mouth. She was able to walk

well without support and to go up and down stairs unaided. There was no voluntary movement of the ankles or toes. The upper extremity was useless and there was no voluntary power. These studies would indicate that even with the removal of the entire precentral motor cortex of man on one side, it was possible to regain sufficient voluntary movement and control of the hip and knee to enable the patient to walk,and all the evidence pointed towards such recovery being due to innervation from the ipsilateral precentral motor cortex, and not due to the basal ganglia.

Zollinger (1935) is the only one to have reported the removal of the left cerebral hemisphere in a right-handed patient. His patient was a female aged 43, who was aphasic before operation. Several hours after the operation she answered "all right" to all questions. "Yes" and "no" were added the following day, but they were not always used properly. She was taught to say "thank you", "please", "good-bye", and "sleep", and began to show a more accurate use of words. She experienced emotional reactions and showed definite understanding. For example, while taking sips of water and replying "yes" and "no", apparently with full understanding of their meaning, she abruptly closed her mouth, and refused to drink or speak following an outburst of laughter on the part of one observer in the room. It was difficult to induce her to reply to

simple questions when the room was full of people. It was Zollinger's impression that the vocabulary could have been gradually increased with training. She died on the seventeenth postoperative day. Although this case is not conclusive, it is strongly suggestive that the right hemisphere was beginning to take over what Jackson would have called "propositional speech".

4. Experimental studies on thalamic projection areas and cortico-cortical connections:

Poliak (1932) and Walker (1938) settled the long debated question of the auditory projection in the primate cortex. Poliak,working with monkeys, experimentally interrupted the thalamo-cortical connections and studied them with the osmic acid method of Marchi. He outlined the course of the auditory fibers from the internal geniculate body to the cortex, the cortical area being the upper half of the superior temporal convolution and only that. Only a few fibers reached the free surface of the temporal lobe. The most richly supplied portion was a small zone in the posterior corner of the Sylvian fossa where there was an elevation comparable to the transverse temporal convolution of Heschl in man.

By the same method he showed that the fibers from the lateral geniculate body go exclusively to the striate area of the same side.

Others, Bremner and Dow, 1939,Woollard and Harpman, 1939, Ades and Felder, 1942 and Tunturi, 1944, etc.), by recording

electrically from the cortex the impulses transmitted from the medial geniculate body were able to confirm the location of the primary auditory projection area. By using different pitches they were able to demonstrate that the organ of Corti was mapped out on the transverse temporal gyrus.

Von Bonin, Garol and McCulloch (1942), by fixing the eye, and flashing a small light while recording from electrodes in fixed positions on the occipital lobe, were able to plot out the visual field on the cortex, and showed that in the primate, off and on responses were sharply restricted to area 17 of Brodmann.

Le Gros Clark (1932) and Walker (1938) made studies of retrograde degeneration of thalamic nuclei following destruction of various cortical regions of the monkey's cortex. From their work it was apparent that certain regions were dominated by projections from certain thalamic nuclei.

The projection from the lateral geniculate body was sharply limited to area 17, which is surrounded by area 18, and in turn by area 19. The occipital region, consisting of these three areas had a close functional organization subserving vision.

Similarly, it was shown (McCulloch, 1944) that the parieto-temporal region was dominated by projections from the medial pulvinar nucleus to the posterior parietal cortex and by the

inferior pulvinar nucleus to the posterior temporal cortex. The whole region played an important role in associated processes. It included areas 31, 7, 39, 40, 37, and 20 and was dominated by areas 39 and 37, which received the bulk of the pulvinar projections. There was no region in the entire cortex with which the parieto-temporal region failed to make contact. Inferentially it served association functions of a high order.

The central or "sensory region" received impulses from the lateral thalamic nuclei, those anterior to the central sulcus coming from the ventrolateral and those posterior from the posterolateral.

The frontal region received projections from the dorso medial nucleus of the thalamus and were most dense in a relatively small area which Walker has called area 46. This region included all cortex anterior to the central sector on the lateral aspect, with the possible exception of area 8. On the medial side it extended just across the sulcus callosomarginalis.

The Sylvian region was dominated by projections from the medial geniculate body to area 41, and with less density to area 42. This region included the opercular area. The sector of the anterior thalamic nucleus lay on the posterior portion of the medial aspect of the cortex and was co-extensive

with the posterior limbic region, consisting of areas 23, and a small retrospherical area which is probably best called area 29. The thalamic projections to the remainder of the iso-cortex were unknown.

Dusser de Barenne (1933) and Dusser de Barenne, Marshall, Nims and Stone (1941) reported the method of stimulation of the cortex with strychnine, and recording electrically the impulses at distal points. The local strychninization method has been used extensively to show the cortico-cortical connections by McCulloch (1944a, 1944b) and his associates. In the occipital region they showed that strychninization of area 17, the area striata, fired in area 18, the parastriate area. Strychninization of area 18 fired the adjacent sector of area 17, almost all parts of area 18 of that hemisphere, the corresponding point of area 18 in the opposite hemisphere and much of area 19, ipsilaterally. Strychninization of area 19 caused only a local response.

They considered area 43 and 44, although firing face area 6, to be part of the Sylvian region, which is dominated by projection from the medial geniculate body to the transverse temporal gyrus. Strychninization showed that area 41, the primary auditory reception area, fired area 42, the "secondary" area. Area 42 fired itself well, fired area 41, and as well as the adjacent portions of area 22. Area 43 fired area 44

and 22; area 22 fired 44 and 43, and probably 22. Thus, this region contained structures necessary for vocal response to auditory stimulation and their cortico-cortical connections were direct. The Sylvian region contained the projection areas for sound and the control of respiration, lingual and laryngeal musculature. The interconnections of these areas, McCulloch (1944b) felt were obviously the precursor of those cortical structures which in man are principally responsible for speech.

In view of all the recent physiological work that has been done one wonders if students of aphasia, in the past, have not considered speech too much as a phenomena of the cortex, and cortico-cortical connections alone. Perhaps these cortical regions, as controlled by thalamic projection and nuclei,will provide a basis on which study can advance from its present rather static basis. The division may be simple and based on known physiological facts, visual agnosia, auditory agnosia and tactile agnosia, on the receptive side, and apraxia (motor) of speech on the expressive side, with an "association aphasia" related to the old amnesia aphasia or formulation aphasia, as described by Nielsen.

III. METHOD OF EXAMINATION

The method of examination of aphasic patients that had been used routinely in the Montreal Neurological Institute was the modified Chesher tests. These were found to be inadequate as illustrated by one patient (E. Wa.) who was able to complete the Chesher tests rapidly and accurately, but when asked to repeat even fairly simple sentences would be unable to do so. Repeating, "Around the rugged rock the ragged rascal ran" , provided a stumbling block for two weeks after he was able to perform the modified Chesher tests correctly. Drawing from Head (1926), Nielsen (1936), Chesher (1937), Somberg and Ingham (1944) and Wells and Ruesch (1945), the method outlined below was prepared:

Examination of Aphasia Patients -

Name:
Case No.:
Age:
Date:
Examiner:
Service:
Handedness:
Language:
Diagnosis:
Operative procedures:

Objects used (two of each) -

Pencil, coin, match, key, spoon, scissors, pin, pen, button, thumb tack, paper clip, marble, comb, watch, toy shoe,

toy cat, toy telephone and paper cards on which are printed some of the above names, numbers, figures (geometrical and symbolic) sentences and letters.

General note as to general condition of patient, how he cooperates, spontaneous activity, speech and fatiguability.

1) Naming objects shown to patient.

2) Pointing to duplicate objects.

3) Pantomime - ask patient to illustrate use of objects.

4) Copying words, sentences, numbers.

5) Recognition of letters.

6) Recognition of numbers.

7) Recognition of symbols.

8) Recognition of geometric figures.

9) Copying of geometric figures.

10) Colours - a) pointing to colours.
b) naming colours.
c) matching colours.

11) Pointing to objects named.

12) Naming other sounds, e.g., keys, paper, bell, rattling of coins.

13) Response to question which will bring out emotional response.

14) Naming objects placed in hands - right and left.

15) Fine movements of hands.

16) Carrying out of commands.

17) Imitating examiner.

18) Completing complicated acts.

19) Repeating - a) words
b) sentences

20) Reading - a) words
b) sentences
c) paragraphs

21) Reading and explaining contents.

22) Silent reading and pointing to objects.

23) Spontaneous writing.

24) Writing from dictation.

25) Calculation, oral, written.

26) Repeating alphabet, counting.

27) Perseveration (pin, pen, pencil test)

28) Recognition of music.

29) Other tests as indicated.

In many cases in the postoperative state we were unable to examine the patients completely at one sitting, many visits would have to be made, and the test modified to suit the patient. The tests endeavoured to demonstrate the presence as well as the degree of agnosia, apraxia or aphasia. Reading, for example, would not only include reading of words, but also of paragraphs from magazines. No effort was made to make complete psychological studies as outlined in Weisenburg and McBride, as we were primarily interested in the anatomical approach. For future use, we were interested in having a series of tests which would enable us to classify the patient

as to the type of aphasia, and then as more cases were adequately examined and classified to see if the different types had any localizing significance.

In order to be able to say that a certain excision has not caused a specific defect in the intellect as well as in gross speech it will be necessary to use these more extensive psychological tests.

IV. CASES STUDIED, FINDINGS AND DISCUSSION OF RESULTS

A) Analysis of Patients:

The cases reviewed in this series were those in whom Dr. Penfield had performed a left-sided craniotomy because of seizures between 1930 and 1946 (there was one right-sided craniotomy in a left-handed patient). Many of these were discarded because of insufficient data, because no excision was carried out, or because the patient had been left-handed,and at no time was there any evidence of speech disturbance to suggest that the speech centers might have been partially on the left side of the brain. Fifty-one excision cases* were studied carefully. All had been suffering from epileptic seizures, and investigation included the usual general and neurological examination, lumbar puncture, pneumoencephalogram and, after 1938, electroencephalogram.

Age - The ages varied from 8 to 50 years, the average being 24. There were four under the age of 15, 39 between 15 and 34 years, 6 between the age of 35 and 40, one 45 and one 50.

Sex - There were 46 males and 5 females.

*Summarized case histories will be found in the Appendix.

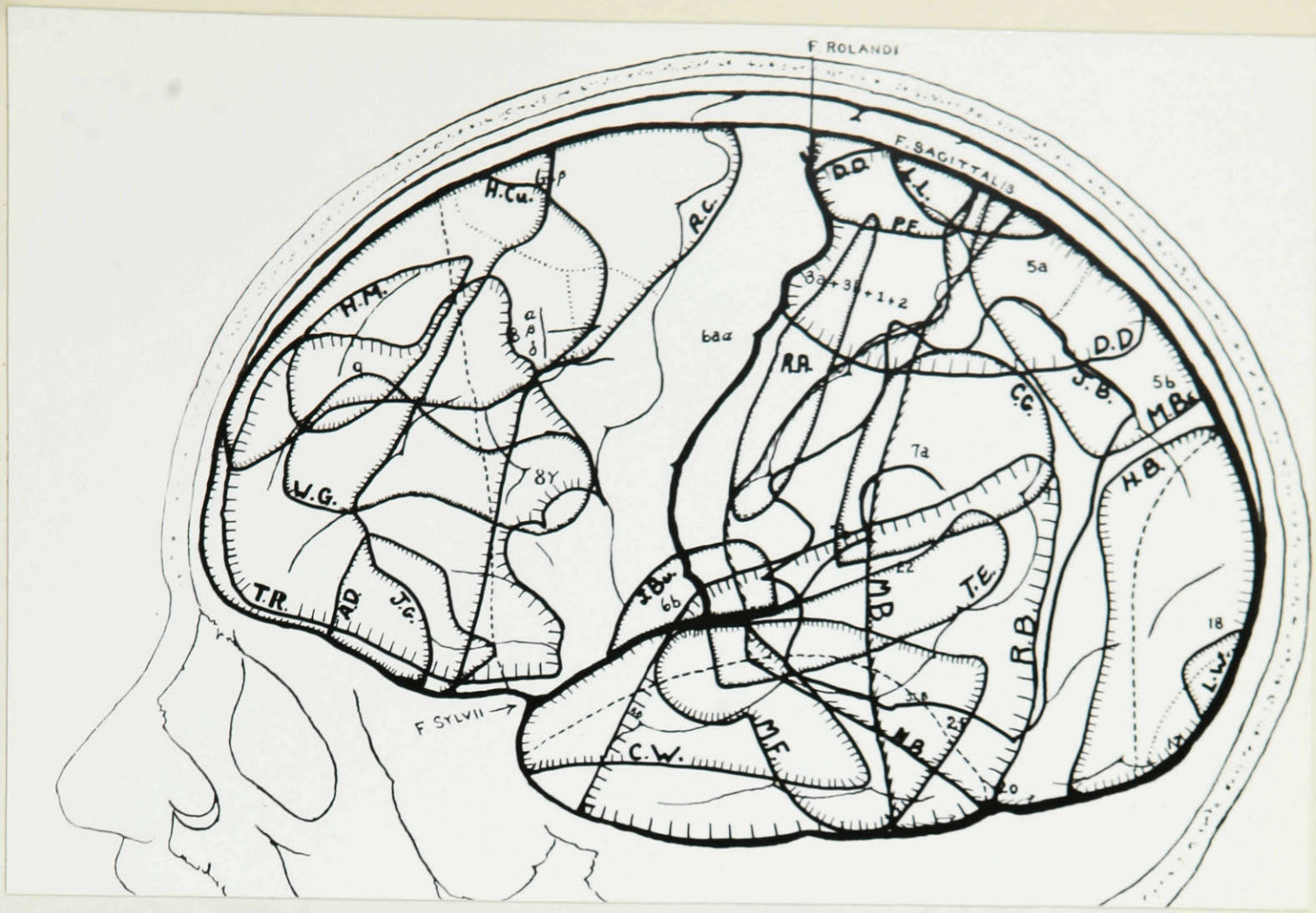

Fig. 4a. Outline of excisions from left hemisphere in right-handed patients who had a complete recovery.

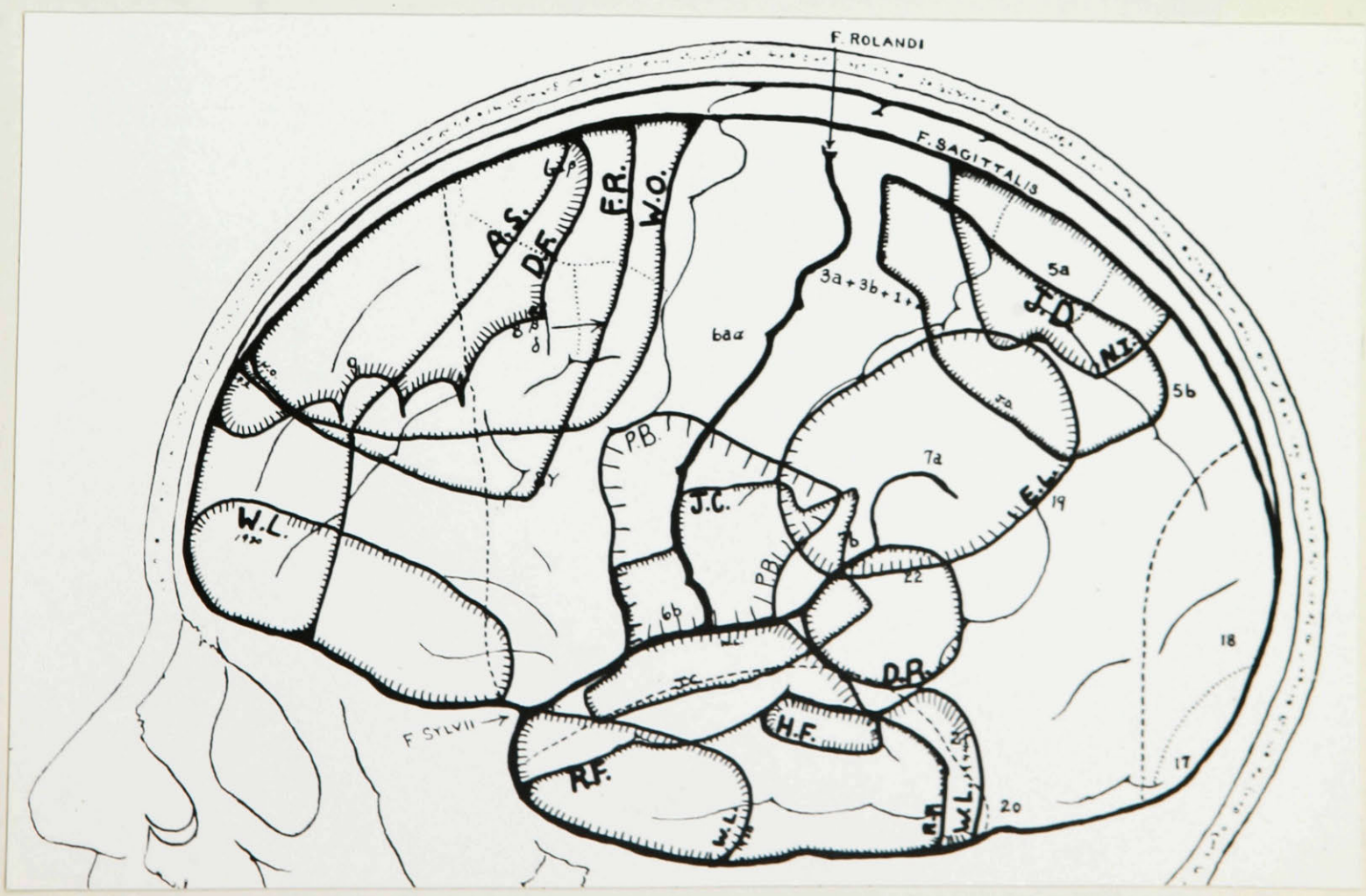

Fig. 4b. Outline of excisions from left hemisphere in right-handed patients who had a complete recovery.

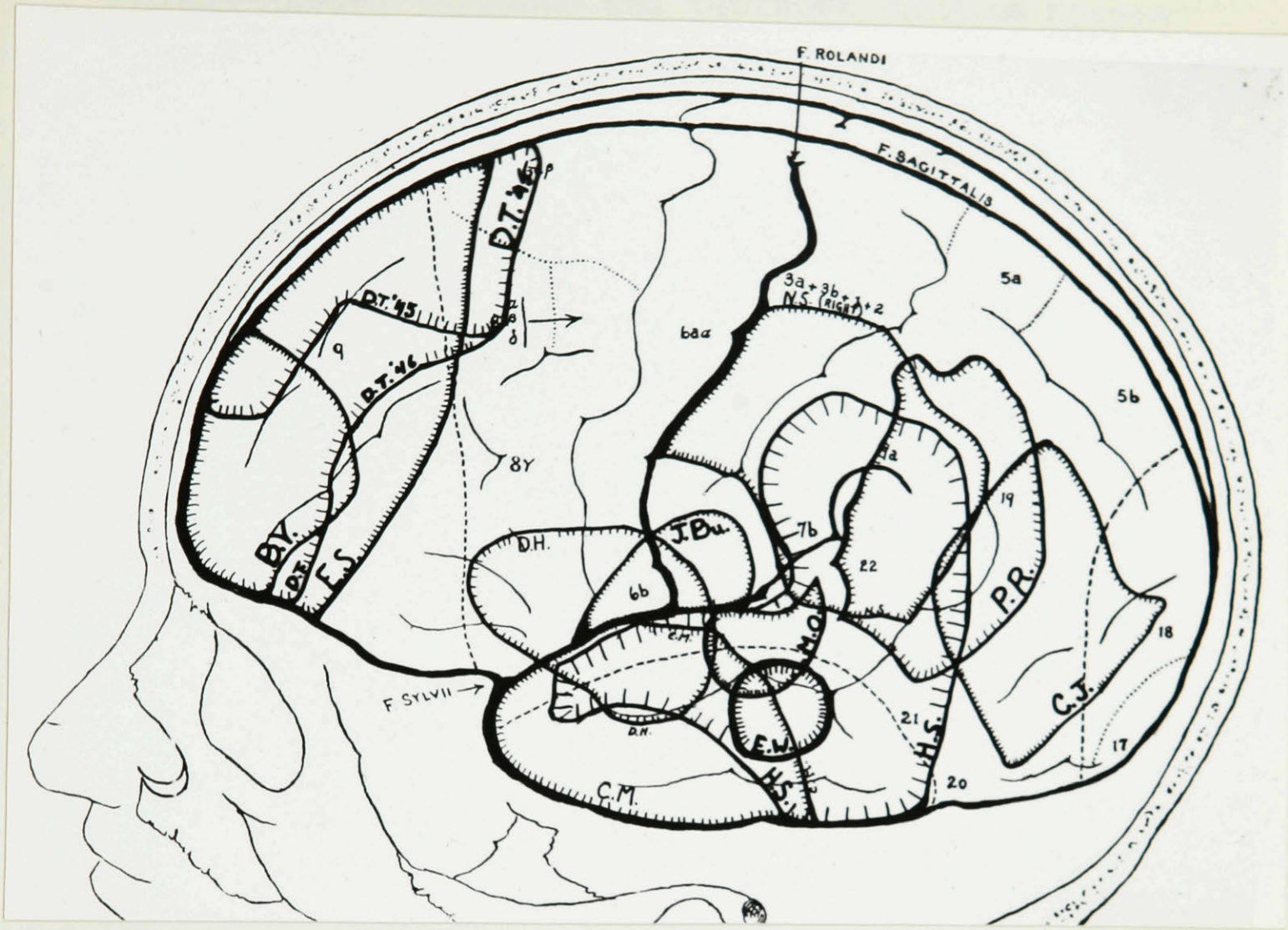

Fig. 4c. Outline of excisions from left hemisphere in right-handed patients who had a complete recovery. (N.S. was from right hemisphere).

CHART I

Right-handed patients and patients in whom speech was proven to be on the left side and on whom a left-sided excision was performed without causing a permanent aphasia:

R.A.	J.B.	M.B.	H.B.	N.B.	P.B.	R.B.	M.Be
J.Bu.	J.C.	R.C.	H.Cu.	A.D.	D.D.	J.D.	T.E.
D.F.	H.F.	M.F.	P.F.	R.F.	J.G.	W.G.	D.H.
N.I.	C.J.	E.L.	L.L.	W.L.	H.M.	C.M.	M.O.
W.O.	D.R.	F.R.	P.R.	T.R.	A.S.	E.S.	H.S.
D.T.	B.V.	C.W.	E.W.	L.W.			Total

Right hemisphere and left hand. N.S. 46

Patients who had aphasia or a speech disturbance at the time of injury, without producing a permanent aphasia:

J.B. R.B. J.Bu. T.E. W.G. W.L. M.O. H.S. N.S. 9

Patients who developed a transient postoperative aphasia:

Frontal:	H.Cu. D.H. F.R. E.S. D.T. E.Wa.	6
Parietal and Occipital	J.B. P.B. M.Be. D.D. J.D. N.I. C.J. E.L. L.L. D.R. P.R.	11
Temporal:	J.C. H.C. T.F. H.F. M.F. R.F. C.M. C.W. E.W.	9
		26

Patients in whom recovery in reading and writing was slow or never complete:

C.G. M.J. I.R. 3

Onset of original injury - There were 10 cases in which the cerebral cicatrix was felt to have been due to a birth injury, or to have come on before the first year and a half of life. There were 12 cases in which the time of origin was not known. Most of these twelve gave histories of normal births and multiple head injuries of unknown severity, not associated with any disturbance of speech. There were 3 cases (H.S., M.O., and T.E.) where the onset was before the age of six years, all of whom had a speech disturbance at the time of their injury. There was one patient who was injured at the age of 3 but no speech disturbance was known. There were 21 patients over the age of 6 years, in whom the brain damage followed a head injury. One case (H.C.) was supposed to have had encephalitis at the age of 33 years. One case (E.L.) had a vascular lesion at the age of 33. There were 2 tumor cases which were included because of unusual interest. One case (E.Wa.) had a glioma in the fissure of Sylvius opposite Broca's area, and one (D.H.) had a calcified astrocytoma beneath the cortex of Broca's area. In one case (C.M.) an excision was done for decompression purposes following a negative exploration.

Handedness - There were 36 who were right-handed, 2 were left-handed and 10 were ambidextrous. Some of these had been originally left-handed and had switched to the right, or some changed from right to left following the injury, others wrote with one hand and played games and other activities with the

other hand. In 3 cases the handedness was not recorded, but in these there was evidence that speech was on the left side of the brain. In none of the left-handed patients was there sufficient evidence to say that speech was entirely ipsilateral. Here was one left-handed patient in whom speech had been proven to be on the right side originally.

B) Operative Procedures:

With minor variations this was the same in all cases. A left osteoplastic craniotomy was performed by Dr. Penfield. Local anaesthetic was used for the first part of the procedure. Frequently, just before the actual excision was carried out, and when the patient's cooperation was no longer needed to determine what area to remove, a general anaesthetic would be given. This was usually avertin. For this reason in most cases it was impossible to say if there were any immediate changes in speech following the excision. After 1938, following the turning down of the bone flap, electroencephalographic recordings would be made from the surface of the dura, the dura reflected and further recordings made. Thus, the area producing the abnormal electrical potentials was more accurately localized. The cortex was then stimulated electrically and the patient's responses recorded. The technique has been described elsewhere (Penfield and Erickson, 1941). In this way the central fissure was accurately localized.

From the information obtained from a former pneumoencephalogram, the electroencephalographic recordings from the cortex, and the visible pathology, the area to be excised was outlined, and removed either in a block or by suction. Great care was taken to leave a wall of healthy cortex covered by pia mater in order to prevent the creation of another epileptogenic focus. Photographs were taken at various stages of the procedure. Following the excision, the area removed and the stimulation markers were drawn onto a previously prepared brain chart.

The difficulty of knowing exactly what area was being removed was overcome somewhat by the electrical stimulation and outlining the central fissure. Although great care was taken to accurately place the area excised on the brain chart, due to the difficulty of transferring a curved surface to a flat one, and the normal differences in length and width of brain, minor errors must be expected. In this study the area drawn on the chart was considered to be the area excised, and the photographs were used for confirmation of the drawing only.

C) Effect of Excision of Cortex and Cicatrix:

1. Postoperative aphasia.

It soon became apparent, as the study progressed, that three things could happen to speech after excision of damaged

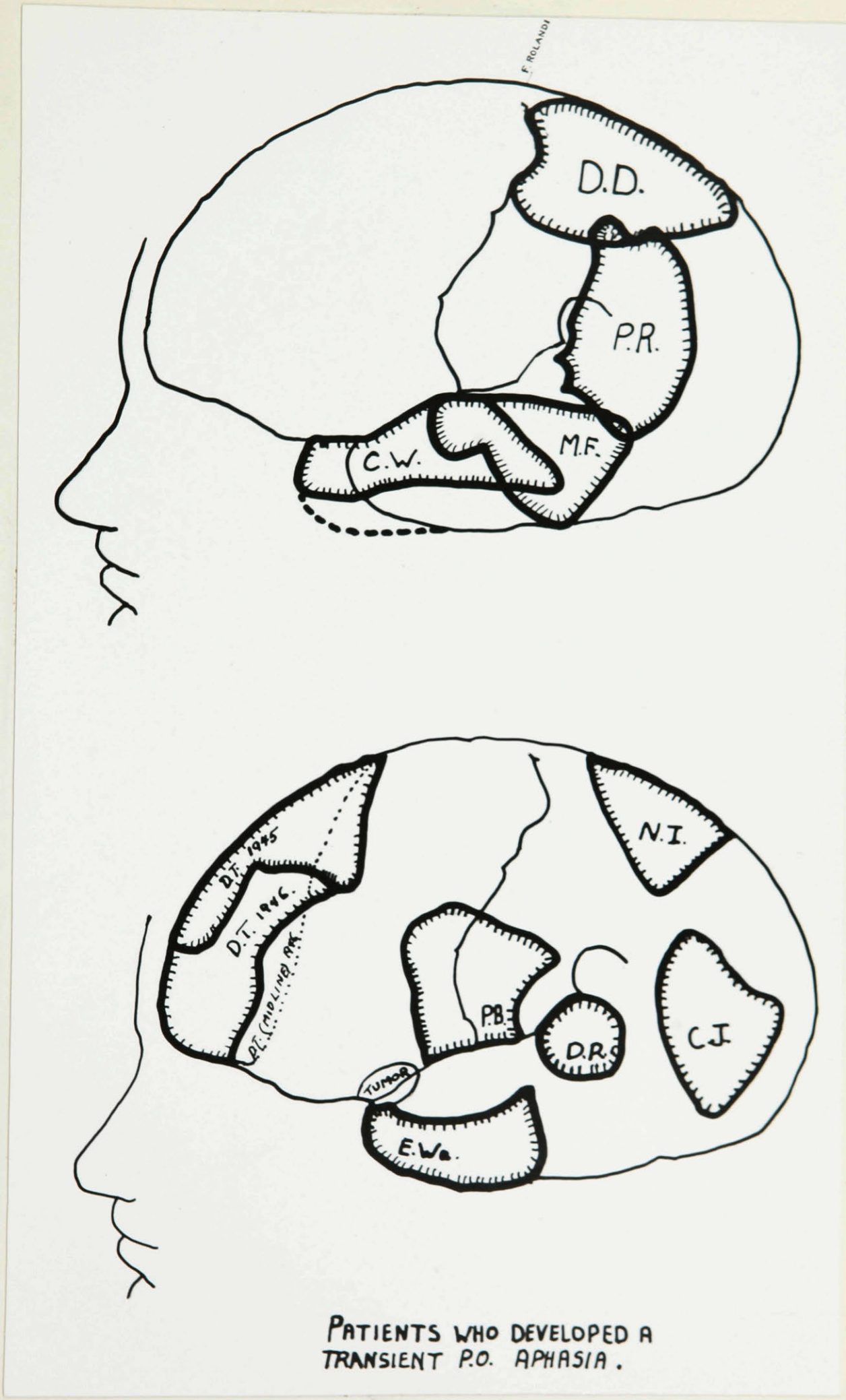

Fig. 5a. Outline of excisions in patients who developed a transient postoperative aphasia. (see Fig. 5b also).

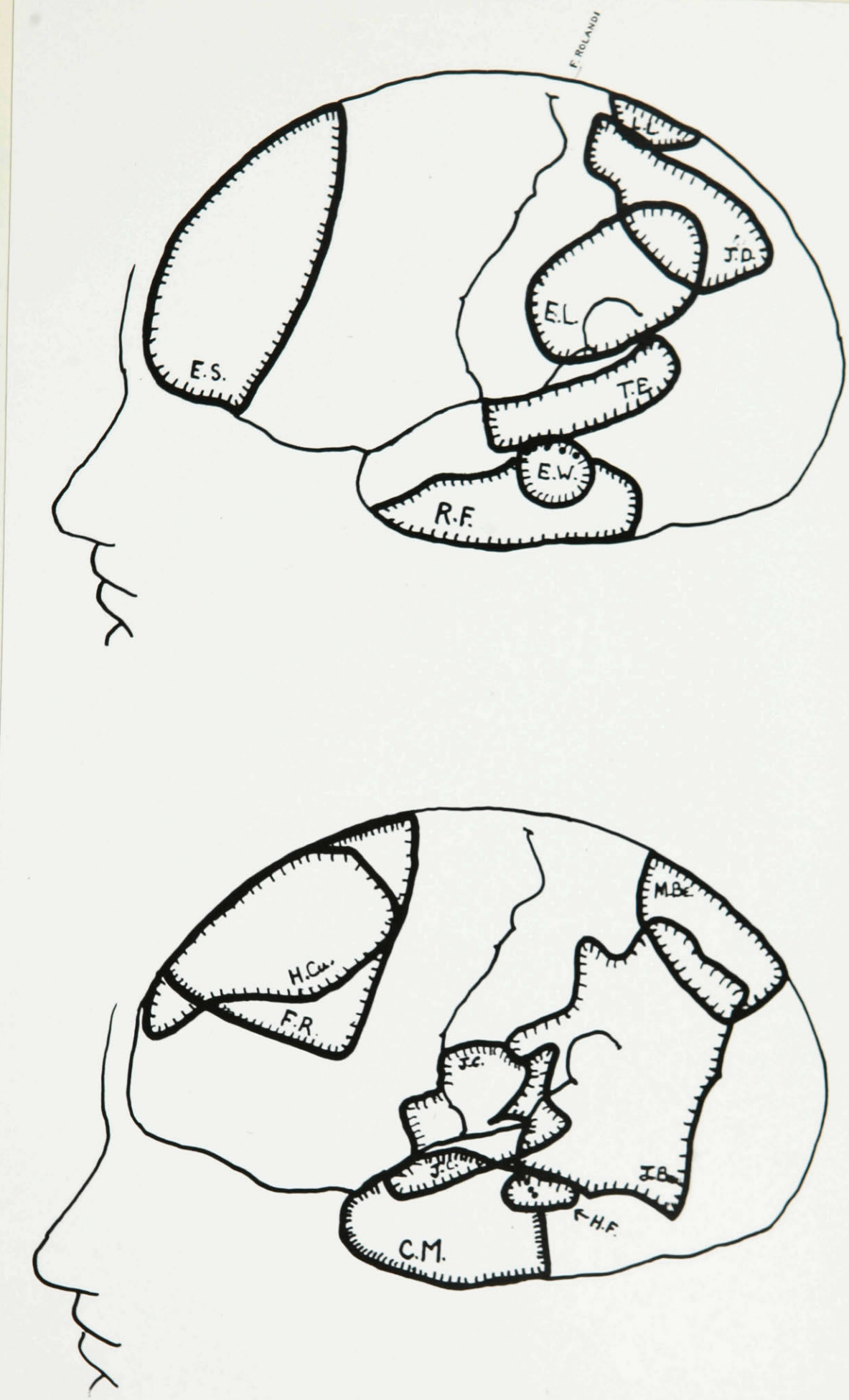

Fig. 5b. Outline of excision in patients who developed a transient postoperative aphasia. (see Fig. 5a also).

cortex from the major hemisphere. First, the patient could develop an aphasia at the time of the excision. Secondly, he could develop an aphasia one or more days after the excision, or thirdly, he could be completely free of any aphasia following the procedure.

The first, coming on at the time of the excision, was due to the removal or damage of cortex, which up to the time had been functioning for speech. It was seldom seen, as most of the cortex removed had not functioned since the injury and as every effort was made to avoid removing known speech areas that might still be functioning.

As a general anaesthetic was usually given before the excision was completed, the patients were seldom tested on the table, and by the time that they had recovered one could not distinguish the first from the second type. Two cases were worthy of note. The first, H.C., a male aged 45, left-handed, was suspected of having a tumor in the left temporal region. A left osteoplastic craniotomy and exploration was carried out by Dr. Penfield. As no superficial tumor could be found the fissure of Sylvius was opened by careful dissection. At the depth of the fissure the first temporal gyrus was injured to some extent. It was observed,during the latter part of the procedure,that the patient seemed to become hard of hearing and the anaesthetist had difficulty in making him understand the spoken word. He had developed an auditory agnosia.

The only damage done to the cortex was to the first temporal convolution deep in the fissure of Sylvius. On the first postoperative day he was unable to understand anything that was said to him, no matter how loud, but he was able to read sentences clearly and to understand them. Recovery seemed to be complete in two months. The second case of this type was P.M., aged 35, who was operated on because of seizures originating in the left temporal region. Immediately following the excision of most of the temporal lobe, he was able to talk, but he could not name anything. There was very poor attention and he was drowsy, having had a grain of codeine, which complicated the picture. However, the fact remained that when aroused he would not name anything. On the first and second postoperative days he was able to speak but could not name objects. The aphasia then became complete, and after a few days began to subside. At the time of writing, four weeks postoperatively, he was able to talk, demonstrate the use of objects, and read and write. There was some difficulty in comprehension of the spoken word and there was some difficulty in naming objects. He showed evidence of fatigue.

The second group were those who developed a secondary type of aphasia, undoubtedly due to a temporary loss of function of brain surrounding the excision. There were 26 of them. Six were removals in the frontal area, 11 in the parietal and

occipital areas, and 9 in the temporal area (Figs. 5a &.b). There was always an interval(between the operation and the onset of the speech defect)in which the speech was normal, or almost so. The type of aphasia varied somewhat with the location of the excision, but for the most part it was a mixed expressive and receptive type, and changed rapidly. This postoperative aphasia was important in that it established the fact that the side operated on was the major one for at least part of speech. In most cases the aphasia could be accounted for by local oedema as the excision was so close to the surrounding speech areas. Recovery was always complete.

There were 5 interesting cases in this group, (M.Be., J.D., D.D., L.L. and N.I.) in which the excision was in the postcentral region, near the midline and local oedema did not seem to be an adequate explanation. It may have been that association tracts were affected by oedema, or the blood supply of more distant areas had been disturbed.

In 7 cases (J.B., J.C., T.E., M.F., E.L., P.R. and D.R.) known speech areas were removed and they developed a secondary type of aphasia. The fact that it did not come until a day or two after the operation would prove that the part excised was not functioning for speech at the time, but the hemisphere still remained active for other parts, if not all of speech. Although they developed a postoperative aphasia and proved

that at least some speech was on the side of the excision, it did not prove that all speech was on this side, or that some part of the opposite hemisphere was not also functioning for speech. In view of the fact that the excisions, with one exception (M.O.) were so large, and that they collectively included all of the temporal lobe and angular gyrus, the possibility of contiguous areas at any area of the same hemisphere seems very remote.

The third group were those who were free of aphasia after the excision. In many cases the explanation was obvious. Speech areas were not involved. In others, speech areas may have been ipsilateral to the dominant hand. However, there was a group in which known speech areas were excised where there was aphasia following the initial injury. Two explanations were possible - either contiguous cortex had taken over or the opposite side had assumed the function of the damaged area following the initial injury. If contiguous cortex had taken over one would expect a postoperative aphasia since some normal tissue was always removed to leave a wall of normal brain. If, on the other hand, the opposite side had assumed the function of the damaged cortex, one would not expect a secondary postoperative aphasia. This latter explanation would account for the absence of postoperative aphasia in certain cases (R.B., C.G., W.L., M.C., H.S. and N.S.).

A third possibility should be considered. In these cases a completely different and distant area of the damaged hemisphere might have assumed the function of the damaged cortex. The fact that cases have been reported (Nielsen, to be published) in which the patient developed a lesion on one side, became aphasic, recovered and later had a lesion of the same area on the opposite side and again became aphasic, without recovery, would argue against this. Also, the modern work on the precentral motor cortex and the thalamic projection areas of the cortex would make this explanation most unlikely and most unphysiological.

There were 51 excision cases studied. Of these there were 46 who were right-handed or in whom speech was proven to be on the left side of the brain and in which excision was carried out without causing permanent aphasia. In some of these right-handed patients there was no history of aphasia at the time of injury, and no postoperative aphasia. In these cases one has no definite proof that speech was on the left side of the brain. However, as the evidence presented by Nielsen (to be published) strongly suggests that there is always a motor speech area opposite the hand with which the patient writes, these cases were included. The outline of the areas excised have been placed on a master chart, in order to illustrate which areas have been removed without producing permanent aphasia (Figs. 4a, b & c).

There were 9 cases who gave a history of having been aphasic after a head injury and had recovered. Only two (T.E. and J.B.) showed any postoperative aphasia.

There were 32 cases in which, at least, part of speech was proven to be on the left side as a result of an aphasia at the time of injury or a transient postoperative aphasia. In these excisions were carried out without producing a permanent speech defect.

2. Relation to age at the time of injury.

The age of the patient at time of the initial injury was considered to be a factor of major importance. The operative excision, for the most part, served only to outline accurately the area destroyed or damaged. In many cases functioning brain was excised in order to leave a healthy line of excision, or because abnormal electrical potentials were coming from it.

The study showed that in right-handed patients the temporal, parietal or occipital lobes, damaged at birth or during the first year and a half of life, could be excised without fear of producing a permanent aphasia. As there was no proven case in which Broca's area was excised in right-handed patients, we are unable to say what effect this would have. The motor speech area is so close to the hand area,

that patients in whom this region was damaged at birth became left-handed, even though for hereditary reasons one would have expected them to have developed as right-handed individuals. One surprising thing was the number of patients who remained right-handed in spite of a very large amount of left-sided brain damage. Cases N.B. and M.B. were outstanding examples.

In view of the fact that in cases of infantile hemiplegia, the opposite side is known to take over the function of speech without difficulty, one would think that when speech areas are involved in birth injuries, the corresponding area of the opposite side would function. This would be supported by the small number of cases injured at birth that became aphasic after operation. However, cases P.R. and C.J. showed that speech need not be taken over entirely by the opposite side, as they developed a transient postoperative aphasia.

In this series, known speech areas damaged for one reason or another, were removed without producing a permanent aphasia or making existing speech disturbances worse. However, it did not prove that the excision of poorly functioning damaged brain facilitated the recovery from an existing speech disturbance.

3. Excisions of the left temporal lobe.

Perhaps the most practical finding was the fact that so much of the temporal lobe could be excised without producing a permanent aphasia. The outline of the excisions of all cases that had been aphasic after the initial head injury were superimposed on a master chart and it was shown that all of the temporal lobe and most of the parietal lobe, including the angular and supramarginal gyri, could be removed from the major hemisphere without producing a permanent aphasia. There were 9 excision cases who had been aphasic after a head injury and who recovered completely. The average age at the time of injury was 16.7. Cases R.B., N.S., J.B., W.L., and H.S. were of particular interest because of the large area excised. Nielsen has reviewed all of the reported cases in which the major temporal lobe was removed and found that complete recovery can occur but that it might take as long as two years. In our cases the brain had been damaged previously and we were unable to say how long recovery took. However, it was our impression that it was done in a much shorter period. This may have been related to the comparatively young age of our patients.

As it is frequently desirable to remove the anterior end of the temporal lobe for decompression purposes, or to expose a deep seated tumor, the fact that it can be removed freely,

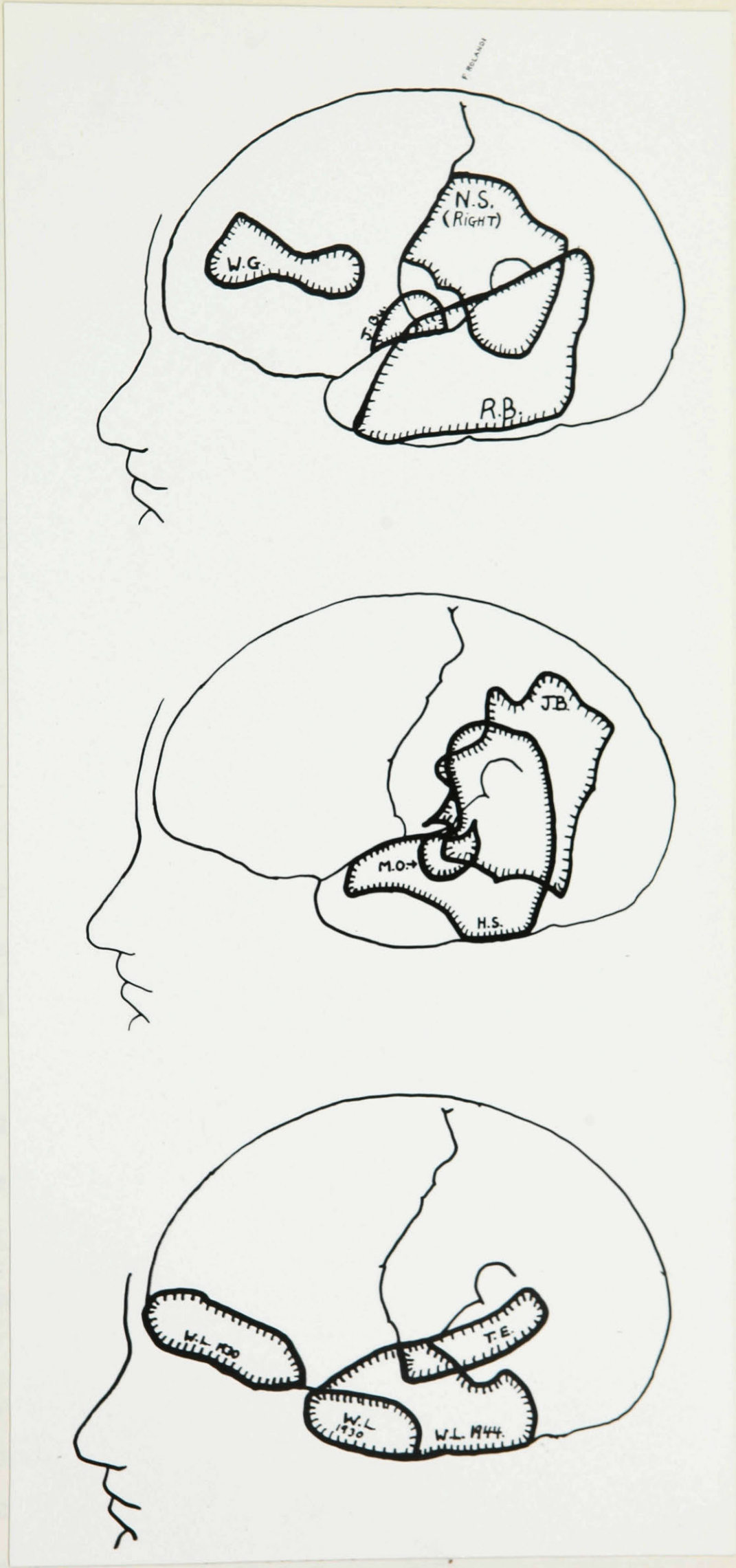

Fig. 6. Outline of excision in patients who were aphasic following their initial injury.

without producing a permanent aphasia, is of considerable importance. In the case of C.M., 4 cm. of the anterior end of the temporal lobe were excised to provide an internal decompression. He had developed aphasia following an exploratory craniotomy and it was necessary to reopen to provide further decompression. He recovered completely.

4. Left-handed patients with ipsilateral speech centers and the role of the minor hemisphere in assuming the function of the major.

Nielsen (1937, 1938) has presented evidence that conversion of a left-handed person to a right-handed establishes an artificial writing center on the minor side. Although this is possible it does not necessarily happen. Gardner (1941) reported a case of a strongly left-handed individual in whom he removed the right hemisphere without producing any aphasia. However, it was noted that three years later he still had not trained himself to write with his right hand. It could have been possible that writing was the only part of speech carried on the right side, but this seems hard to believe. Nevertheless, the case proved definitely that all of vocal speech can be ipsilateral to the dominant hand. In other reports, the evidence is only suggestive. Case H.C. as far as could be determined, was completely left-handed and yet he developed a marked auditory agnosia which progressed to a jargon aphasia, and almost complete loss of understanding of the spoken word. Case E.Wa. wrote and ate with his left hand, but played some games with his right. He had a partial removal of a tumor

from the left fissure of Sylvius, after which he developed a secondary mixed type of aphasia. There was no way of telling, in these two cases, whether the other side was functioning as well. Case J.B. was of paramount importance and proved that in his case motor speech was in the left hemisphere, opposite to the writing hand, and receptive speech was in the right hemisphere. He had the isthmus of the temporal lobe excised, as well as the posterior superior part of the temporal lobe, most of the parietal, and part of the occipital lobe, leaving him hemianoptic and separating association fibers of the remaining portions of the temporal and occipital lobes from the rest of the hemisphere. Following the initial accident he had had difficulty with speech which came back by degrees. Two days after the operation he had some slurring of speech and he groped for words, showing that there was still some motor speech on the left side. By the sixth postoperative day his speech was considered normal.

Cases J.C. and T.E. had their primary and secondary hearing areas excised on the major side. In both it was felt that the areas removed could not be functioning and yet the patients developed secondary transient aphasia. In both, the minor side must have been functioning for the perception of sound but the major, left side, still functioned for other phases of speech. If this were not so they would not have developed a secondary postoperative aphasia. Nielsen has

stressed the fact that either hemisphere can be the major hemisphere for different phases of speech. Our findings would agree with this. One can say, from the cases studied, that the function of the primary and secondary hearing centers are easily assumed by the opposite side. Whether higher association centers are as easily transferred is not as certain.

5. Excision of motor speech area.

Although no definite excisions of Broca's area were carried out for the treatment of post-traumatic epilepsy, one tumor case should be mentioned. D.H., a young man of 20 had had seizures since the age of 3 years. Plain x-rays demonstrated a calcified lesion in the region of Broca's area which at operation proved to be an astrocytoma. The tumor was first exposed in 1932 by an incision through the posterior (lower) end of the third frontal convolution and the calcified mass was evacuated through the incision without greatly damaging the cortex. Following this he had an expressive aphasia which lasted 30 days. A second craniotomy was done in 1942 and this time the cortex, along with the tumor, was removed from the region and there was no postoperative aphasia. So much of the area had been disturbed by the procedure that if the left side had still been functioning, it was felt there would certainly have been some aphasia. The evidence in this case

was very strong in favor of the fact that the minor side had taken over the function of the major one for motor speech after the first operation.

6. Cases of incomplete recovery.

There were no cases in which the excision made patients worse. There were three cases in which recovery of the ability to read and write had never been complete following the head injury. The first one, I.R., at the age of 35 years had sustained a severe laceration of the brain in the left parietal region following which he developed a complete loss of speech and weakness of the right hand. The speech defect recovered rapidly and at the time of admission, a year and a half later, the speech was considered normal. However, he read very little and did not write. A large area, including the angular gyrus, was excised. Immediately following operation, his speech was considered normal. Writing and reading were not tested. On the first postoperative day he developed a mild aphasia which subsided rapidly. The difficulty with writing, and to a lesser extent, reading, persisted. Eight years later he was able to read the newspaper but he did not do it as well as he had before the injury. He would not write, and it is difficult to say how much he could have recovered if he tried. He was a farmer and there was little occasion for him to write. Once his son wrote from overseas and made a special request that his father write to him. With great effort, and by getting away alone, he was able to write a few lines to the son.His wife stated that some days he is able "to write and read, etc.,

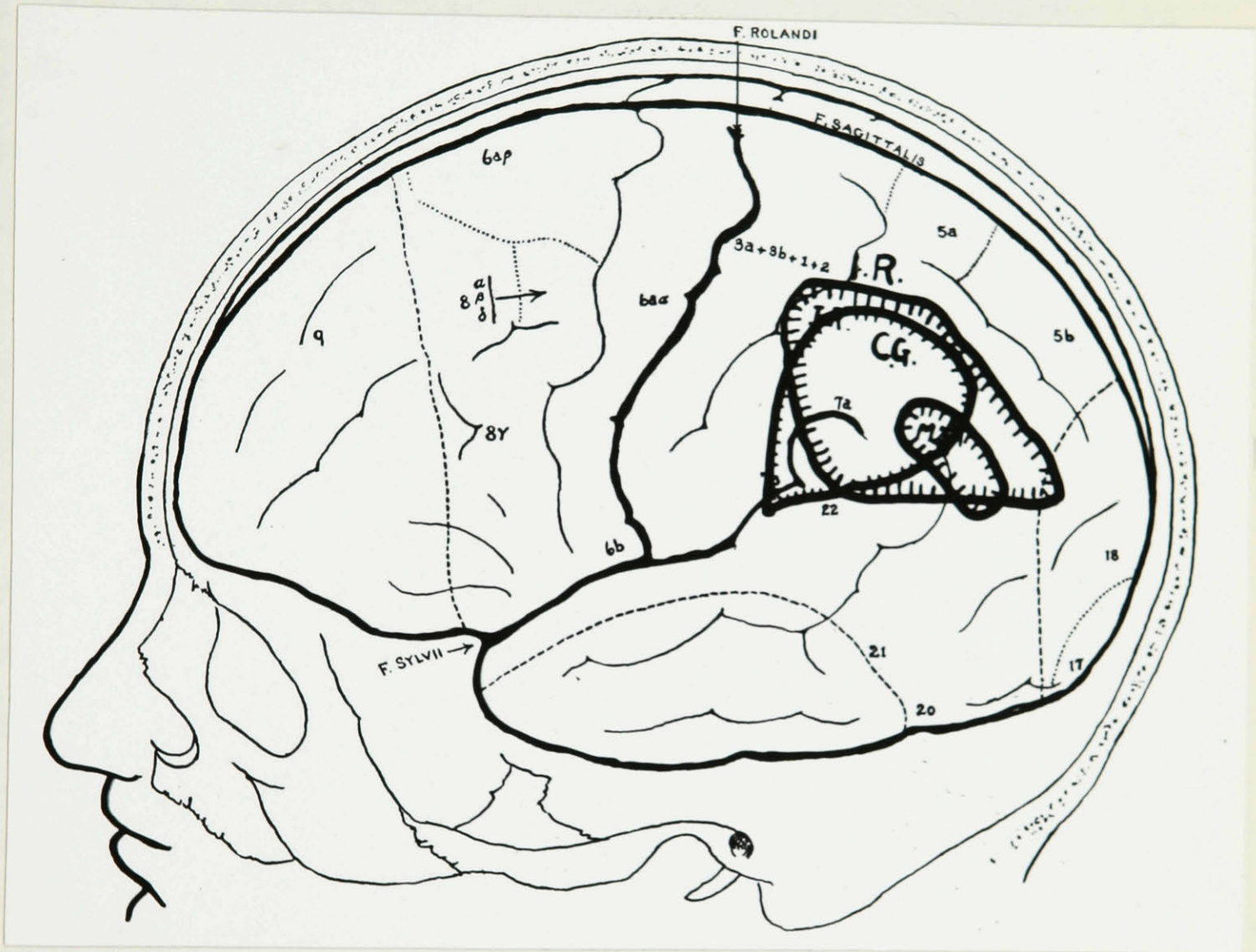

Fig. 7. Outline of excision in patients who had an incomplete recovery following the initial injury.

much better than others and some days it is an effort just to write his own name."

The second case with incomplete recovery was C.G., a man aged 38, who had been profoundly aphasic following the initial injury at the age of 25 years and also following a craniotomy done in 1932. On examination, at the time of admission in 1939, he had trouble writing and as a result wrote very little. He could read the newspaper but made occasional mistakes with more difficult words. His spontaneous speech was considered normal. The chief disturbance was in the field of written language. Following a much more extensive excision than had been done before, there was no change in his speech that could be detected. Nine months later it was felt that he had improved. He could write simple sentences and could read, making fewer mistakes.

The third case, M.J., is recorded for completeness sake. He had a small excision from the major angular gyrus which was followed on the second day by a secondary aphasia. At the time of discharge spontaneous speech was considered normal and there was no record as to whether he could write. A year later a friend wrote saying the patient could not write. As there is no evidence as to whether he ever could write, the case is of little value. I have been unable to trace the patient for further follow-up.

D) Effect of Stimulation of Cortex on Speech.

At the outset of the study it was hoped to find a method of temporarily paralysing the function of a small area of the cortex in order to determine whether it still functioned for speech. Injection of local anaesthesia was tried in three cases, but the method was abandoned as the results were negative and Gardner (1941) showed that the injection formed small cysts, rather than diffusing, throughout the brain.

In the same way as electrical stimulation of the cortex was used to produce simple movement and sensation, Dr. Penfield was able to temporarily block cortical function. By stimulation of various points on the cortex he was able to arrest counting. Case P.M. was of particular interest. Before excision of the major temporal lobe the patient was asked to count, name objects and read during stimulation. When the lower precentral region was stimulated, counting would cease after one or two numbers, and then the patient would be unable to continue until stimulation had stopped. When the supramarginal gyrus was stimulated he was able to say syllables like "Ka, Ka", when trying to name an object. He said numbers but neither the pronunciation nor the order were accurate. During stimulation of the posterior temporal region, in the vicinity of the angular gyrus, he was unable to name objects. He could say, "That is a ....." but he could not complete the sentence with the key word. Stimulation at some points in the vicinity of

the angular gyrus arrested reading. At one point he was able to name an object after some deliberation but was unable to read. There were other points further away from the angular gyrus in which reading, naming of objects and counting were not affected by stimulation.

To date the controls have not been adequate. In reviewing the stimulation records of Dr. Penfield's excision cases, Rasmussen (1946) found that counting can be stopped by stimulation of the sensory motor strip almost equally on both sides. Speech had been stopped on the left side by stimulation of areas outside the pre and postcentral gyri, but not on the right. No conclusions can be drawn, as not enough effort has been made to stop speech on either side outside of the sensory motor cortex. The possibility of arresting speech by stimulation of suppressor areas must also be considered (Garol, 1940).

E) Cortical Localization of Types of Aphasia.

Localization studies were of little value. The cases reviewed did not have sufficiently detailed examinations recorded to adequately classify the type of aphasia. Also, as the initial injury had occurred so long before the excision, other areas had assumed the function of the damaged area, and the excised area for the most

part was not functioning for speech. Case H.C., however, was important. Although his case was complicated by the fact that he was left-handed, nevertheless his left hemisphere was essentially normal at operation and damage to the first temporal convolution, deep in the fissure of Sylvius, produced almost a pure auditory agnosia. There were several cases which tended to confirm Nielsen's view that area 37 produced an amnesic, or what he called, formulation aphasia. The most persistent speech defect in patients who had the posterior inferior part of the temporal lobe damaged, or excised, was this amnesic aphasia. Case P.M. was able to read, hear and understand the spoken word long before he was able to name a simple object, such as a pen or a pipe. He would casually say, "Pass the ash tray please", but was unable to name this object when it was presented to him. Case C.M. had 4 cm. of the temporal lobe excised and his aphasia passed off in a few days after the operation, whereas in the case of P.M., 7 cm. were removed and the amnesic aphasia persisted for several weeks.

The three patients in whom there was incomplete recovery of the ability to read and write had lesions in, and around, the angular gyrus (C.G., M.J., I.R.).

F) Stammering in Patients Following Recovery from an Aphasia.

There were three patients who had sustained extensive injuries to their major hemispheres with a resultant aphasia that recovered, but who were left with a definite stammer or hesitancy in their manner of speech. Patients R.B. and W.L. had most of the left temporal lobe excised and the patient N.S., who was left-handed, had a large area removed from the postcentral region, including the angular gyrus and the posterior part of the first temporal gyrus. Each developed stammering speech which persisted as they recovered from the initial aphasia. They all retained their original handedness after the accident. Following the excision they were all free of any aphasia. This stammering may be a common finding in patients who have had a major transference of speech centers, and should be watched for in future cases. It may be akin to the stuttering seen in children who are made to write with the minor hand.

V. SUMMARY

1. A method of examination of patients with aphasia has been prepared in order to classify the type of aphasia from which they suffer.

2. Fifty cases of excision of previously damaged cortex from the left hemisphere in right-handed patients or patients in whom part of speech was proven to be in the left hemisphere were reviewed. One case of excision, from the right hemisphere in a left-handed patient in whom speech was proven to have been in the right hemisphere, was also studied. The average age of these patients was 24 years.

3. There were 32 patients in whom at least part of speech was proven to be in the hemisphere operated on, either because the patient had been aphasic following the initial injury, or because he developed a transient postoperative aphasia. All of these patients recovered completely. There were three patients in whom the ability to read and write had never returned to normal, following a brain injury. The excision of damaged brain did not change them or make them worse. In all three the excision was in the vicinity of the angular gyrus.

4. There were 26 patients who developed a transient postoperative aphasia which for the most part came on a day or two after operation. Most of them could be accounted

for by oedema and temporary loss of function of nearby speech areas. In five of these cases the excision was in the parietal lobe near the mid line, and no adequate explanation could be given for the aphasia.

5. By superimposing the excisions of the major temporal lobe it was found that it had been removed completely without producing a permanent aphasia. In one patient it was removed completely and in others almost completely without producing a permanent aphasia. When 4 cm. from the tip posteriorly were excised the postoperative aphasia was only very transient.- When 7 cm. were excised the postoperative aphasia was more persistent and consisted mostly of an amnesic aphasia and an auditory agnosia.

6. When the primary and secondary hearing centers were excised from the major transverse temporal and first temporal convolutions, recovery took place as a result of the opposite side assuming the function completely, and the patients were able to hear and understand. Whether the minor side functioned in other phases of speech, such as reading, writing and motor speech, when the major area was excised, was not proven conclusively. However, the evidence was strongly in favor of this being the means of recovery.

7. It was proven that all phases of speech need not be in the same hemisphere. Motor speech could be contralateral

to the dominant hand, and receptive speech, all, or in part, could be ipsilateral.

8. The number of cases in which some part of speech was proven to be ipsilateral to the dominant hand emphasized again the importance of not assuming speech to be opposite the dominant hand.

9. There were no cases to prove or suggest that excision of a partially functioning damaged area in an aphasic patient facilitated the recovery from the aphasia by allowing the opposite side to assume complete control.

10. For reasons explained above, the study was of little value in localizing the various phases of speech to areas of the cortex as they function normally. One patient with a normal hemisphere, in whom the first temporal convolution was traumatized deep in the fissure of Sylvius during an exploratory craniotomy, developed an auditory agnosia. The most common symptom in a patient with a lesion of the posterior inferior part of the temporal lobe was an amnesic aphasia. Patients with persistent reading and writing defects had lesions of the angular gyrus and its vicinity.

11. The work done to date by Dr. Penfield on the paralysing effect of an electrical stimulation of the cortex on speech was reviewed. As controls had not been adequate, no conclusion as yet could be drawn.

VI. COMMENT

This has been a study of the effect of cortical excisions on speech in patients with previous cerebral injuries. Every effort has been made to present the findings as objectively as possible and not to draw any conclusions unless the evidence was beyond question. In all of the cases here studied, the recovery of speech was complete or almost so. There were no patients left with an inability to speak. From this it must not be concluded that all cases recover from aphasia. Two patients were known to the author in whom there was profound damage to the left hemisphere, including the basal ganglia and the thalamus, who over several years never recovered the ability to say a word. In one the amount of damage was determined by a pneumogram, and one could not be certain that there was no damage to the opposite side. In the other the right side was found to be normal at autopsy. In both these cases the defect of speech was almost completely a motor one. They both had a remarkable ability to understand and communicate by gestures.

In the cases studied almost all of the damage was confined to the cortex and the white matter immediately below. Whether their recovery was related to the fact that deeper structures were not involved was not determined. The part which the thalamus plays in speech is almost unknown.

It need hardly be said that this work is incomplete. Until there is more knowledge of the actual physiology of speech, any work on aphasia will be incomplete. It would be better to say that this is to provide the background for further work and research on the problem.

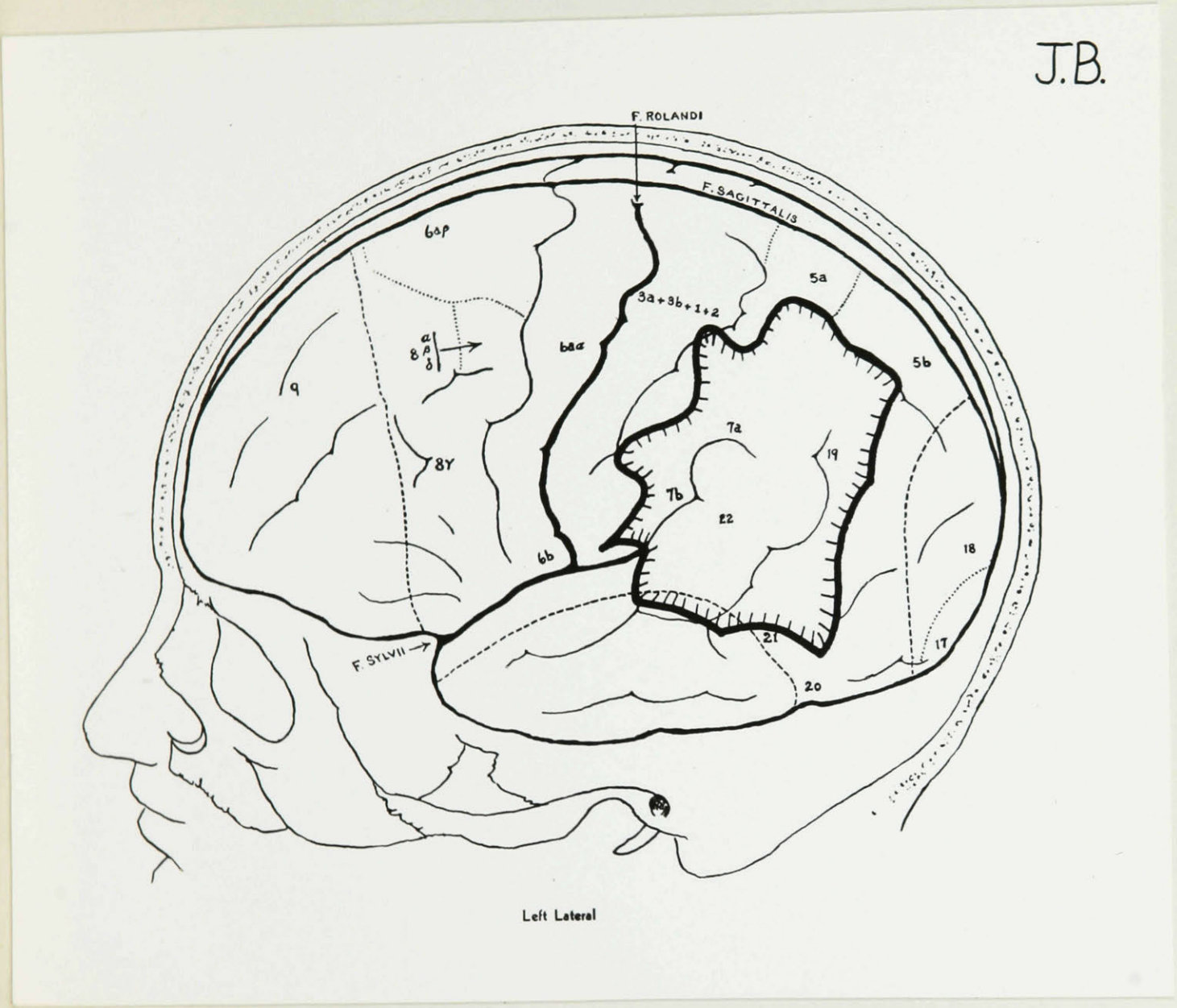

Fig. 8. Case J.B. Excision of an area in a right-handed patient in which part of speech was proven to be in the left hemisphere.

VII. APPENDIX

I. Summarized case reports of important cases:

Case J.B., male, aged 16, was admitted to the Montreal Neurological Institute on January 14th, 1940 because of Jacksonian seizures. They had started at the age of 12 years, 2 years after a depressed skull fracture in the right frontal and left parieto-occipital region. Following the accident, he said, "I had a little difficulty with speech, but this came back by degrees." He was right-handed, wrote with his right hand, but there was a slight weakness of this hand. His speech was considered normal. There were no sensory differences. X-rays of the skull showed an osseus defect in the left parietal bone, with some intracranial bone lying beneath the superior margin of the defect. Pneumoencephalogram showed a moderate dilatation of the lateral and third ventricles. Electroencephalogram suggested a deep lesion in the left posterior temporal region.

On December 17th, 1940, Dr. Penfield performed a left osteoplastic craniotomy under local anaesthesia. He found and excised a cerebral cicatrix including the posterior portion of the temporal lobe, the anterior portion of the occipital lobe, and the inferior part of the parietal lobe. The scar tissue extended down to the wall of the inferior horn of the lateral ventricle. This was not opened.

On the night of the operation the patient responded well. He had a definite right hemiparesis but there was no evidence of any motor aphasia. Two days later he showed a right homonymous hemianopsia, slurring of speech and he groped for words.

On December 23rd, the sixth postoperative day, Dr. Penfield noted that he talked perfectly, and yet had a complete cortical sensory loss for the right hand, and the movement of the right hand was very limited. At the time of discharge, the twenty-fifth postoperative day, there was no evidence of aphasia.

Six years later he still had difficulty with fine movements of the right hand and he had learned to write and eat with his left hand. His speech was normal.

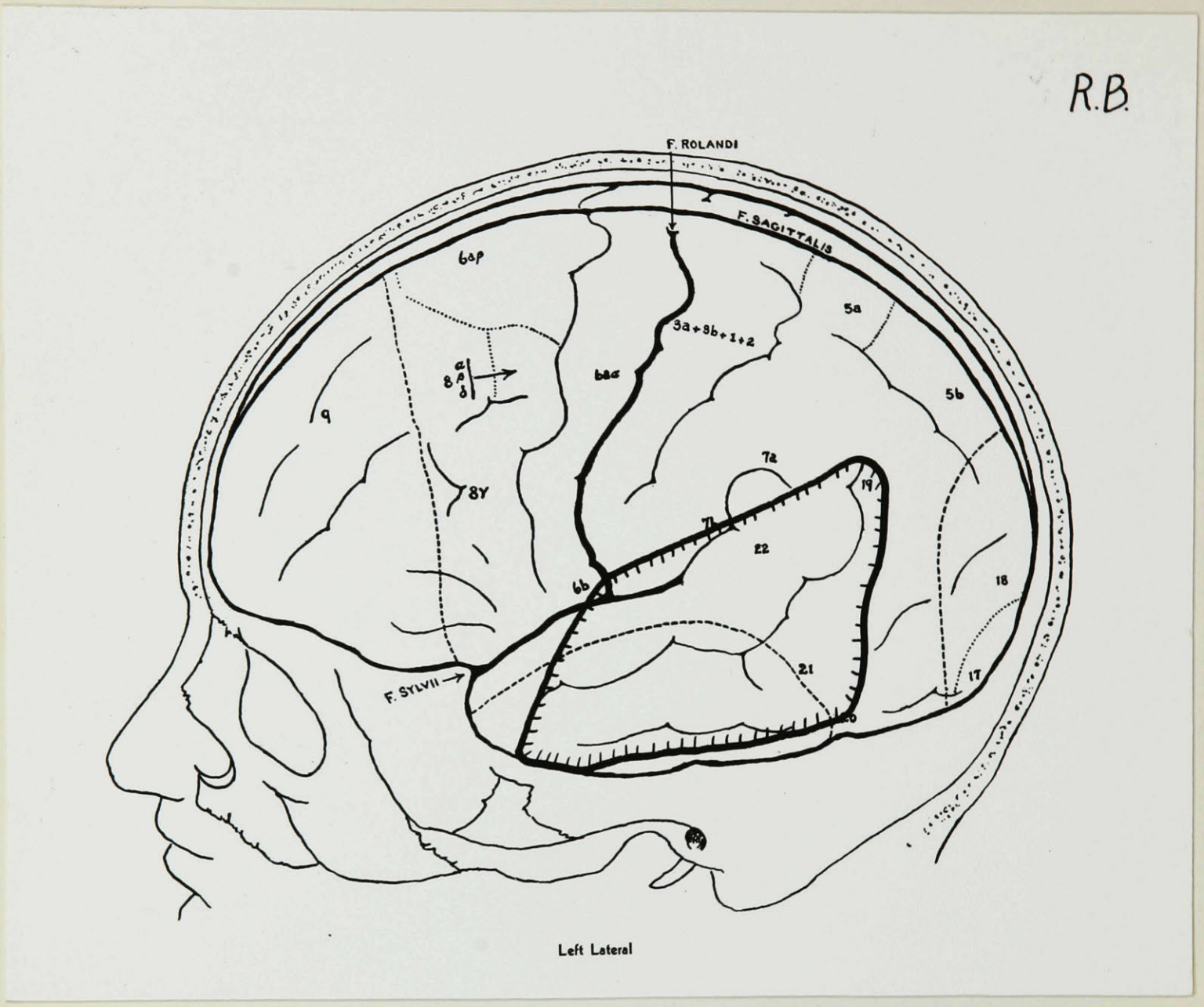

Fig. 9. Case R.B. Excision of a left temporal lobe in a patient who was aphasic following the initial injury.

Case R.B., male, aged 26, was admitted to the Montreal Neurological Institute on August 6th, 1936 because of epileptic seizures of increasing number and severity of six years' duration. At the age of 11 he had had a head injury and been unconscious for two weeks. Following this there had been some transient difficulty in spelling, in reading certain words, and it had been necessary for him to attend special classes. In 1931 he had been examined by Dr. Spiller who had been unable to detect any distinct word deafness, word blindness or motor aphasia. However, he did have a definite stutter or hesitancy in speech. He was right-handed, above average intelligence, and had been a feature writer for newspapers. Pneumoencephalogram demonstrated a marked atrophy of the left temporal lobe, especially in its anterior portion.

On August 12th, 1936, Dr. Penfield carried out a left osteoplastic craniotomy under local anaesthesia, and removed a cerebral cicatrix involving the major part of the temporal lobe.

Following the operation he was able to express himself well. The selection of words and the pronunciation was good, although there was considerable hesitancy between words and short phrases, as if he was searching for the proper word. This was a little more marked than it had been before

operation. He talked much better when alone, than when he was surprised or ill at ease.

Preoperativelythe visual fields had been normal. Postoperatively he showed a right inferior homonymous quadrantic anopsia. On the sixth postoperative day Dr. C.K. Russel was unable to demonstrate any agnosia, apraxia or aphasia.

This case has previously been reported by Dr. I. Wechsler (1937) as one of complete removal of the left temporal lobe without causing aphasia.

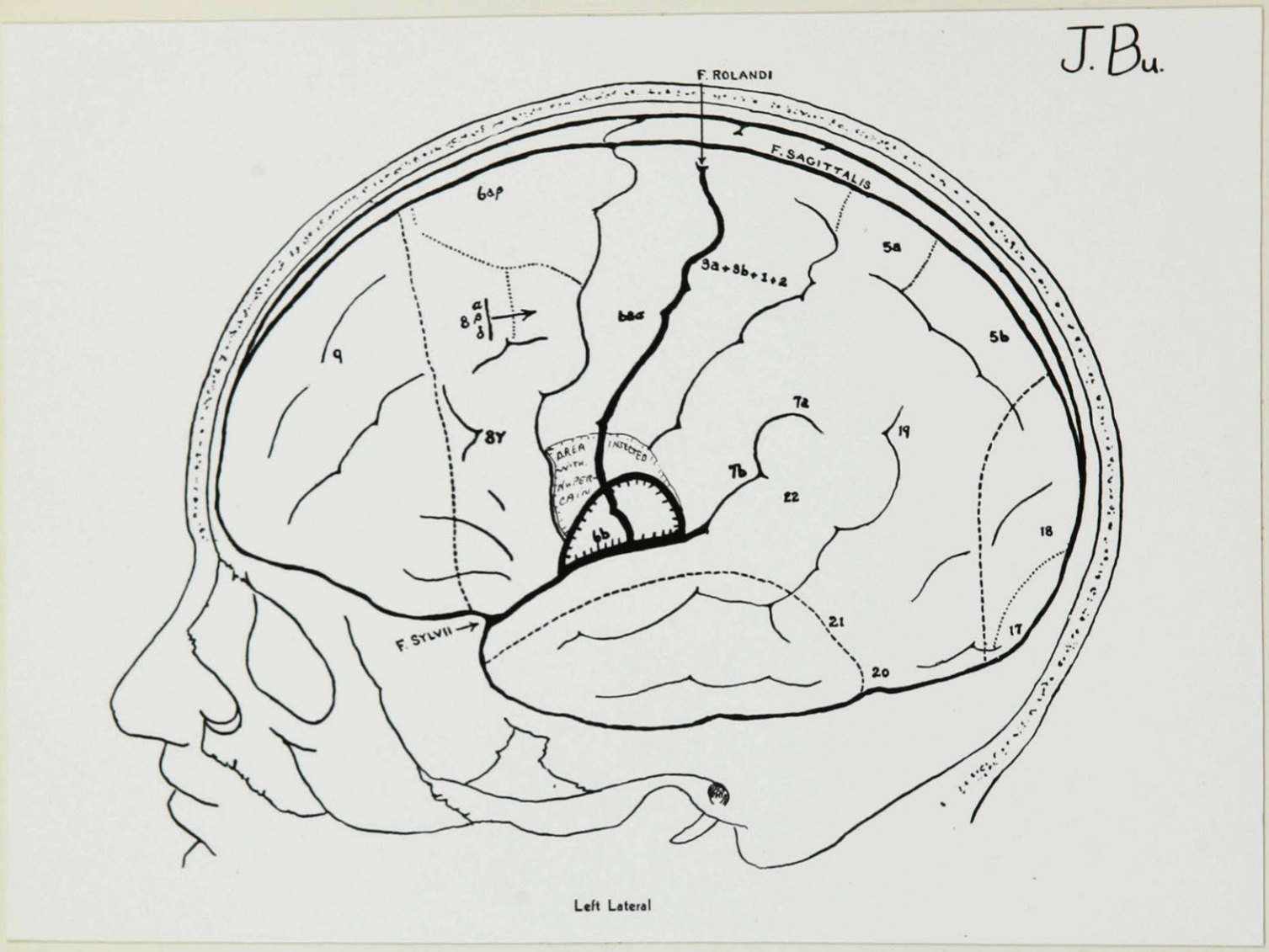

Fig. 10. Case J.Bu. Excision of an area in a right-handed patient who was aphasic following his initial injury.

Case J. Bu., male, aged 50, was admitted to the Montreal Neurological Institute on January 27th, 1944, because of ten seizures which had followed a head injury three years before. Following the injury he had been unable to talk, and on the third day he developed a hemiparesis which cleared in a month and a half. He was right-handed and his speech was considered normal on admission.

On February 9th, 1944, a left osteoplastic craniotomy was carried out by Dr. Penfield under local anaesthesia. A cerebral cicatrix was found at the lower end of the pre and postcentral gyrus which was excised.

In the postoperative period he had some dysarthria, but there was no evidence of aphasia at any time.

Case H.C., male, aged 45, was admitted to the Montreal Neurological Institute on July 25th, 1939 for investigation of epileptic attacks of six years' duration. He gave a history of having had encephalitis twelve years prior to admission. As a result of previous investigation a neoplasm in the left temporal region was suspected. Attacks observed during hydration consisted of objects coming nearer, unlocalized movements of the extremities with aphasia, cyanosis, holding of breath and salivation. The patient would return to normal about one minute after the attack was over. Between attacks there was no evidence of aphasia . He was left-handed.

On July 27th, 1939 a left subtemporal myoplastic craniotomy with exploration was carried out by Dr. Penfield under local anaesthesia. A definite patch of adhesions at the posterior end of the fissure of Sylvius was found. Exploration was carried around underneath the temporal lobe and anterior to it without finding any abnormality. The fissure of Sylvius was then opened by careful dissection. The pial covering was left quite carefully on the island of Reil and also upon the gyri above the fissure. The first temporal gyrus was injured to some extent only at the depth of the fissure during this dissection. An exploratory biopsy needle was passed in two directions to the temporal lobe

and also one exploratory puncture was made upward and forward into the frontal lobe. A small amount of indurated tissue was removed from the surface of the island of Reil, certainly not more than 2 mm. in diameter. (Pathology later reported this to be normal brain tissue).

It was observed during the latter part of the procedure that the patient seemed to become hard of hearing, and the anaesthetist had difficulty in making him understand. At the end of the operation he was able to count to 20. A little later, when told he would have avertin by rectum, he objected as he felt he "might be needed" and so he was willing not to go to sleep. Although there was difficulty at this time in making him understand the spoken word or making him hear, one could not be sure of which, he was still able to understand well.

In spite of the fact that no tumor was found it was felt that there was a tumor deep to the island of Reil. On the first postoperative day he was unable to understand anything that was said to him, however loudly, but he could read sentences written on paper quite clearly and understand them. On the second postoperative day his speech was very confused, he had difficulty understanding what he read, but was able to carry out orders conveyed to him by pantomime.

On July 31st, four days postoperatively, Dr. Penfield made the following note: "His failure to understand developed

on the operating table. There could have been no oedema at the time. There was no difficulty pronouncing words and no tendency to jargon, once or twice he asked an important question, for example, "how long will I be here?" I replied, "three weeks". He repeated the words, "three weeks", and nodded his head and smiled. His words this day, at times, were well formed. He stopped often, repeated frequently, but the word was used properly and pronounced accurately. A typical sentence was," you must do something for me here" pointing to his head, "in order to make it possible --- to make it possible -- to make it poss-- for me -- to talk -- because --- because --- I can't go back --- this way - people --- will look at me --- and it will be --- bad --- be very bad." He became clearer and understood more as the conversation went on. There seemed to be a complete block when the conversation started, both for talking himself and for understanding. He was able to carry out calculations presented to him on paper and correct errors of his own or other people."

He understood the written word, partly understood the spoken word, and he still kept saying the correct word, and at times, showed some jargon. As indicated, his aphasia seemed to vary.

On August 1st he said himself, "I can hear everything but I cannot understand". "I hear everything but I cannot

make it out". He used many words incorrectly, and would not repeat words correctly, frequently getting the first letter correct and then ending in "iss" or "ass" or "es". When reading aloud, he would frequently substitute, "I", for "your" or "me", for"you", but on re-reading he would correct this. Towards evening he would fatigue and the mistakes would increase.

On the seventh postoperative day he had improved a great deal and mistakes were few.

On the thirty-fourth postoperative day a note was made to the effect that he seemed to have more difficulty understanding visitor's speech, more so than those who visited him frequently. He also had difficulty in understanding the simile and the metaphor - more complicated forms of speech. There still was some auditory agnosia, although other forms of aphasia had passed. Comment was made on the attention factor, which seemed to play a part in his agnosia. Things referring to himself he understood perfectly, but he did not listen to discussion not referring to himself and it was on these matters alone that he required repetition and evidenced difficulty in comprehension. At the time of discharge, September 15th, he said he had difficulty in understanding only those persons whose remarks he felt were not worth listening to. He would look

interested, and at the same time think of other things of a more personal interest to him, then he would realize he had not understood and would have to ask them to repeat. Follow-up six years later revealed his speech and comprehension to be normal.

Summary - A 45 year old, left-handed male, who because of seizures had the left temporal lobe explored. A definite patch of adhesions were found at the posterior end of the fissure of Sylvius. The Sylvian fissure was separated and the island of Reil examined, in an effort to find a suspected deep-seated tumor. No abnormalities were found. During this procedure some damage was done to the first temporal gyrus. Immediately following this he developed an auditory agnosia. A few days later he showed very marked jargon aphasia which cleared rapidly. The auditory agnosia persisted after the jargon aphasia had cleared. During this he understood familiar voices better than strange ones, and simple English better than the more complicated forms. He could understand things he felt were important or concerned himself, whereas things that were not important he would not understand. This he attributed to an attention factor.

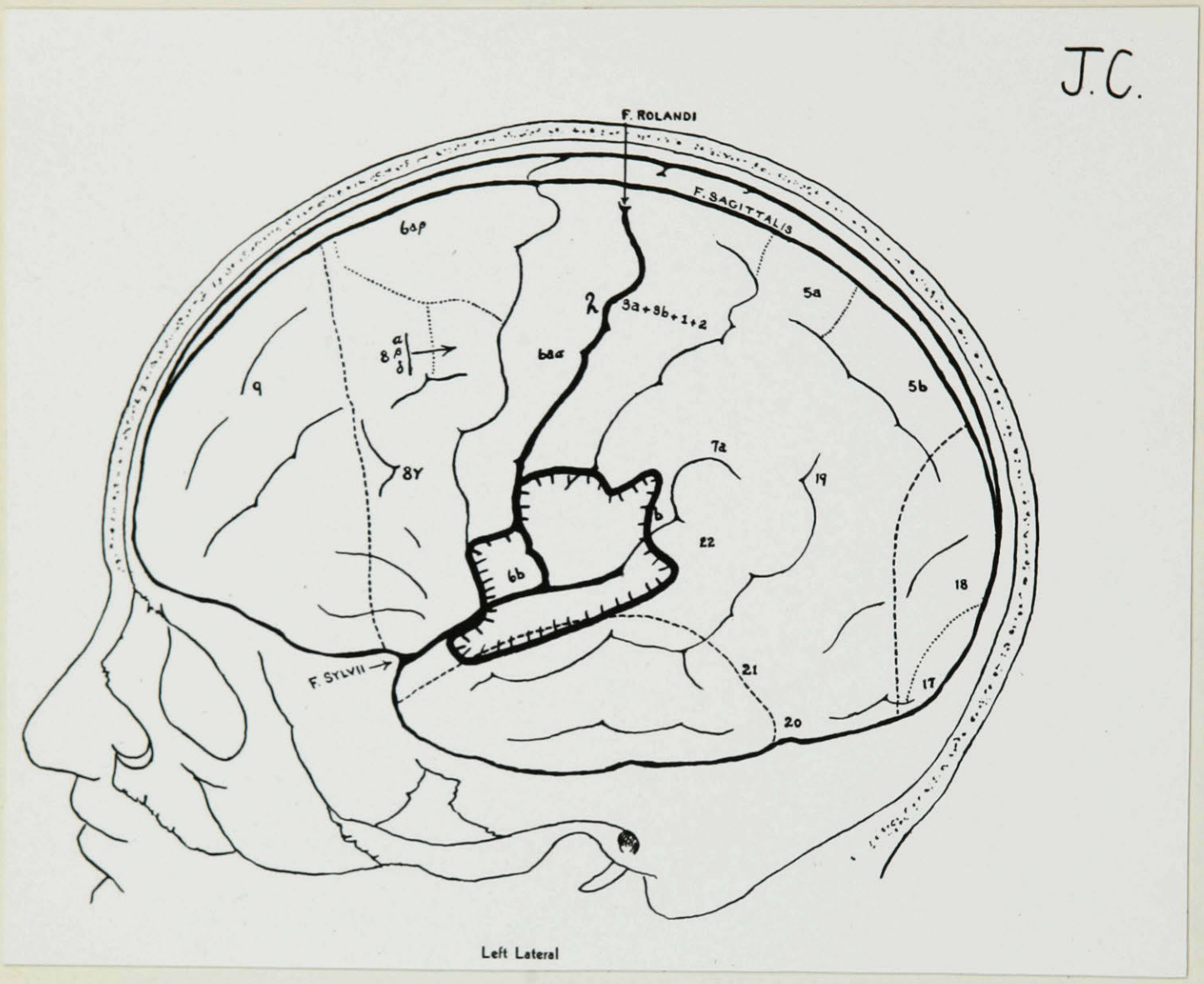

Fig. 11. Case J.C. Excision of an area in a right-handed patient who developed a transient secondary postoperative aphasia.

Case J.C., male, aged 23 was admitted to the Montreal Neurological Institute on August 11th, 1942 because of epileptic seizures of six months' duration. While on active military duty in Iceland, 1940, he had been struck on the left side of the head and was unconscious for about thirty minutes. He had been able to return to work the following day. In 1941, while in England, he was struck again on the left temporal region during an air raid. In neither case was there any history of aphasia or speech difficulty.

He was right-handed and apart from a palpable depression in the left parieto-temporal region of the skull, the general physical and neurological examinations were normal.

On September 9th, 1945 under local anaesthesia, Dr. Penfield performed a left osteoplastic craniotomy and removed a meningo-cerebral cicatrix. The area damaged was placed exactly upon the fissure of Sylvius including the first temporal convolution, the lower end of the postcentral and a little of the precentral gyri. The first temporal convolution was removed down as far as the bottom of the fissure of Sylvius and over a measured extent of 5 cm. exploring the island of Reil.

On the night of the operation he was able to move the right arm and leg as well as the left. He was "groggy" but named objects accurately, with the exception of a bed

pan, which he called a pencil on one occasion. On the first postoperative day he was drowsy and had difficulty talking. During the next twenty days he had a rapidly changing aphasia and weakness of the right hand. On the fifth postoperative day it was at a maximum when he was only able to say "I", "no" and "o.k.", but he understood what was said to him and made his wants known by gestures. On the fourteenth post-operative day he had improved greatly, but was still unable to name objects shown to him. On the twenty-first post-operative day the only speech difficulty was when reading aloud. This was done slowly but with understanding. Six weeks later speech was considered normal.

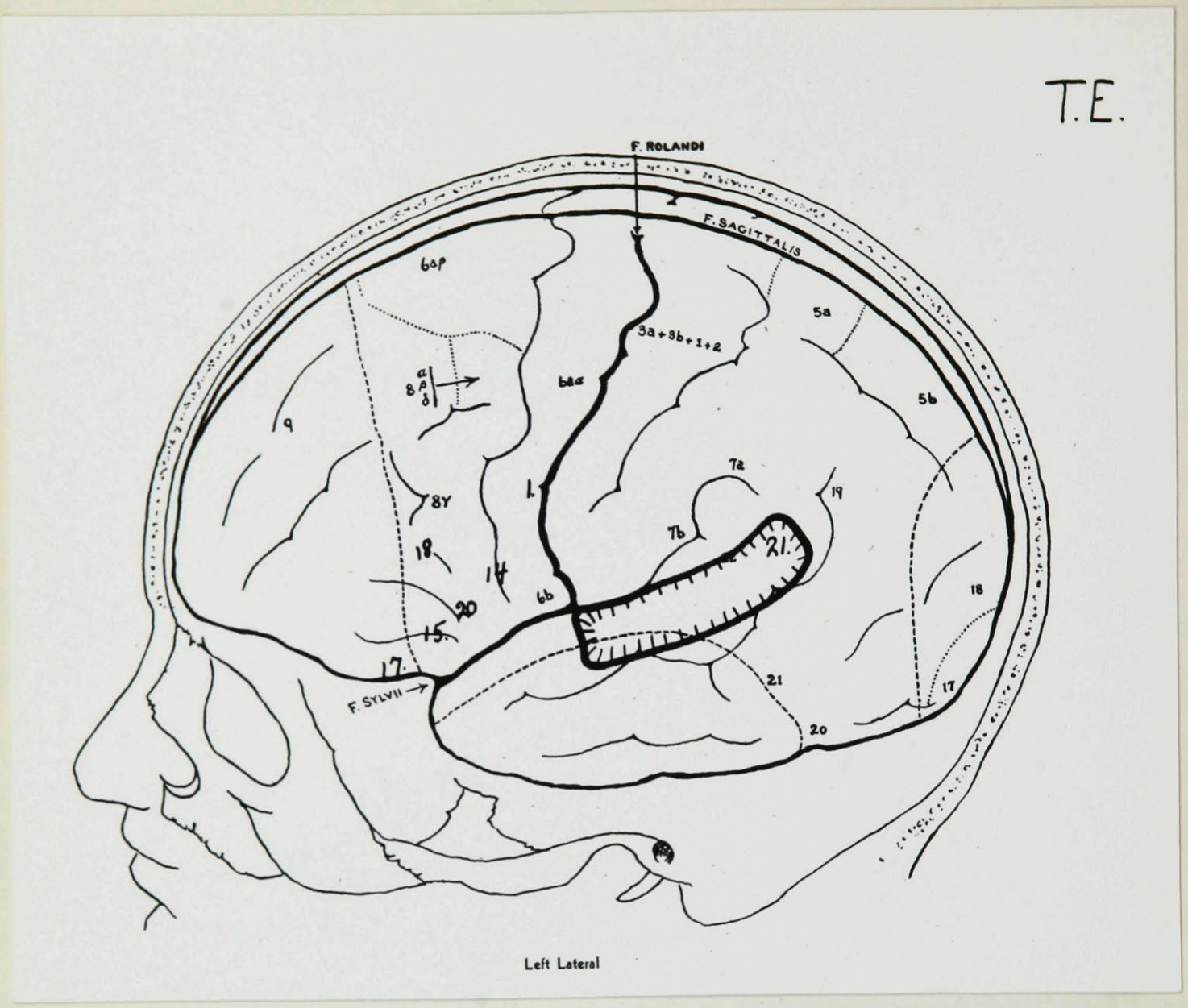

Fig. 12. Case T.E. Excision of an area in a right-handed patient, who was aphasic following the initial injury and again postoperatively.

Case T.E., male, aged 20, was admitted to the Montreal Neurological Institute because of unusual epileptic seizures of three years' duration. At the age of five he had had a severe illness, the exact nature of which was not known. He was said to have vomited, developed a severe headache, and two or three days later became aphasic. The record did not say for how long. However, at the time of admission his speech was normal. He was right-handed. The electroencephalogram pointed to a lesion in the left temporal region.

On April 22nd, 1944, Dr. Penfield performed a left osteoplastic craniotomy and removal of cerebral cicatrix under local anaesthesia. Filamentous adhesions were found between the dura and the arachnoid in the temporal region extending up just above the fissure of Sylvius. The posterior part of the first temporal convolution was narrow and covered a cyst. The cyst extended downward along the fissure of Sylvius, being a measured depth of 3 cm. and measuring about 5 cm. from the floor back. It seemed most likely that it was the remnant of an old intracerebral haemorrhage. The first temporal gyrus was removed as far forward as the lower end of the postcentral gyrus and backward to include the angular gyrus. There was no injury to the surface of the upper bank of the fissure of Sylvius.

Electrical stimulation of the cortex during the operation produced some interesting effects. Vocalization was produced at point 1. Counting was arrested on two occasions at point 15 and he could notthink why points 17, 18 and 20 failed to arrest speech. At point 14 he was interrupted and he said it was due to the fact that he could not close his mouth. Stimulation of point 21 brought on different replies, including "It was as if a sound was going in both ears. It was like a quiet buzzing".

On the night of the operation he responded well verbally. On the fourth postoperative day he had a little difficulty in finding words, and on the sixth and seventh day he was "slightly aphasic". At the time of discharge, the tenth postoperative day, his speech was considered normal.

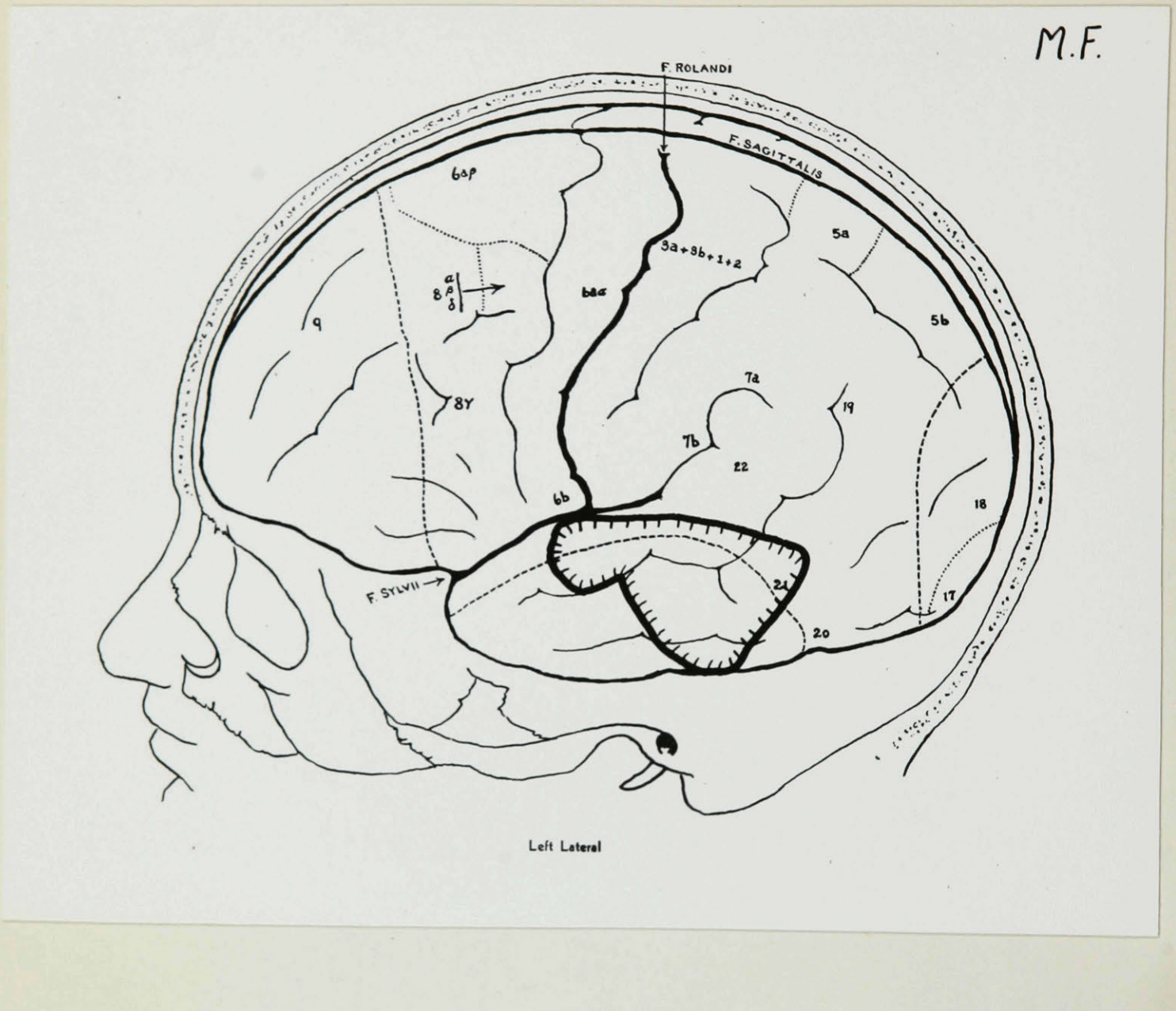

Fig. 13. Case M.F. Excision of an area in a right-handed patient who developed a transient, secondary postoperative aphasia.

Case M.F., male, aged 32, was admitted to the Montreal Neurological Institute on April 8th, 1939 because of epileptic seizures. Two years previously he had sustained a left parietal and basal skull fracture during an automobile accident. Unconsciousness, followed by irrationality, lasted for sixteen days. He recovered and was well until nine months later when he had his first seizure. He was right-handed and his speech was normal. Pneumoencephalogram suggested some diffuse cerebral atrophy, particularly in the left cerebral hemisphere, the maximal loss being visible in the region of the left temporal lobe. The electroencephalogram suggested an epileptogenic focus in the same region.

On April 29th, 1939 a left osteoplastic craniotomy was carried out by Dr. Penfield under local anaesthesia. Just below the fissure of Sylvius, in the posterior part of the temporal lobe, dense adhesions were found and the brain was converted into a yellowish gelatinoid scar. This included the first and second part of the third convolution. The scar was excised and at no time during, or after, the procedure was there any evidence of aphasia. He named readily and accurately a series of objects and showed no mispronunciation or hesitancy in talking.

Sixteen hours after the operation he began to lose his speech and soon was able to say only a few words. He had a

series of seizures and a right hemiparesis. On the ninth postoperative day he was still very slow to understand and limited in comprehension. The visual fields were normal. On the twenty-third day his speech was considered normal. He was examined a year later and there was no evidence of any aphasia or speech defect.

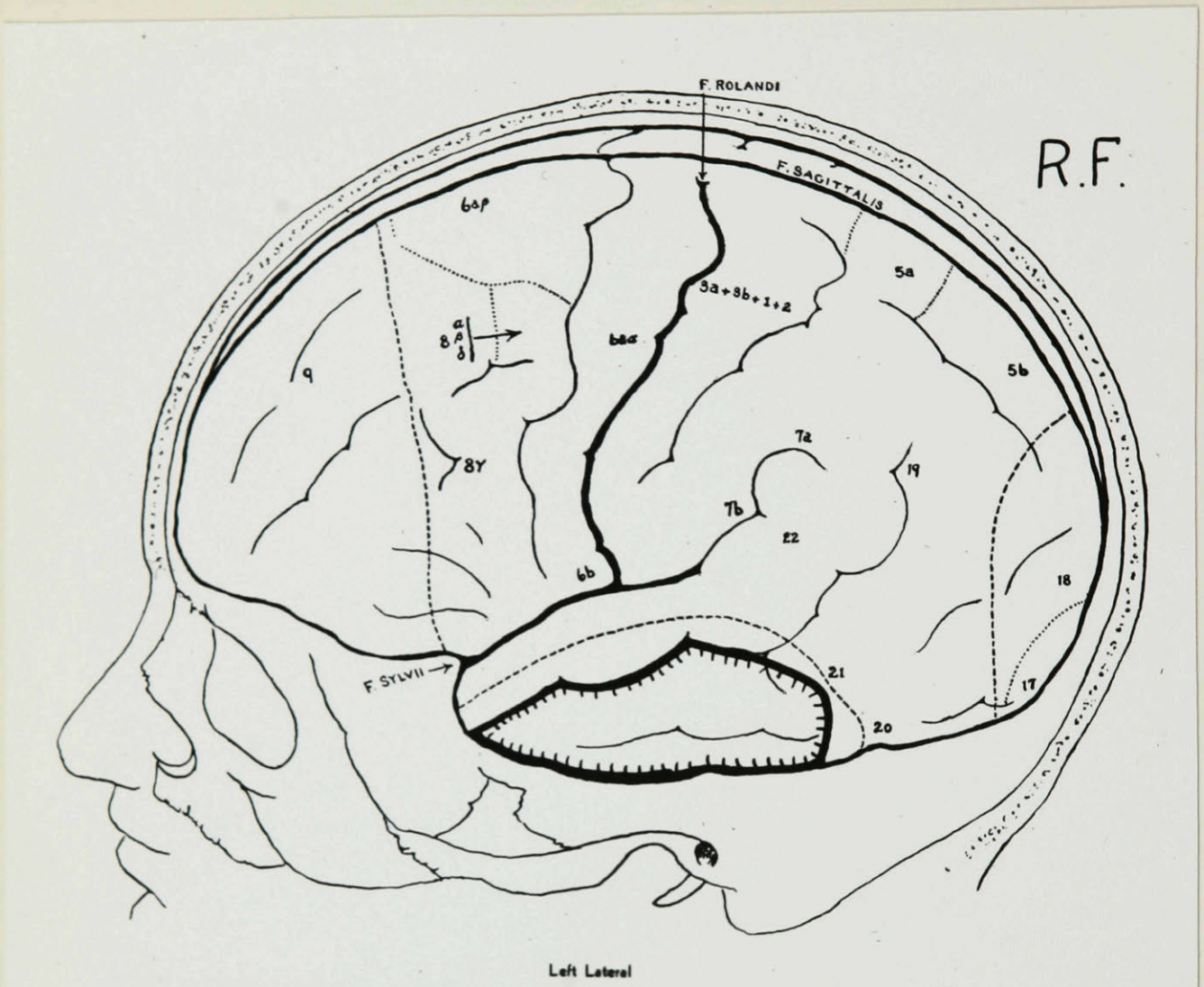

Fig. 14. Case R.F. Excision of an area in a right-handed patient who developed a transient postoperative aphasia.

Case R.F., male, aged 27 was admitted to the Montreal Neurological Institute on March 15th, 1939 because of epileptic seizures of three years' duration. There was a history of a head injury at the age of 12, of uncertain severity, otherwise there was nothing to suggest a cause. He stated that he used to be left-handed but changed to the right and was unable to do things that he formerly could with the left hand. His speech was considered normal.

On March 31st, 1939, a left osteoplastic craniotomy was carried out by Dr. Penfield under local anaesthesia. Adhesions between the arachnoid and the dura were cut. The second and third temporal convolutions were removed as far forward as the tip of the temporal lobe and down to the lateral wall of the inferior horn. The first temporal convolution was not touched. During the procedure phonation occurred when point "H" was stimulated. The phonation continued for two or three seconds after the stimulation was withdrawn.

During the latter part of the operation a general anaesthetic was given (Avertin and ether). The following day he had not completely regained consciousness, remaining drowsy and restless. The wound was re-opened because of suspected hemorrhage but nothing was found. The next day his responses to painful stimuli were those of a conscious person, but he did not speak. On the third postoperative

day he was very much alarmed, crying and almost completely aphasic. All he could say was "Oh my God" and "Oh my head". On the tenth postoperative day he still did not use any words. He was able to hold a pencil in his hand and make a series of "Js". He was able to show his teeth on command but when asked to take the examiner's hand, he still showed his teeth. On the twenty-third day he showed a marked aphasic jargon and from that time on his speech slowly improved. As his speech improved there was a good deal of substitution. On the fifty-third day speech was considered normal but reading was stiff and difficult.

C.G.

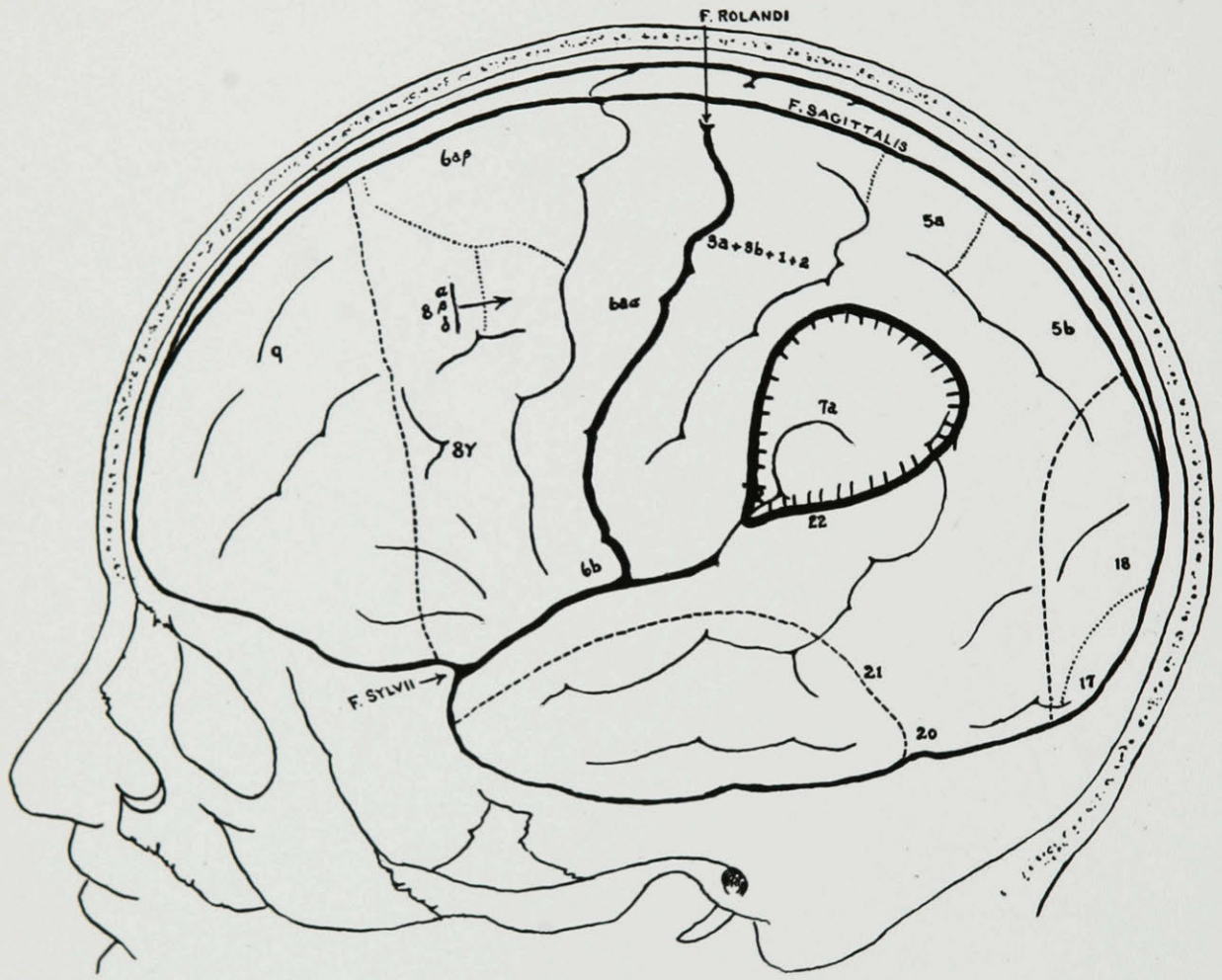

Fig. 15. Case C.G. Excision of an area in a patient who had been aphasic following the initial injury, with incomplete recovery. No change postoperatively.

Case C.G., male, aged 38, right-handed, was admitted to the Montreal Neurological Institute on August 18th, 1939 because of epileptic seizures which followed a gun-shot wound of the head fifteen years before. He had been operated on at the time and the bullet was said to have been removed in several pieces. Following this there was a long period of amnesia and inability to talk. In 1932 a second craniotomy was done through a small opening. How much the brain was disturbed is not known, however, he again became completely aphasic. His recovery was slow. He first began to use words incorrectly, however he could read, and indicate what he wanted by pointing to papers on which words were written. If he became excited the defect would become more marked. He was able to do simple calculations in his own mind but would be unable to give the right answer. He would be aware of his mistakes. He would understand what was said to him, and could read the newspaper. He had difficulty in writing, comparable to that of speaking and at first could not even write his own name. He stated that he had had to relearn everything, including the alphabet. He found this easier than learning addition over again.

At the time of examination he could read the newspaper but had difficulty in writing and, as a result, would write very little. Spontaneous speech and the reception and

understanding of the spoken word were very good. There was some difficulty with more complicated tests, but simple tests were carried out correctly. He made some mistakes with more difficult words when reading, but was able to correct them. He made many mistakes with letters and many mistakes were made when writing. He copied well. Calculation was poor. The most serious defects were those of writing, calculation and naming letters. One gathered that he had more difficulty with letters than with words. He also had a marked memory defect for recent events and was retarded. Dr. Penfield was of the opinion that he had a memory failure without any connected aphasia.

On August 22nd, 1935, under local anaesthesia, Dr. Penfield performed a left parietal osteoplastic craniotomy, and removed the cerebral cicatrix down to the ventricle.

There was no change in the patient's speech defect, following the operation, that could be detected. Nine months later his speech and general alertness had improved. This, it was felt, could be related to a decrease in the number of seizures. He could write simple sentences, but made mistakes on the more difficult words. He made occasional mistakes when reading difficult words, such as "condolences" and "Vatican". He still had trouble picking out letters named to him, but he had no trouble naming letters that were pointed out to him. The chief difficulty was in the field of written language.

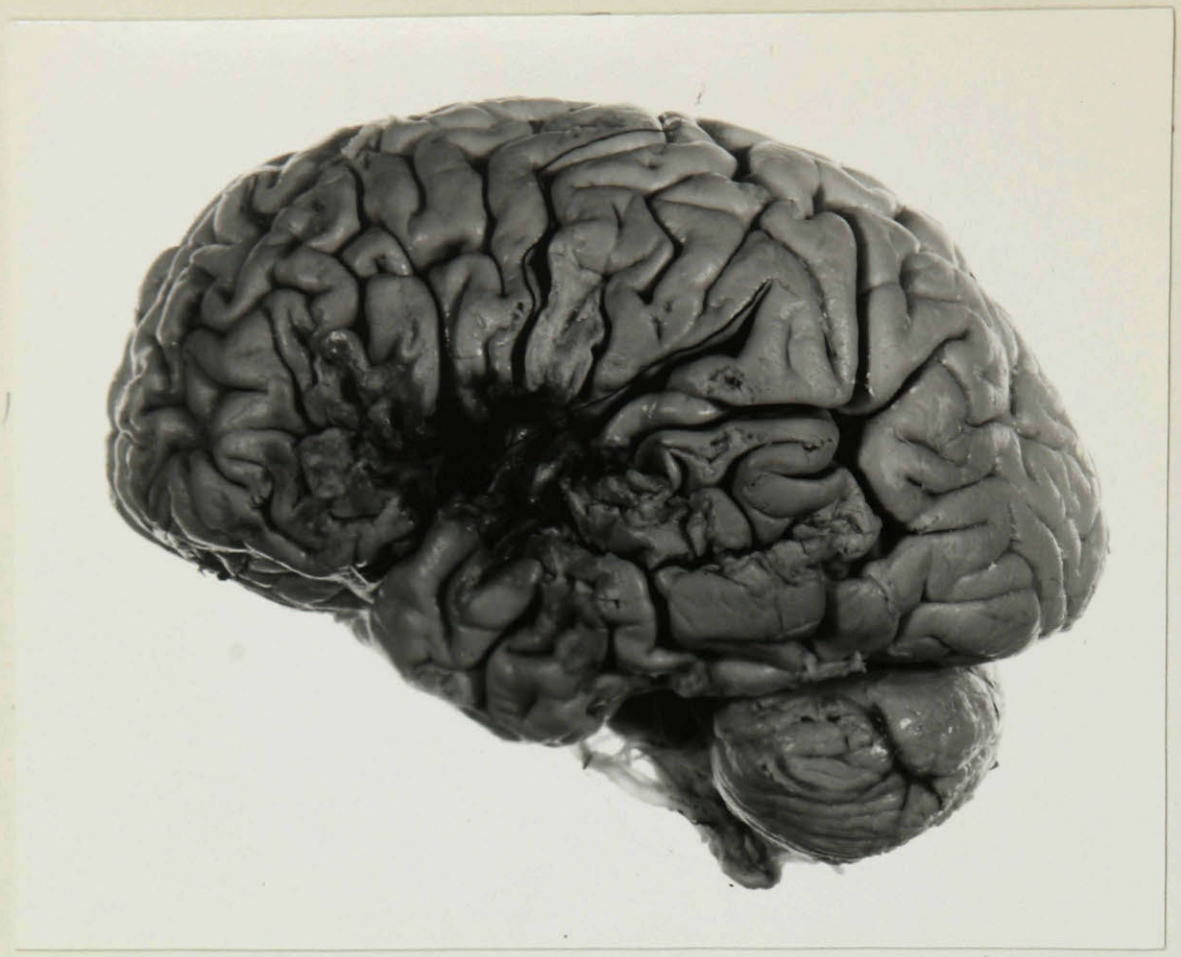

Fig. 16. Case D.H. Lateral view of left hemisphere showing excised and damaged area.

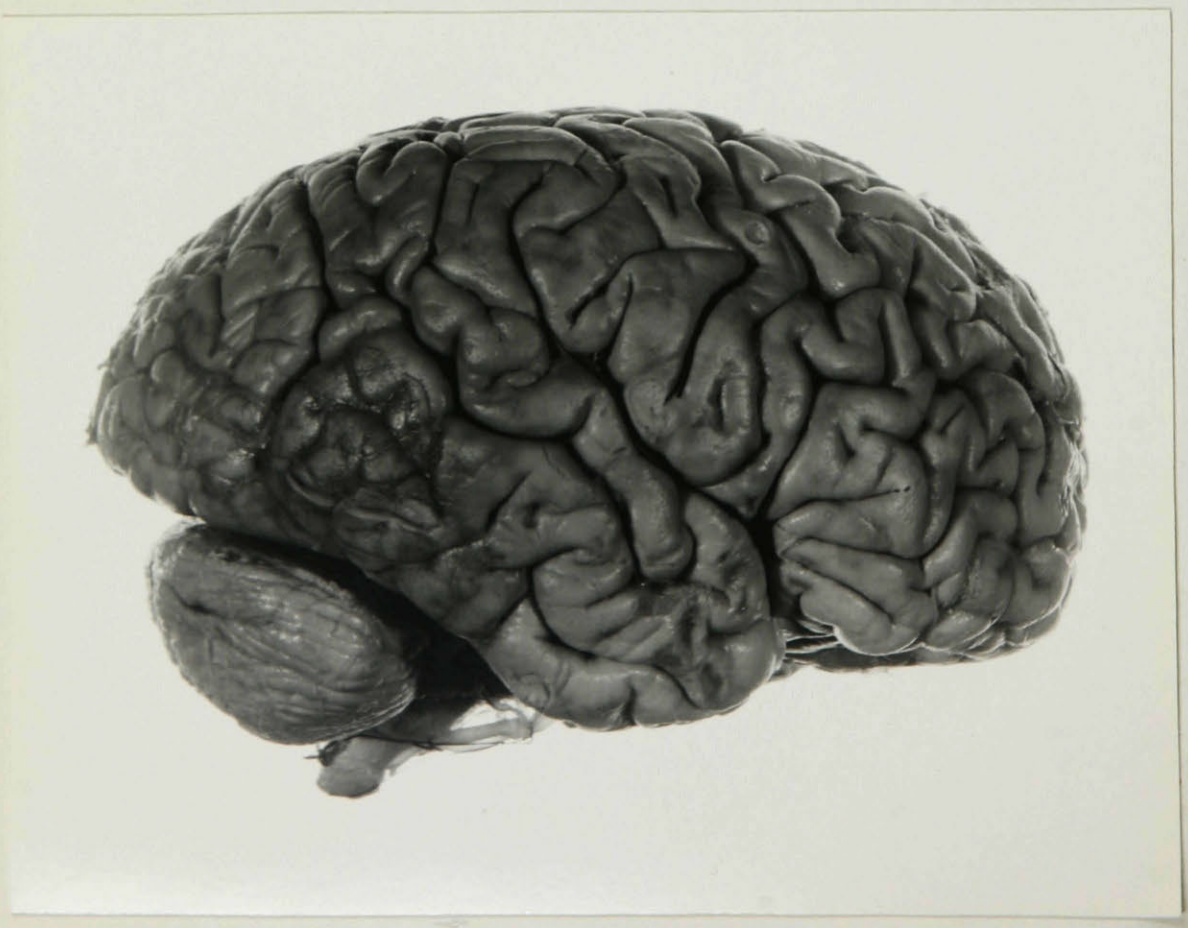

Fig. 17. Case D.H. Lateral view of right hemisphere for comparison.

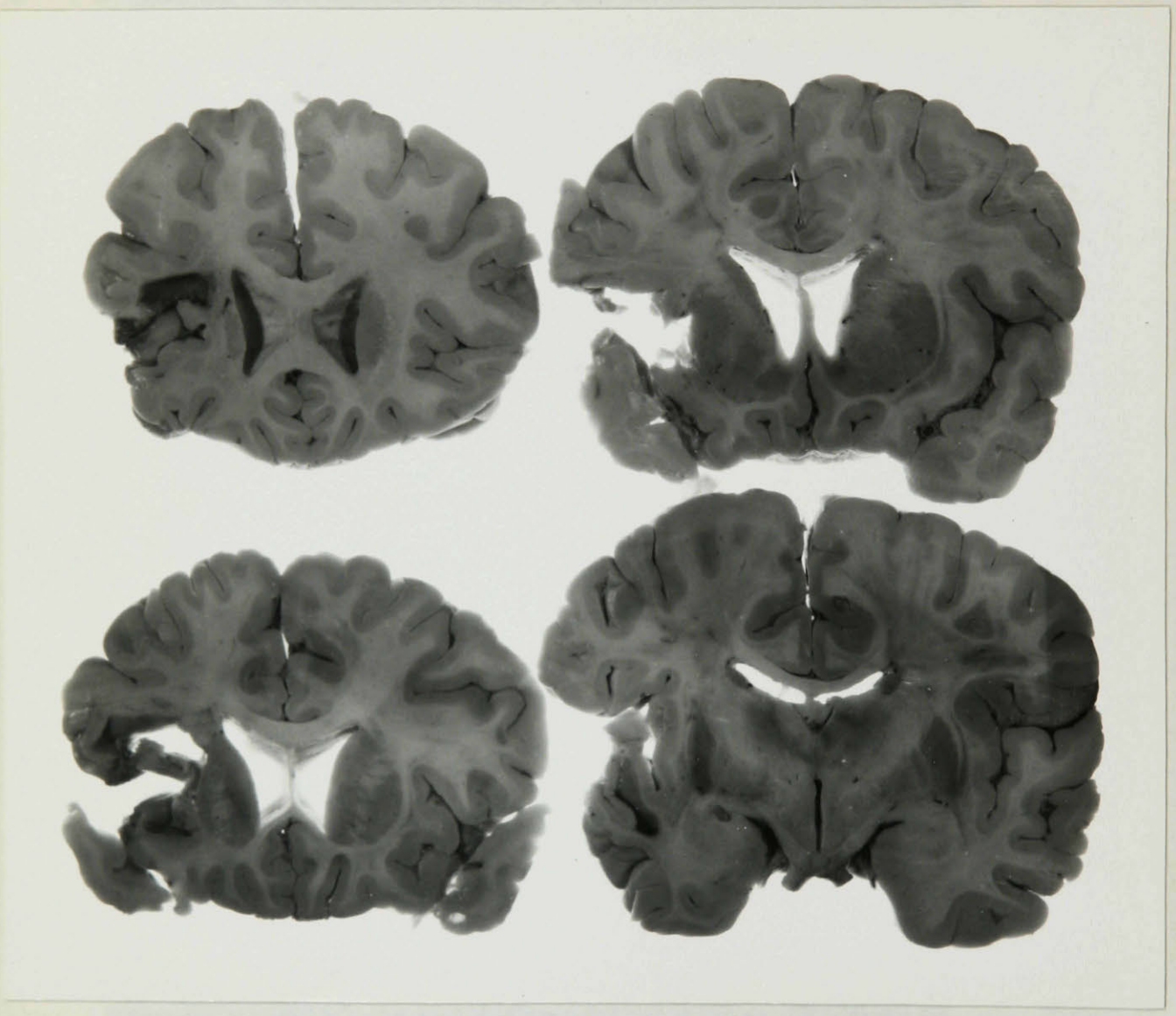

Fig. 18. Case D.H. Coronal sections of brain showing depth of excision.

Case D.H., aged 18, male, was admitted to the Royal Victoria Hospital on July 22nd, 1933 because of convulsive attacks since the age of three and a half years. These attacks were Jacksonian in type, involving the right arm and frequently were associated with an inability to speak without loss of consciousness. He was right-handed and without speech defect. X-rays demonstrated a calcified lesion in the lower precentral region.

On June 29th, 1933, under local anaesthesia, Dr. Penfield performed a left parietal osteoplastic craniotomy and removed the calcification and surrounding tumor. An incision was made parallel to the fissure of Sylvius just in front of the central fissure. The tumor was removed through this incision, partly by suction and partly with a spatula. Throughout the procedure the patient talked quite well and there was no evidence of aphasia. On the second postoperative day he was unable to speak clearly, could not construct sentences and explained what he meant by signs. His expressive aphasia increased until the eighth postoperative day when he was able to say a few words clearly. On the tenth postoperative day he was still improving. On the day of discharge, July 22nd, 1933, there was still a little residual speech defect. Two years later his speech, although slow, was considered normal.

The pathological diagnosis was astrocytoma with calcification.

On September 13th, 1942 he was readmitted because of recurrence of the seizures. His speech was considered normal.

On September 30th, 1942 a second craniotomy was performed. The lower end of the precentral gyrus was removed to an extent of 1.5 cm. Removal was carried out of what would be called Broca's area, down to the full depth of the fissure of Sylvius. The first temporal convolution was also removed in its anterior half as the tumor had invaded this area as well. He continued to talk throughout the procedure.

In this case there was no postoperative speech disturbance, in spite of the fact that the removal involved cortical tissue, whereas in the first operation, the cortex was incised only but not removed, the tissue removed being subcortical. Although the description of the postoperative speech defect was not good, one can say that it was an expressive type. The only explanation I can offer for the lack of speech defect, following the second operation, is that the right side had taken over the function of the left.

Autopsy, three years later, revealed an extensive destruction of the cortex in the vicinity of the posterior end of the third frontal convolution and the anterior end of the first temporal convolution. There was also extensive destruction of the subcortical tissue, including all of the external capsule. There was no recurrence of the tumor or had there been any speech disturbance (see Figs. 16, 17, 18).

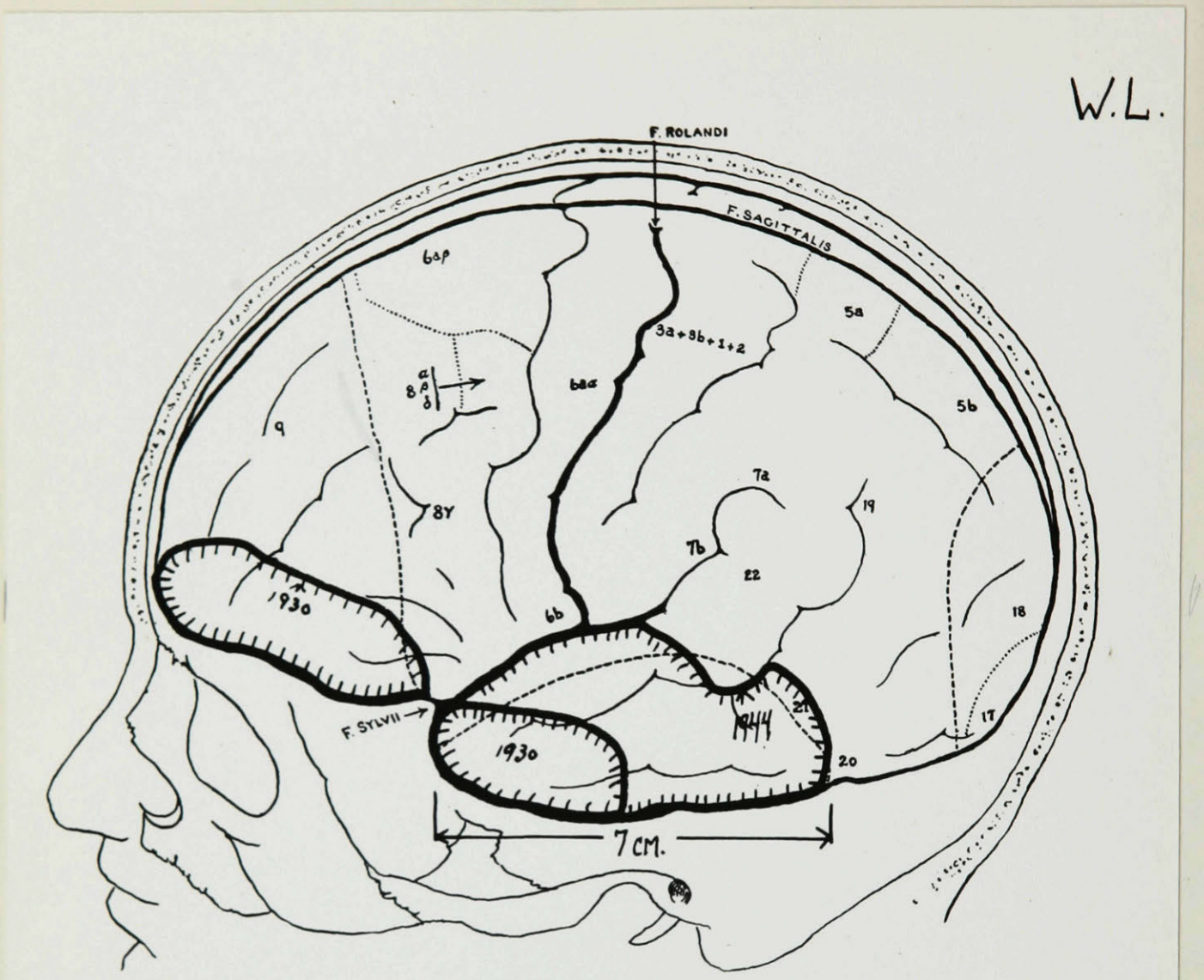

Fig. 19. Case W.L. Excision of an area in a right-handed patient who was aphasic at the time of initial injury.

Case W.L., male, aged 35 was admitted to the Royal Victoria Hospital on April 21st, 1930 because of epileptic seizures. In 1916, at the age of 20, he had been struck by a high explosive shell and had been unconscious for three weeks. Following this he stated he had some speech difficulty, the nature of which was not clear. He was right-handed and had some hesitation in his speech.

On May 7th, 1930, Dr. Penfield performed a left osteoplastic craniotomy under local anaesthesia. After cutting adhesions between the dura and brain, the under surfaces of the frontal and temporal lobes were found to be somewhat gelatinous, and the rest of the exposed brain did not appear quite normal. After stimulation the gelatinous-appearing brain was excised.

At no time following the operation was there any evidence of change in his speech, although in the postoperative period he had a transient hemiplegia which included the face. He also had a series of focal right-sided attacks involving chiefly the right hand. During one he was able to speak.

On April 11th, 1944, he was readmitted to the Montreal Neurological Institute because the seizures had recurred. He still used his right hand for writing, which was considered normal. He threw a ball with his left hand. His stream of talk was slow, deliberate but clear.

On April 21st, 1944, a second left osteoplastic craniotomy was performed by Dr. Penfield and further removal of the

temporal lobe was carried out to a point posterior to the motor area, plainly exposing the island of Reil. There was no interference above the fissure of Sylvius. The removal included all of what one would call the temporal lobe, but not the angular or supramarginal gyri (7 cm. from the tip of the temporal lobe posteriorly). He was able to talk throughout the whole procedure.

He was free of any speech disturbance in the postoperative period.

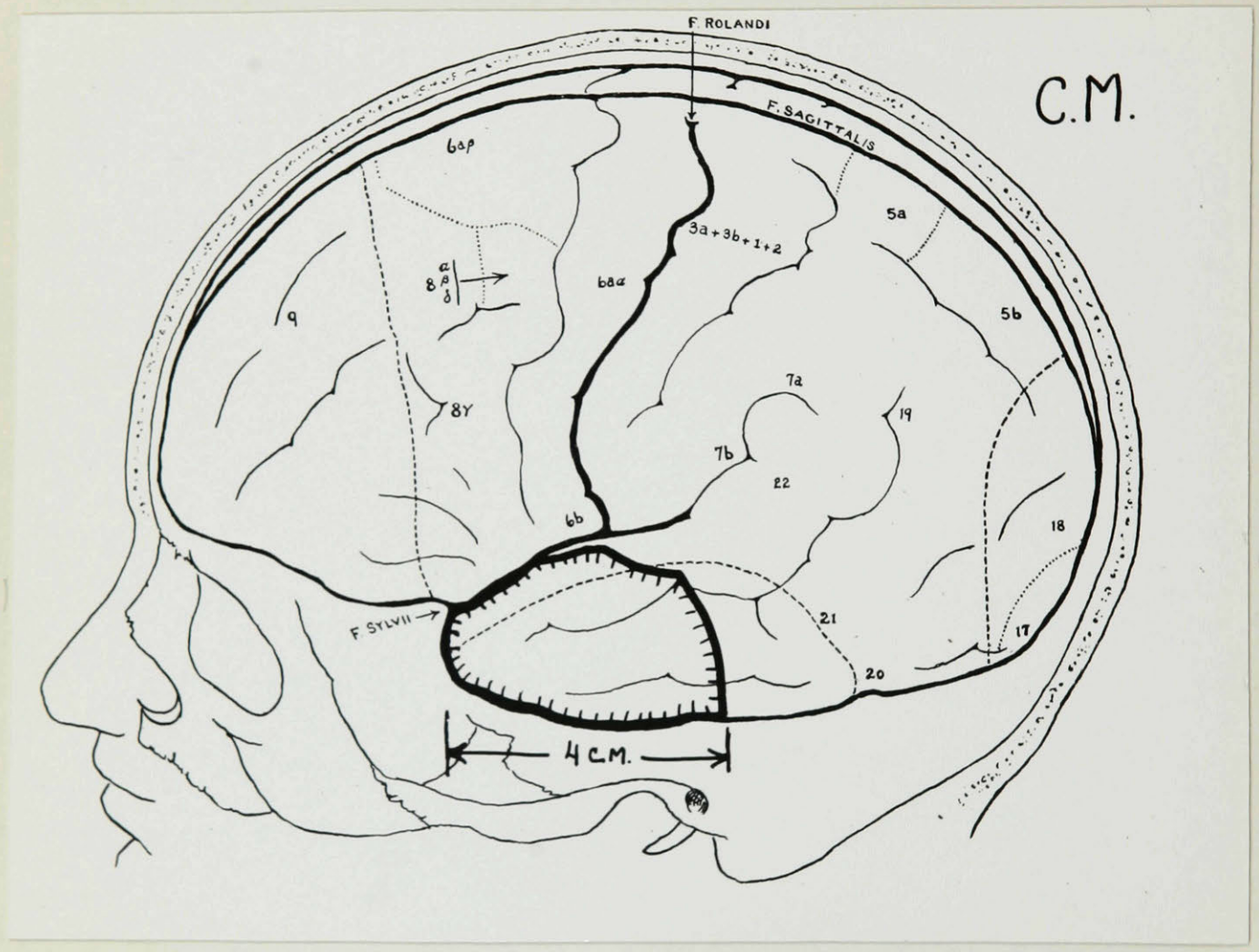

Fig. 20. Case C.M. Excision of 4 cm. from the anterior end of the major temporal lobe for decompression purposes.

Case C.M., male, aged 51, was admitted to the Montreal Neurological Institute on February 12th, 1946, because of headaches of unusual severity. He was right-handed.

An intracranial tumor was suspected and on February 15th, 1946 a left subtemporal decompression and exploration was carried out by Dr. Penfield, under local anaesthesia. No tumor was encountered, and a subtemporal decompression was left.

His speech was normal for 24 hours, then an aphasia slowly developed. On the fourth postoperative day he had a very marked aphasia. He was unable to name objects, recognize their name, or demonstrate their use, yet he was able to copy and read simple words. He was able to talk and make casual comments. He was unable to write spontaneously. As he had not improved on the sixth postoperative day, the craniotomy was reopened. The temporal lobe was under increased pressure, and this continued to be true even after a lumbar puncture was done. When the dura was opened further forward the whole temporal lobe bulged. The anterior end of the temporal lobe was then sucked out and some was removed for examination. The removal was carried from the tip, back 4 cm.

The patient remained aphasic for several days with a similar type of disability as described above. He then slowly recovered, the amnesic aphasia being most persistent. On March 19th, a month later, he was completely free of aphasia.

He understood what was said to him completely. He could name unusual sounds accurately. He could read well aloud with comprehension. His spontaneous writing was normal and he could name objects seen and felt without difficulty.

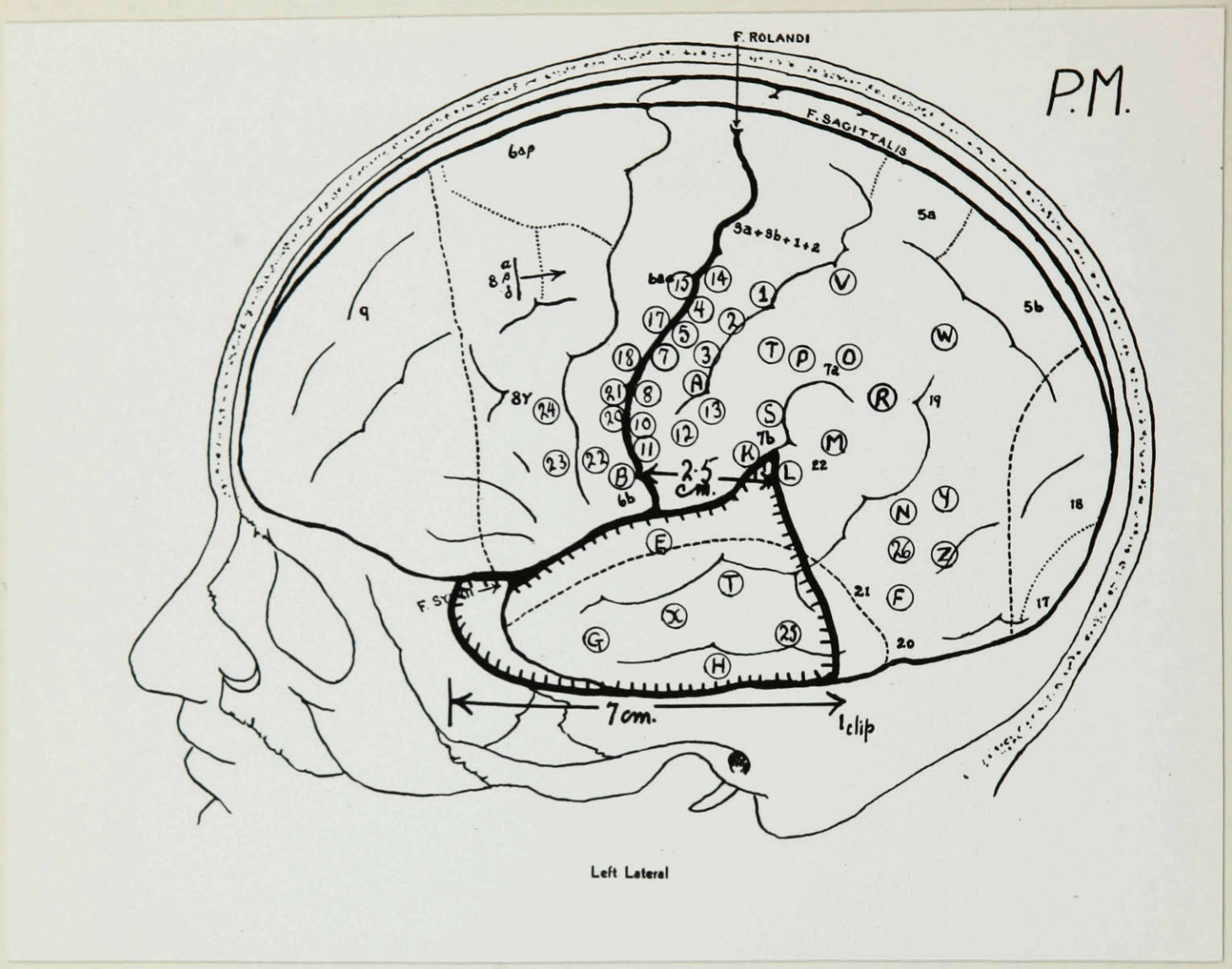

Fig. 21. Case P.M. Excision of the major temporal lobe followed by a postoperative aphasia.

Case P.M., male, aged 36 was admitted to the Montreal Neurological Institute on February 20th, 1945 because of seizures of five years' duration. In June of 1941 he had had a head injury, following which he was unconscious for fifteen minutes. He was admitted to this hospital at that time and diagnosis of traumatic subarachnoid hemorrhage was made from which he seemed to recover completely. At this time he gave a history of having had a severe illness about five years before, the nature of which was not clear but during which he was said to have been unconscious for two weeks.

In 1942 he was readmitted to the Institute for investigation of the seizures, and pneumoencephalography demonstrated a focal atrophy of the left temporal lobe. Surgical therapy was considered but it was felt he should have a longer trial of medical therapy.

He was readmitted on February 20th, 1946 because he had failed to respond to medication, and he seemed to be showing some mental deterioration. A pneumoencephalogram showed the focal atrophy of the left temporal lobe to have increased considerably.

On March 19th, 1946, Dr. Penfield performed a left osteoplastic craniotomy under local anaesthesia. The temporal lobe was found to be thinned out, yellow and tough. There were a number of small gyri and a good deal of tissue that could

have been giving rise to epileptic activity. The abnormality extended back to a point about 2 cm. behind the central fissure. The rest of the brain appeared normal. The temporal lobe was removed to a point exactly 7 cm. from the anterior end of the middle fossa. The line of removal was 2.5 cm. posterior to the central fissure. The removal was carried down to the ventricle. The anterior end of the temporal lobe was probably not functioning, but the first temporal convolution and the part that lay on the fissure of Sylvius was more or less normal in appearance. This was removed down to the island of Reil. Electrical stimulation of the cortex produced vocalization at point 17. Stimulation of 20 stopped counting. Stimulation at point 21 did not stop counting. Stimulation at 22 stopped counting twice and once it did not affect it. Stimulation of point 23 (Broca's area) caused counting to slow down and stop. Point 24 was the same. During stimulation of point 25 he was able to name objects correctly. During stimulation of point 26 he was unable to name objects correctly. He eventually called a key a "lock for key". When 26 was stimulated again he was shown a pen. He said, "That is a ...." then yawned. After withdrawal of the stimulation he said, "That is a ...." and made motions of writing with his hand. Two minutes later he was able to

name the pen correctly and the maker. He was unable to name objects when point "N" was stimulated and this persisted for one to two minutes after stimulation was withdrawn.

Stimulation of point "O" caused no interference with naming when the stimulator was at 2 volts. It was increased to 3 volts, and he could not name a pen nib. He called it part of a pen. At point "P" he called a key a fountain pen, then named a pencil correctly during stimulation. At point "R", after some deliberation, he named a match correctly during stimulation.

At point "S" he could not name objects during stimulation and all he could say was "ka - ka". At point "T" he was able to read aloud during stimulation. At point "S", after stimulation, he was able to continue for a few words, then said "ga, ga". Stimulation at "U" did not stop reading. Stimulation at "W" did not stop reading. Stimulation at "Y" did not stop reading. the first time, then, when repeated twice, he would say two words correctly and then the words would be inaccurately used and pronounced. Stimulation at "Z" did not stop reading. Stimulation at "M" interfered with reading. Stimulation at "K" interfered with reading numbers. He tried to read the numbers but got them wrong. Stimulation of point "J" prevented him from naming objects. Stimulation

of point "X" prevented him from naming objects correctly, for example, when presented with a pair of scissors he said, "That is a rink" ... "that is a pair....". Then he signified with his hands that he knew what it was. Stimulation at "R" caused him to slow down gradually and he said "kur - kur". Stimulation at "P" failed to stop counting. Stimulation at "L" caused him to slow down. When repeated, he continued counting without interruption. Stimulation at "T" failed to arrest counting. Stimulation at "J" stopped counting slowly. When repeated he was able to continue counting. Stimulation at "K" caused him to skip when counting.

In the postoperative period he developed an amnesic type of aphasia which increased to a complete aphasia, from which he was recovering ten days later, the time of writing.

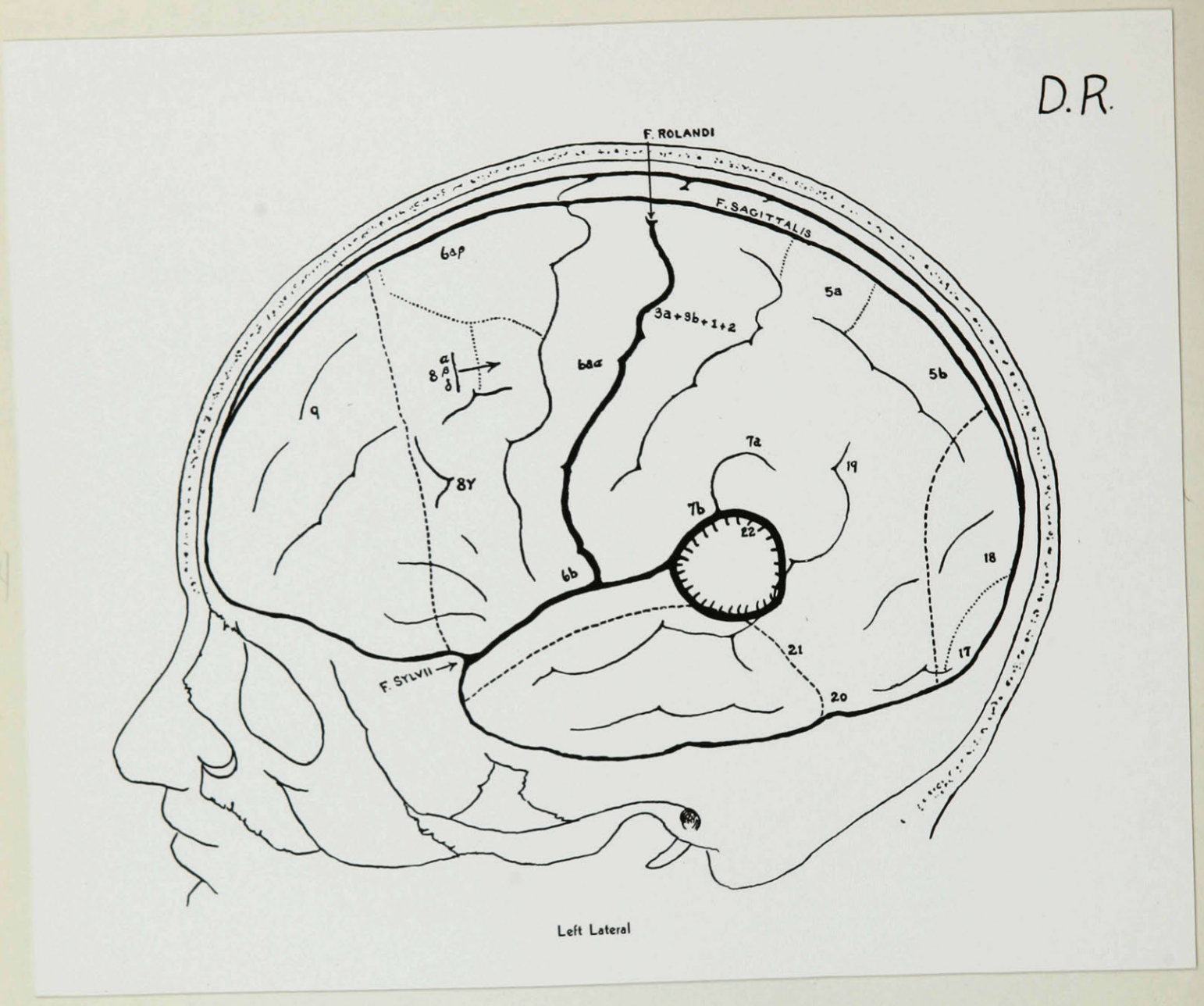

Fig. 22. Case D.R. Excision of an area in a right-handed patient who developed a transient secondary post-operative aphasia.

Case D.R., male, aged 18, was admitted to the Montreal Neurological Institute on May 2nd, 1935 because of epileptic seizures. He had had a head injury at the age of seven years at which time a bilateral subdural decompression was done in the Montreal General Hospital. A subdural haematoma was found on the right side, which was evacuated. The next day he developed right-sided seizures and a left subtemporal decompression revealed oedematous brain with areas of ecchymotic extravasation. No note was made of his speech at this time. At the time of admission he was right-handed, considered to be retarded mentally, but his speech was considered to be normal.

On May 14th, 1935 Dr. Penfield performed a left osteoplastic craniotomy under local anaesthesia. Adhesions between the brain and the dura were present at the site of the old decompression over the first temporal convolution. A hollow was found on the superior temporal lobe corresponding to the dilatation seen by encephalography in the inferior horn of the ventricle. An attempt was made to produce loss of function by placing 2 per cent novocain on the surface of the brain in the region of the hollow. This seemed to be without effect. The area was removed down to the ventricle. Following the incision he was able to talk, answered several questions, and said he would like

some vanilla ice-cream. He also gave his home address.

The night of the operation he spoke quite clearly, but was unable to name objects. The next day his speech was mostly jargon, and he did not understand when addressed. On the fourth postoperative day he understood the spoken word, but his speech was unintelligible. On the fourteenth day he was improving. He was discharged on the twenty-first day. The clinical examination did not demonstrate any abnormality, but the patient said at times he did not speak normally. When seen four months later in the out-patient department his speech was considered to be normal.

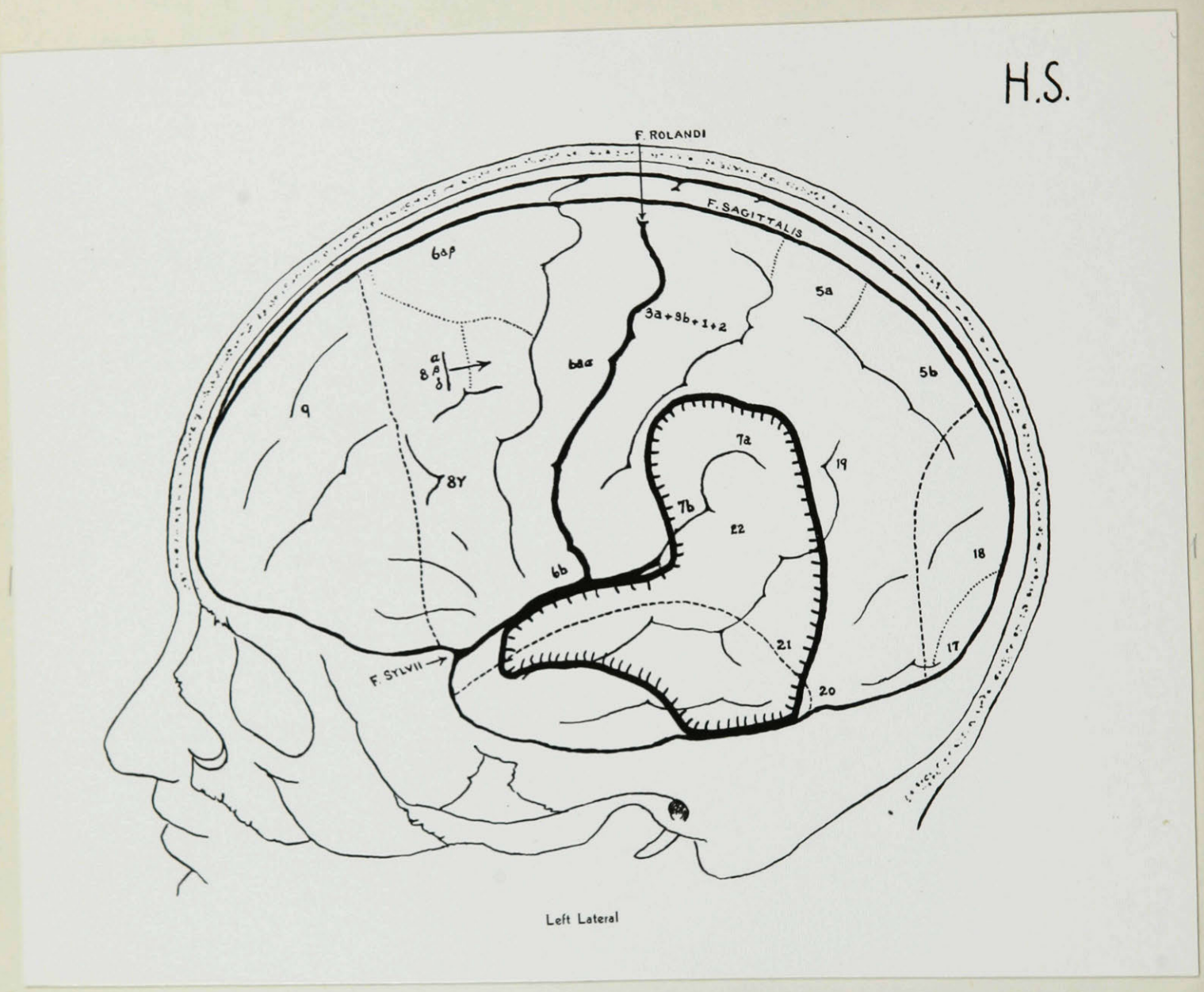

Fig. 23. Case H.S. Excision of an area in a right-handed patient who was aphasic following the initial injury.

Case H.S., male, aged 8, was admitted to the Montreal Neurological Institute on June 12th, 1941 because of epileptic seizures and weakness and ataxia of the right arm. At the age of four he had sustained a fracture of the skull with subdural and extradural haemorrhages. Operative decompression was done. He remained unconscious for five days and was unable to speak for three weeks. Prior to the accident he had had a good vocabulary. His recovery started with simple words and progressed rapidly until it had returned to its former state. He had been right-handed, but because of the weakness and ataxia he used his left for most things, including writing. He had a right homonymous hemianopsia.

On June 19th, 1941, Dr. Penfield performed a left parietal craniotomy and removed a large meningo-cerebral cicatrix. Following the operation he was free of any speech disturbance.

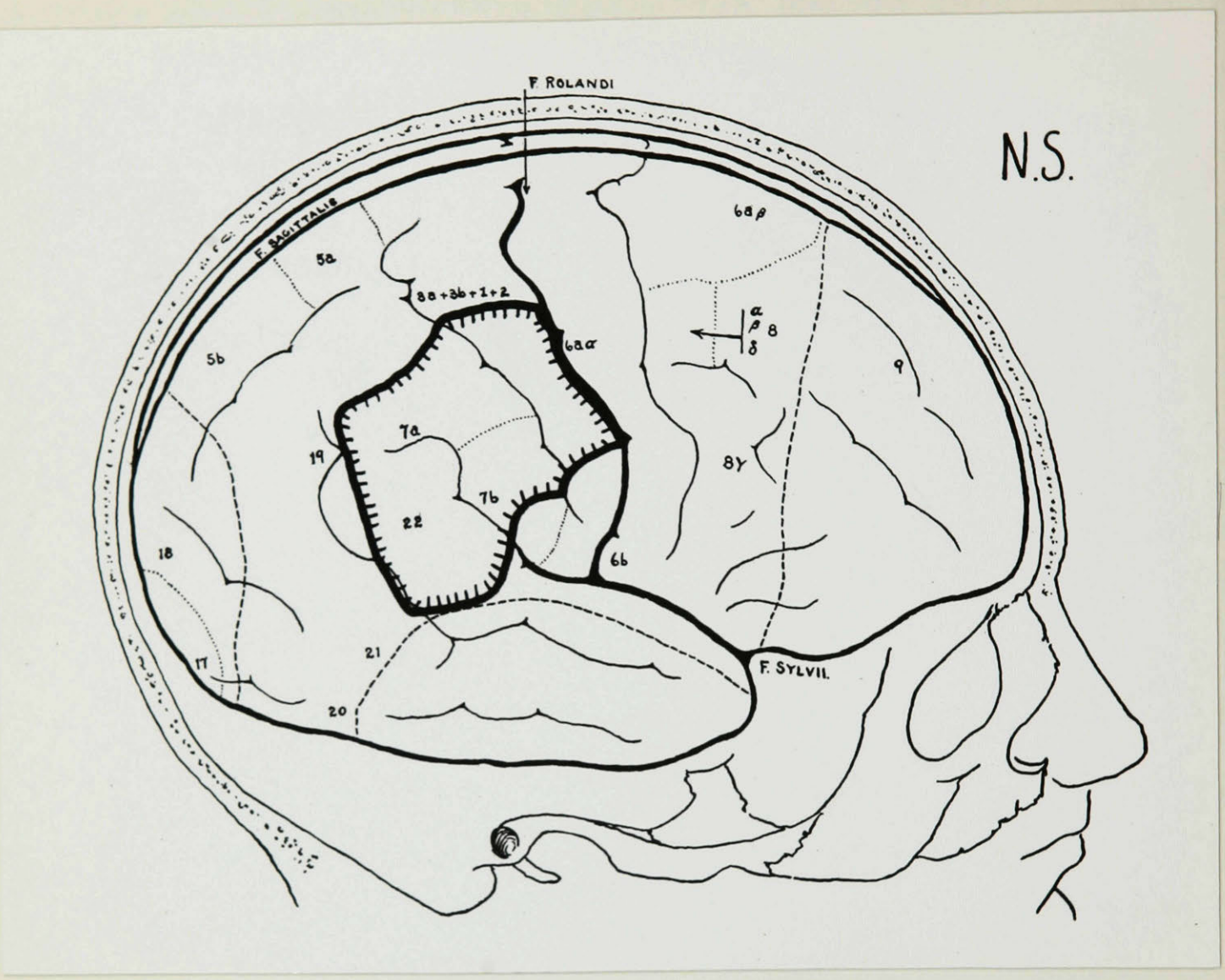

Fig. 24. Case N.S. Excision of an area from the right hemisphere in left-handed patient who was aphasic following the initial injury.

Case N.S., male, aged 28, was admitted to the Montreal Neurological Institute on February 11th, 1946 because of seizures. He was left-handed. In June 1930 he was struck by a falling tree and sustained a puncture of the right side of the skull. He was unconscious for thirty-six hours and on recovery his left side was paralyzed, and his vision and speech were markedly disturbed. A decompression was done, leaving a large skull defect in the fronto-parietal area. Following the accident he was unable to speak at all for a week, he had difficulty for about four weeks and his memory was faulty. His manner of speaking had been normal, but since the accident he developed a definite stammer.

On February 27th, 1946 a right osteoplastic craniotomy was performed by Dr. Penfield under local anaesthesia. A scar on the cortex was found extending from the posterior half of the postcentral gyrus, across the supramarginal gyrus and into the angular gyrus. The depth of the scar was 1 to 2 cm. After stimulation, the lower part of the post-central gyrus was removed and the removal was carried downward and around the upper end of the fissure of Sylvius into the first temporal convolution.

Stimulation in the region of the supramarginal gyrus produced only the slightest interference with speech.

Following the procedure he was free of aphasia. He had some attacks of weakness of the left hand, associated at the onset with some dysarthria. The hand weakness persisted for several weeks, but the speech returned to its preoperative state. The stammer remained unchanged. It was difficult to test the writing due to the weakness of the left hand, but he could write a little with the right.

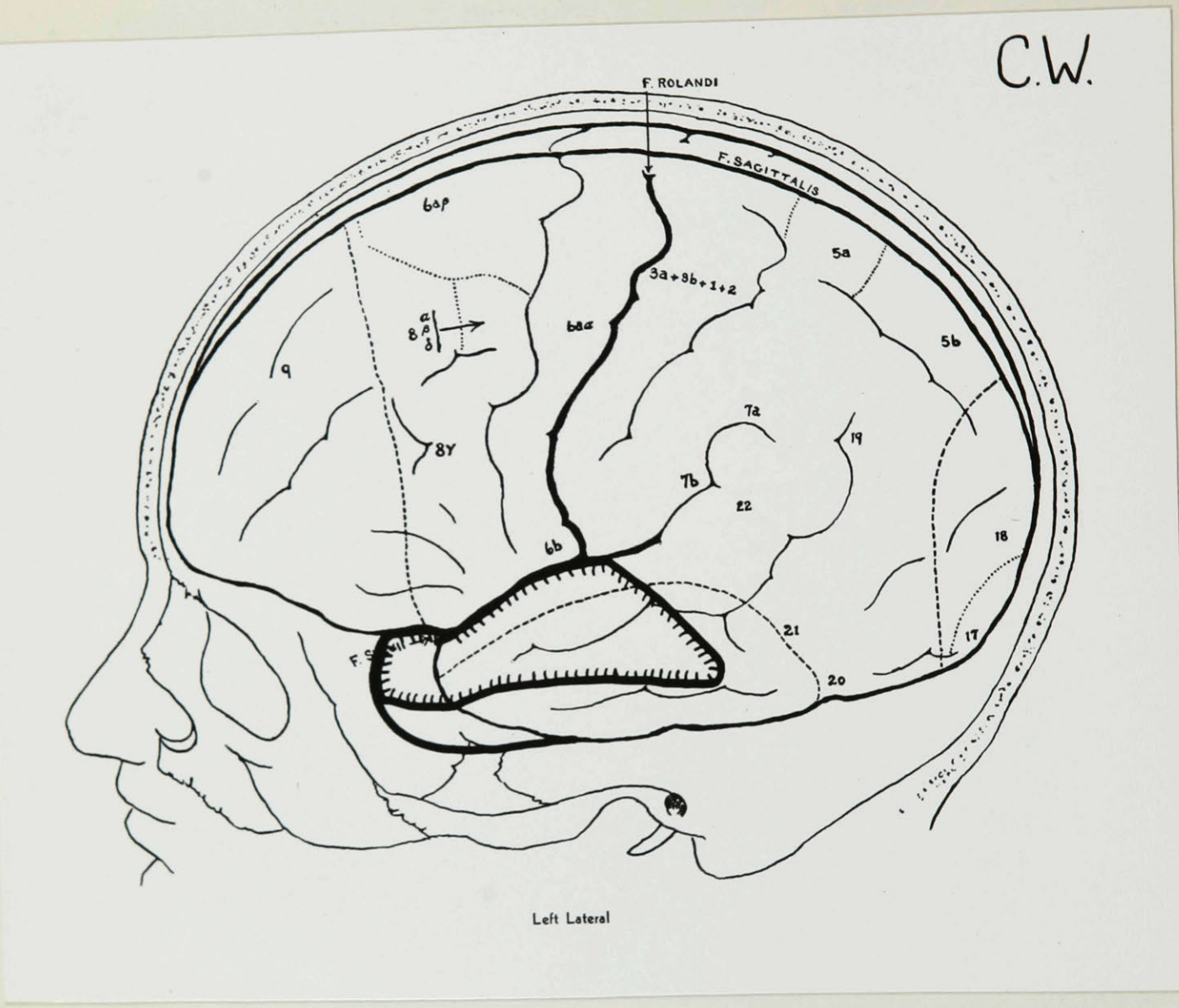

Fig. 25. Case C.W. Excision of an area in a right-handed patient who developed a transient post-operative aphasia.

Case C.W., male, aged 34, was admitted to the Montreal Neurological Institute on June 2nd, 1945 because of epileptic attacks which followed a skull fracture in 1933. He was right-handed. Electroencephalogram suggested an abnormality in the left temporal region and x-rays of the skull demonstrated an area of calcification in the same region. There was no history of any speech difficulty following the initial accident, and the speech on admission was considered normal.

On July 6th, 1945, Dr. Penfield performed a left temporal osteoplastic craniotomy under local anaesthesia. A yellowish gyrus was found at a point 5 cm. from the tip of the temporal lobe. This gyrus proved to contain the calcification. Following stimulation, 6 to 7 cm. of the first temporal convolution were removed. His speech was tested before, during and after the excision, and no change could be demonstrated. His expressive speech was normal. He named objects seen, felt and heard. Reading was not tested. Calculation was normal.

On the day following operation he developed a fairly complete aphasia. He was unable to name any object or repeat any word. However, he was not completely mute and an occasional word would slip out. He would occasionally obey an oral command and usually could imitate movements

of the examiner. He recovered slowly over twenty-seven days at which time he was considered to be normal. Six months later there was no evidence of any aphasia. He read poorly but this it was felt was as good as it had been before the accident. His schooling had been limited and he had never read much.

During the period in which he was aphasic he was given an intravenous infusion of 50 per cent dextrose. Following this the aphasia was felt to improve a little, but the improvement was not great enough to make a definite statement asto its effect.

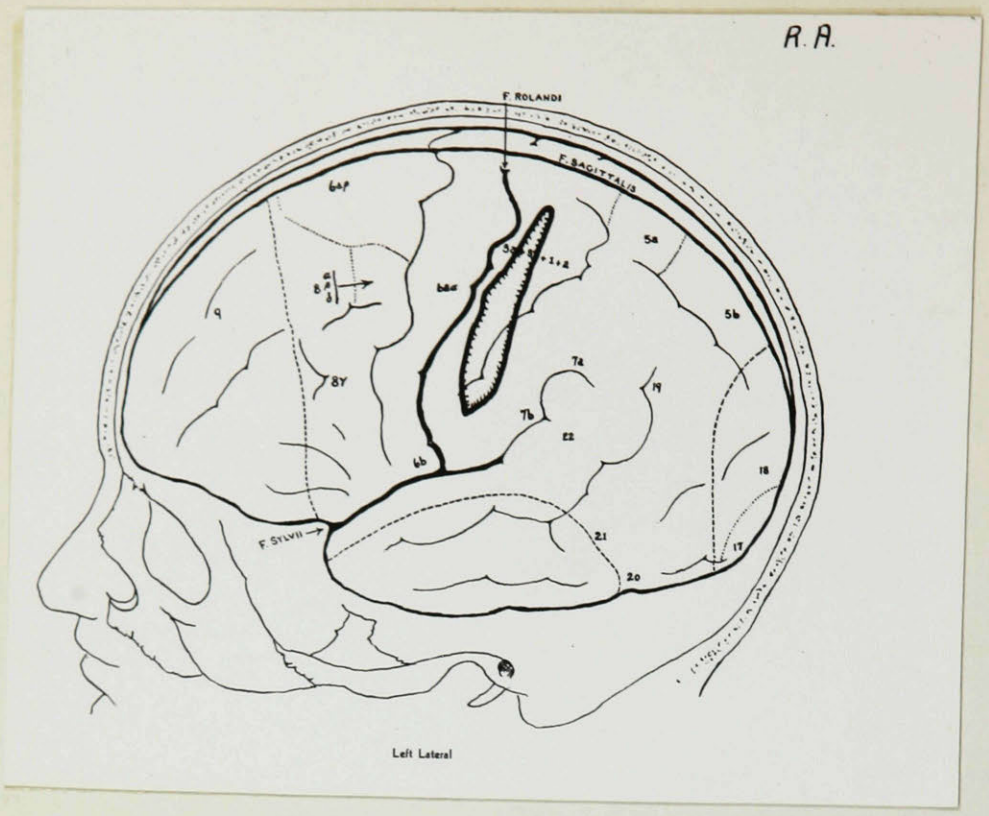

Fig. 26. Case R.A. Excision of birth injury in right-handed patient without producing aphasia.

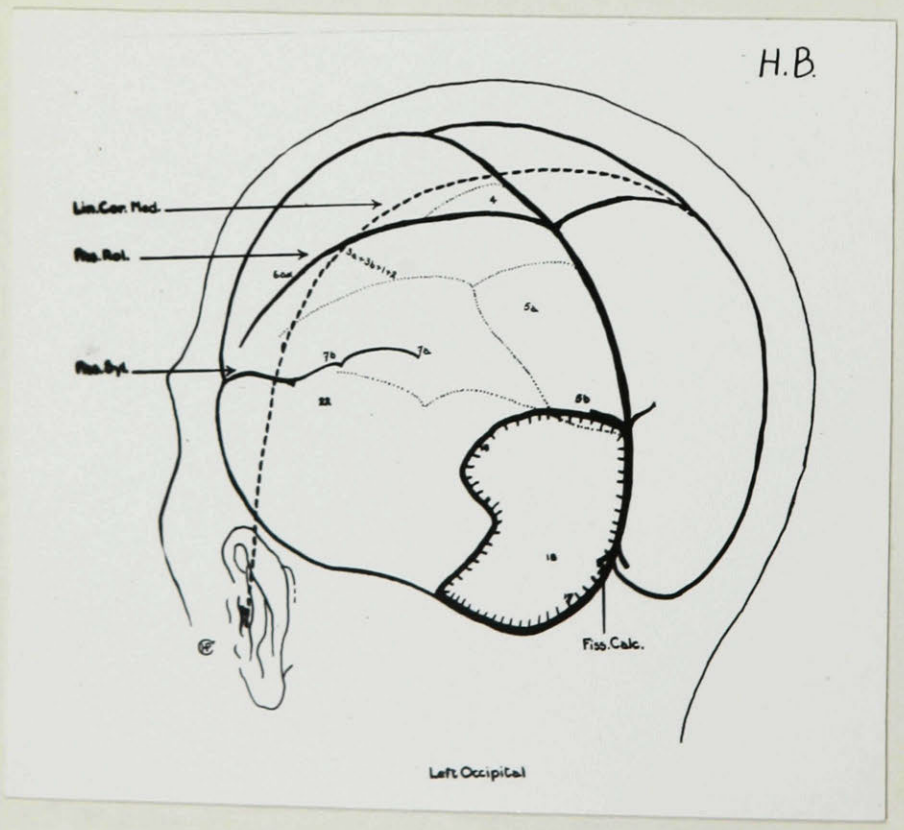

Fig. 27. Case H.B. Excision of an area in the left occipital lobe in a right-handed patient without producing any speech disturbance.

II. Summarized case reports of patients of lesser importance:

Case R.A., male, aged 15, admitted January 4th, 1940 because of seizures of fourteen years' duration. There had been a head injury at birth. He was right-handed but had been taught to write with his left. A left osteoplastic craniotomy, with excision of cicatrix from the postcentral region, was carried out. There was no postoperative aphasia.

Case H.B., male, aged 13 was admitted March 10th, 1939 because of seizures since birth. He was right-handed. A left occipital osteoplastic craniotomy and excision of a cicatrix from the occipital area was performed without producing any disturbance in speech.

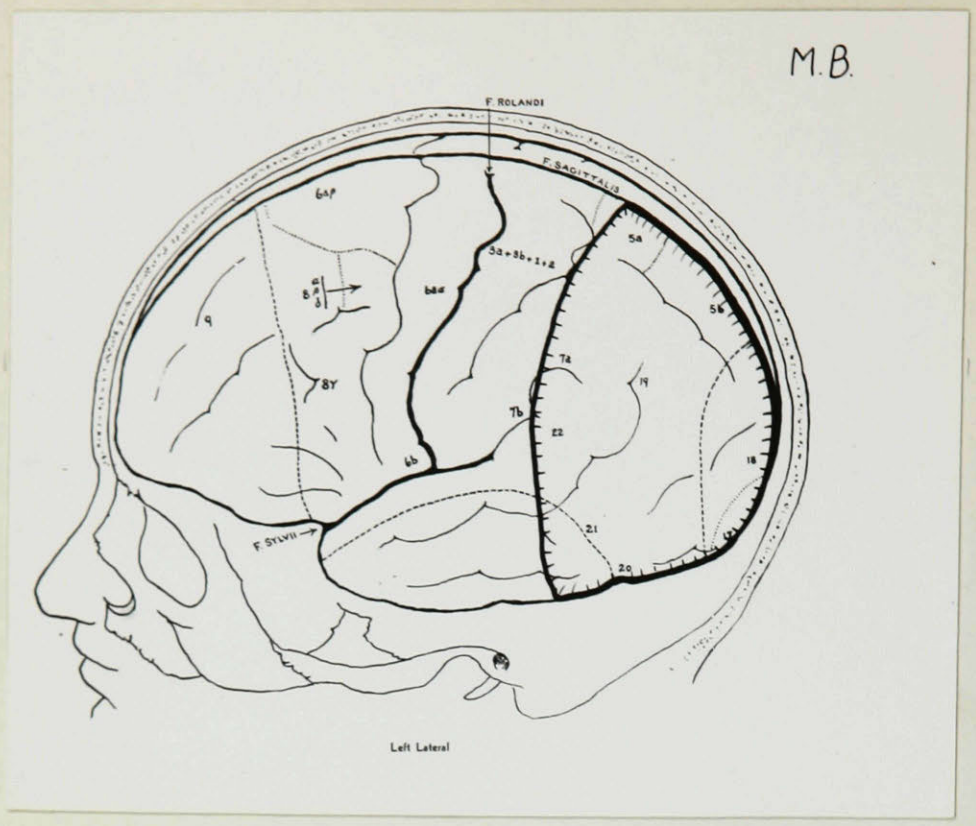

Fig. 28. Case M.B. Excision of a large cicatrix from the posterior third of the left hemisphere in a right-handed patient without affecting speech.

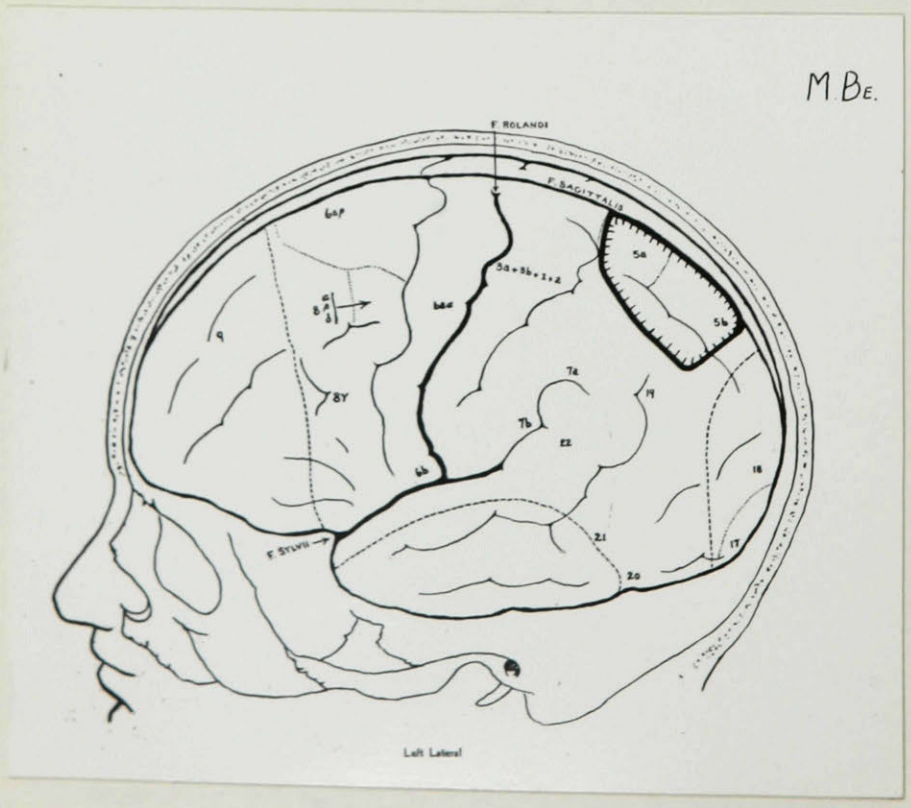

Fig. 29. Case M.Be. Excision of an area in a right-handed patient producing a transient post-operative aphasia.

Case M.B., male, aged 20 was admitted November 27th, 1940 because of seizures of 13 years' duration. He had been injured at birth and was right-handed. A left parietal osteoplastic craniotomy, with excision of cicatrix and cyst in the posterior third of the left hemisphere, was performed. There was no postoperative aphasia.

Case M. Be., female, aged 30 was admitted on March 17th, 1936 because of seizures of sixteen years' duration. There was a history of head injury at birth. She was right-handed. A left osteoplastic craniotomy and excision of an area of atrophy from the mid-parietal region was performed. An expressive type of aphasia developed on the second post-operative day and lasted nineteen days.

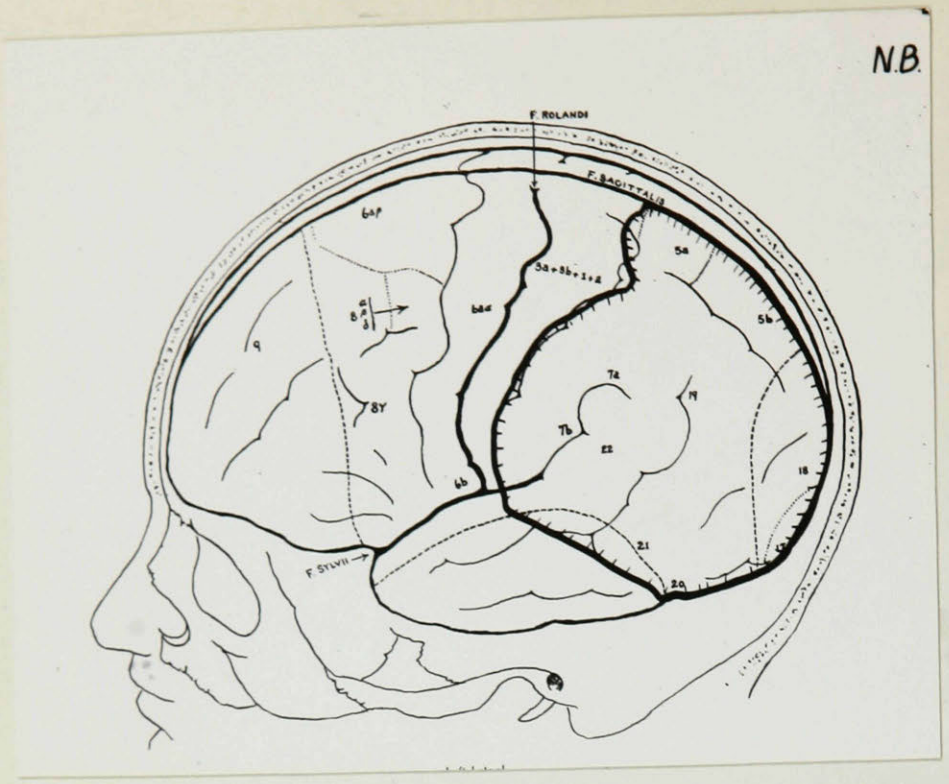

Fig. 30. Case N.B. Excision of large cyst from the posterior half of the left hemisphere in a right-handed patient without affecting speech.

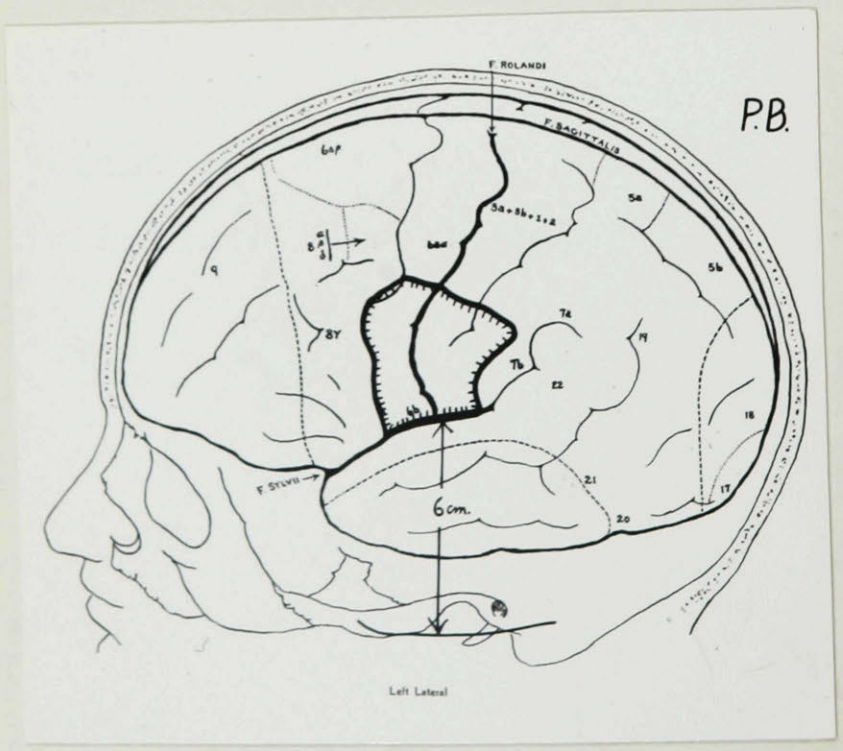

Fig. 31. Case P.B. Excision of area from left supramarginal region in left-handed patient, who became aphasic and recovered completely.

Case M.B., male, aged 23 was admitted on May 10th, 1939 because of seizures of three years' duration. The age at the time of injury was not known, but it was probably at birth. He was right-handed. A left posterior osteoplastic craniotomy and excision of a cyst and cicatrix from the posterior half of the left hemisphere was carried out. There was no postoperative aphasia.

Case P.B., male, aged 35 was admitted on March 20th, 1946 because of epileptic seizures for 33 years. There was a history of right-sided paresis which started with a series of convulsions at the age of three years. He was left-handed, the right one having been weaker than the left. A left osteoplastic craniotomy was performed andan area was excised from above the fissure of Sylvius. Following the operation he had an expressive type of speech disturbance, which returned to normal in two weeks.

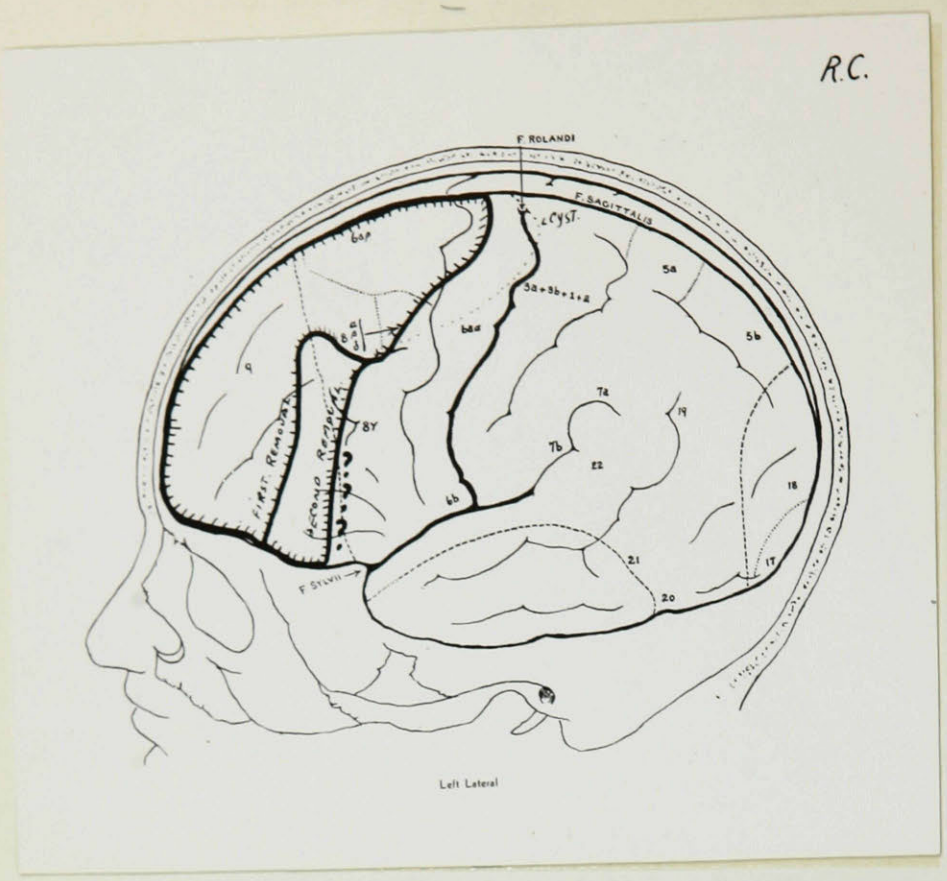

Fig. 32. Case R.C. Excision of an area from the left frontal lobe in a right-handed patient without producing a permanent aphasia.

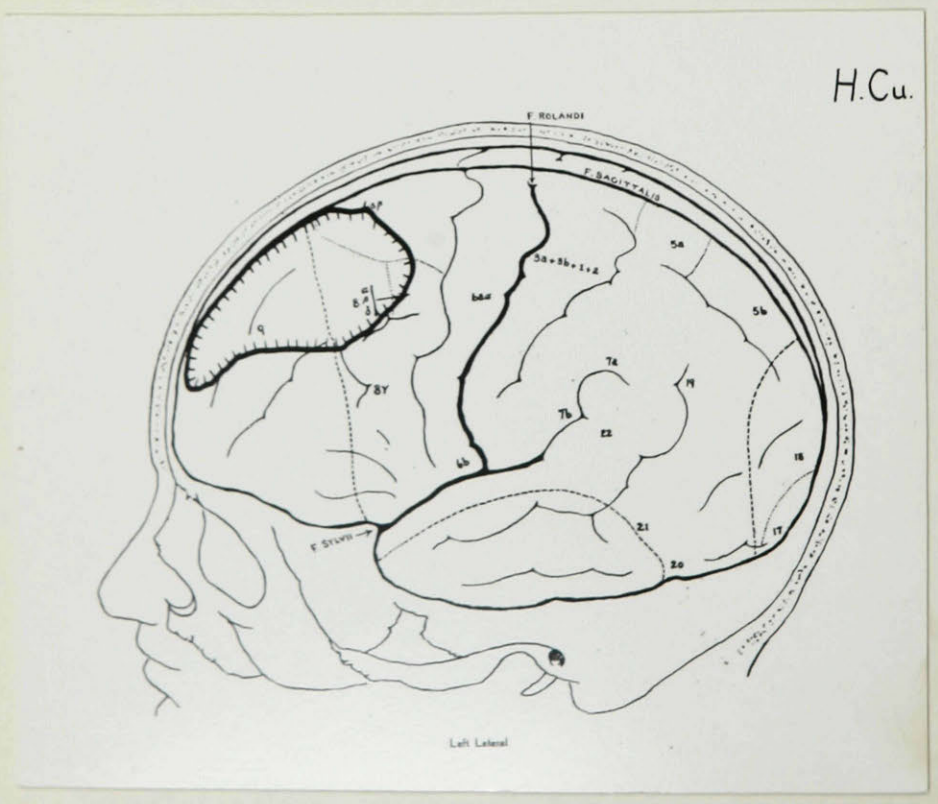

Fig. 33. Case H.Cu. Excision of an area from the left frontal lobe in a right-handed patient producing a transient postoperative aphasia.

Case R.C., male, aged 15 was admitted on January 20th, 1941 and again on June 29th, 1942 because of seizures. He had been injured at birth and was right-handed. A left osteoplastic craniotomy was performed and a cicatrix was removed from the left frontal region. Further excision was carried out on the second admission. There was no postoperative aphasia.

Case H.Cu., male, aged 38 was admitted on October 23rd, 1944 because of seizures of twenty-one years' duration. At the age of 8 years he had sustained a depressed fracture of the skull. He was right-handed. A left osteoplastic craniotomy and excision of cicatrix from the frontal lobe was performed, followed by a secondary postoperative aphasia of predominantly expressive type which lasted thirty days.

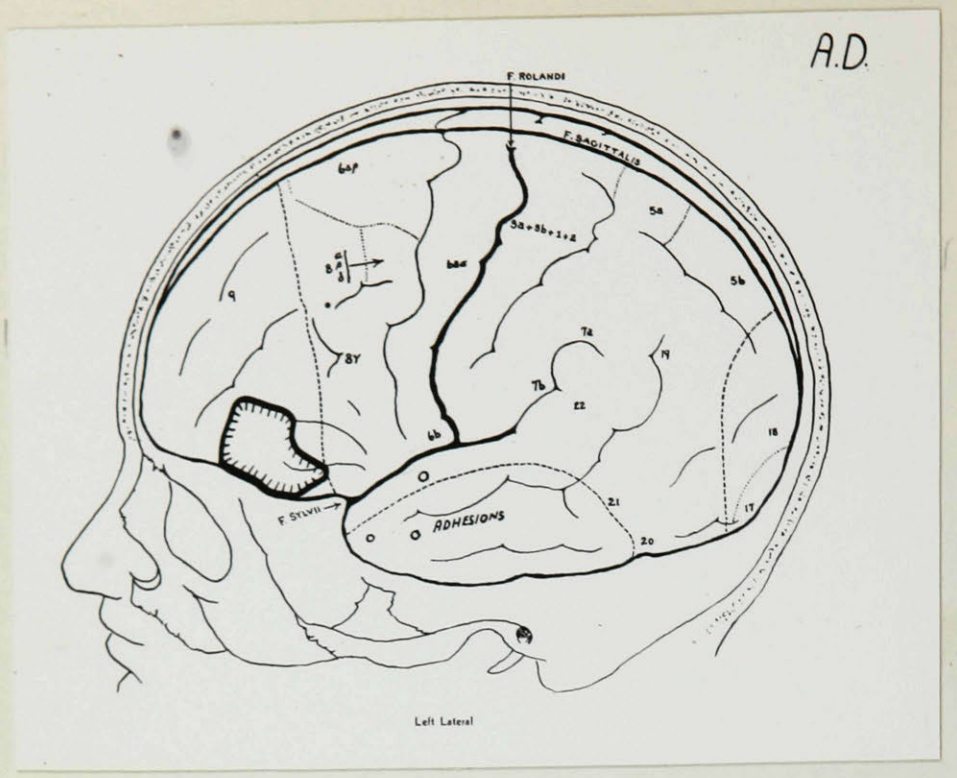

Fig. 34. Case A.D. Excision of area from lower part of left frontal lobe in a right-handed patient without affecting speech.

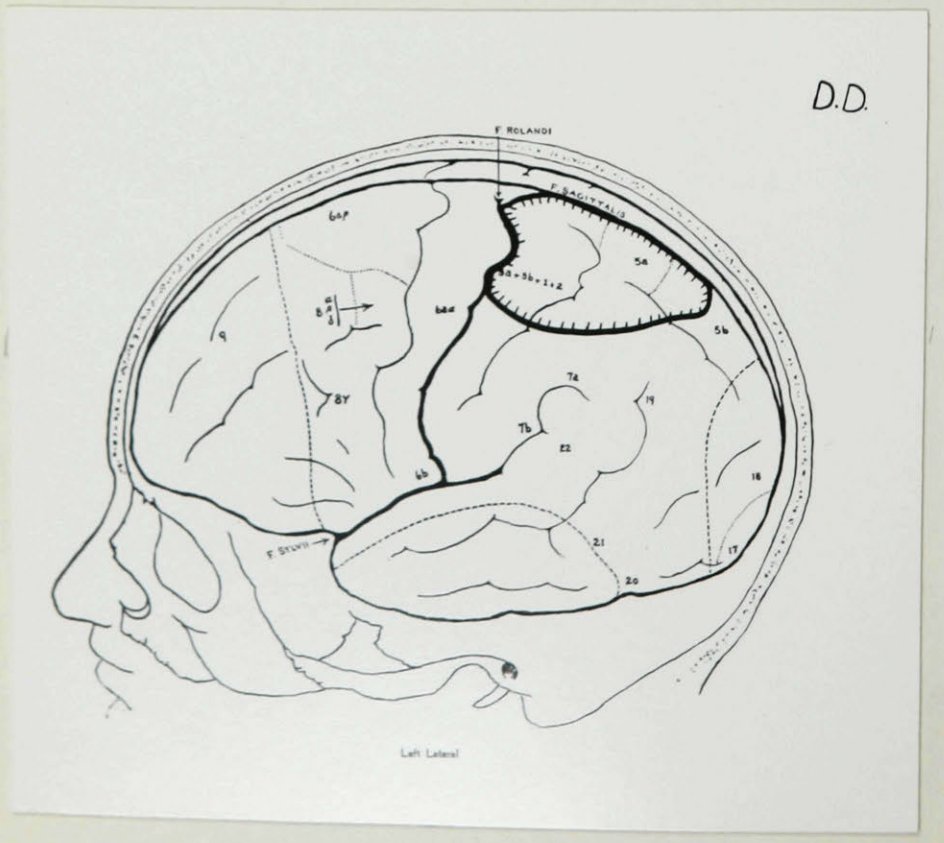

Fig. 35. Case D.D. Excision of an area of the left parietal lobe in a right-handed patient producing a transient secondary post-operative aphasia.

Case A.D., male, aged 12 was admitted on July 17th, 1940 because of seizures of one and a half years' duration. The time of injury was not known. He was right-handed. A left osteoplastic craniotomy and excision of cicatrix from the lower part of the frontal lobe was performed. There was no postoperative aphasia.

Case D.D., male, aged 16 was admitted on July 18th, 1934 because of seizures of two years' duration. The time of injury was not known. He was right-handed. A left osteoplastic craniotomy and excision of a cicatrix from the parietal region was performed. A mixed type of aphasia developed on the second postoperative day and lasted twenty-one days.

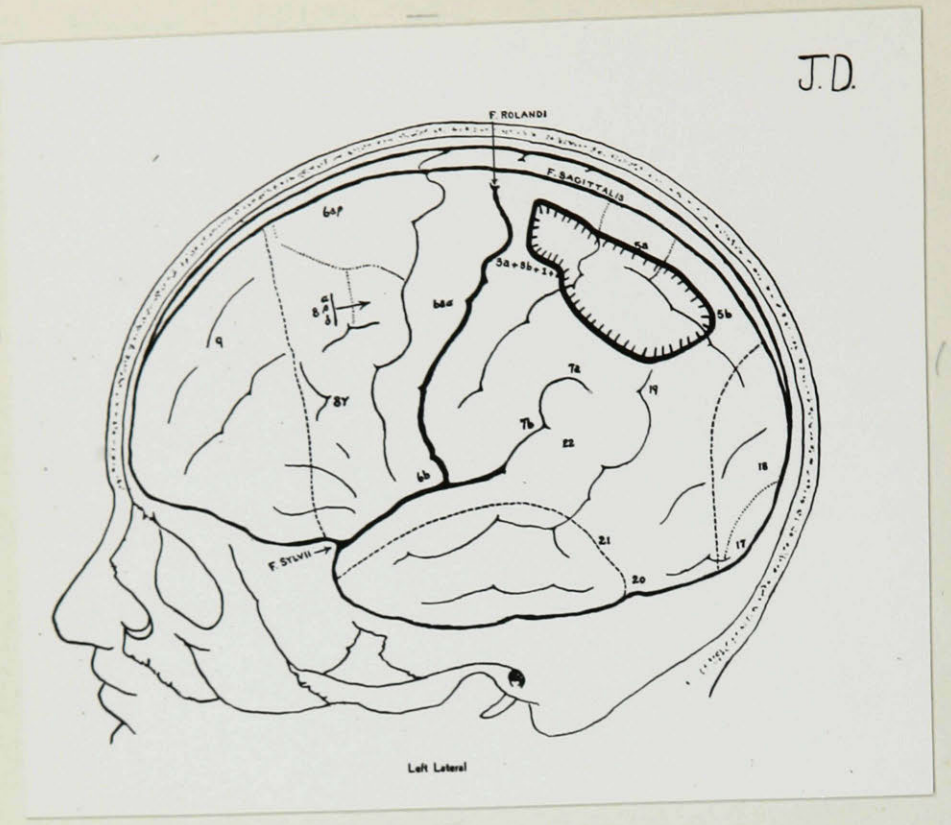

Fig. 36. Case J.D. Excision of area of the left hemisphere in a right-handed patient producing a secondary, transient postoperative aphasia.

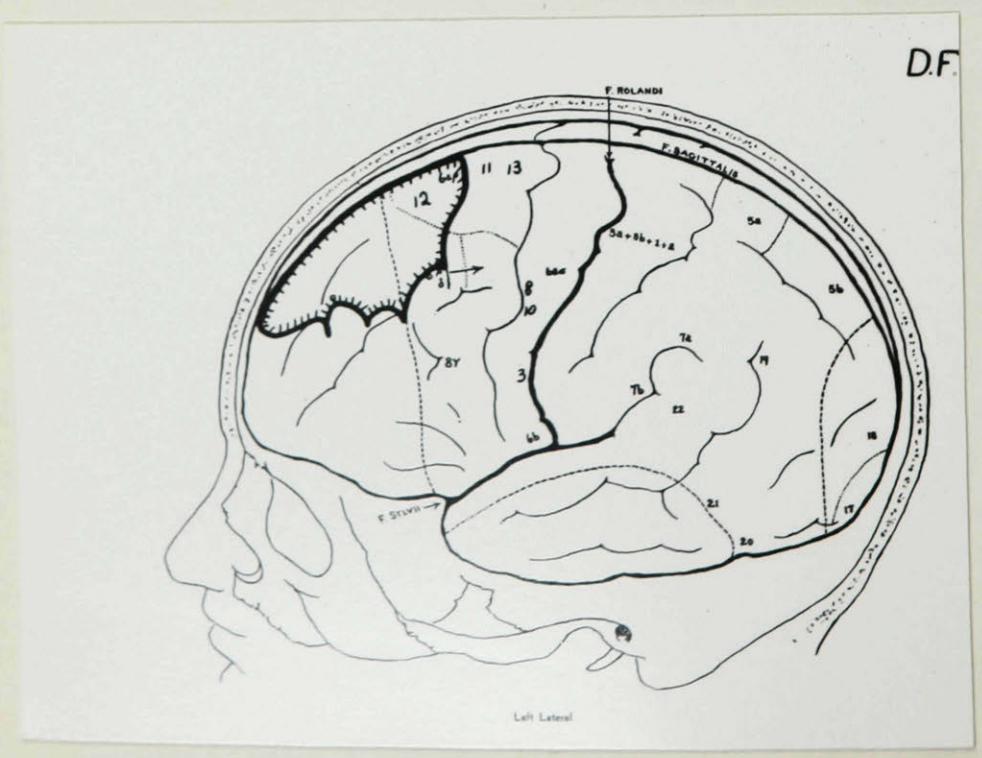

Fig. 37. Case D.F. Excision of area from the left frontal lobe in a right-handed patient without producing a postoperative aphasia.

Case J.D., female, aged 17 was admitted on November 17th, 1933 because of seizures of three years' duration. The age at the time of injury was not known. She was right-handed. A left osteoplastic craniotomy, with subtemporal decompression and excision of cerebral cicatrix, was performed. On the fifth postoperative day she developed a mixed type of aphasia which lasted three weeks. Immediately following the operation it was noticed that the left side was completely paralysed. After reviewing the case it was felt that she probably had had a vascular lesion of the internal capsule. The paralysis of the right hand persisted. The onset of the aphasia did not coincide with the onset of the paralysis, and the aphasia was transient.

Case D.F., female, aged 29 was admitted on November 27th, 1945 because of seizures of 16 years' duration. At the age of 13 years she had a fracture of the frontal bone. She was right-handed. A left osteoplastic craniotomy and excision of a cerebral cicatrix from the left frontal lobe, near the midline, was performed. There was no postoperative aphasia.

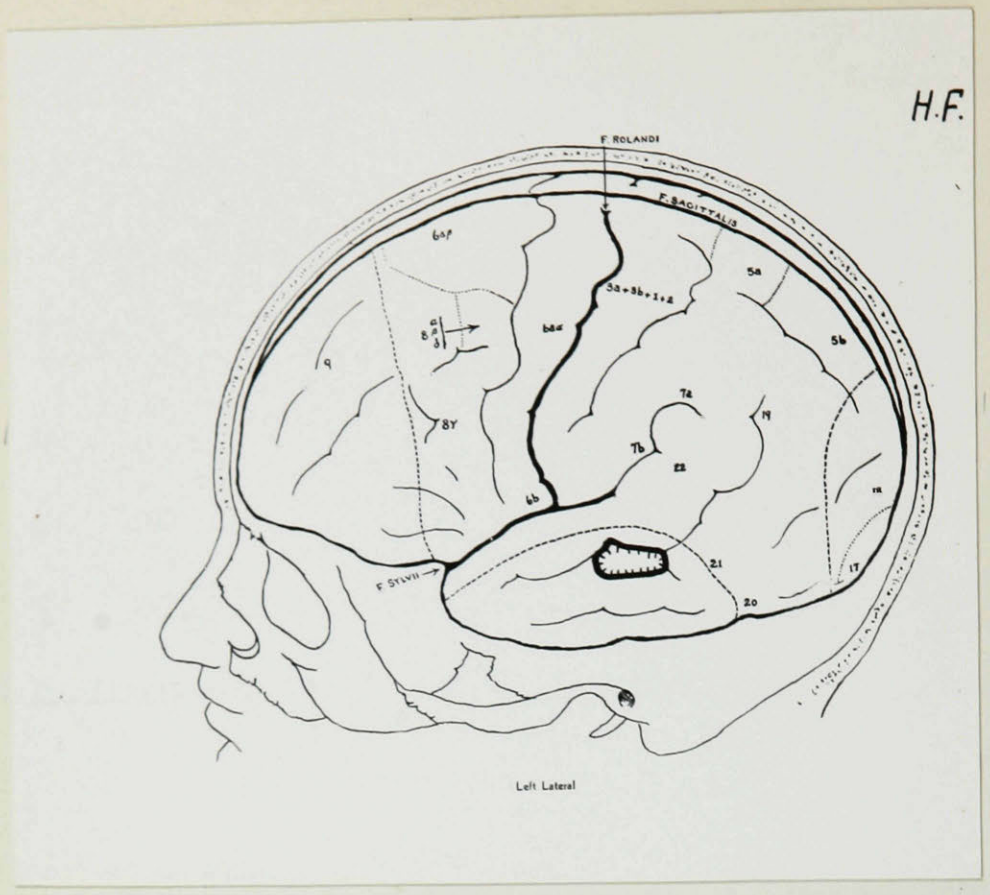

Fig. 38. Case H.F. Excision of small area from the left temporal lobe in a right-handed patient, followed by a secondary, transient aphasia.

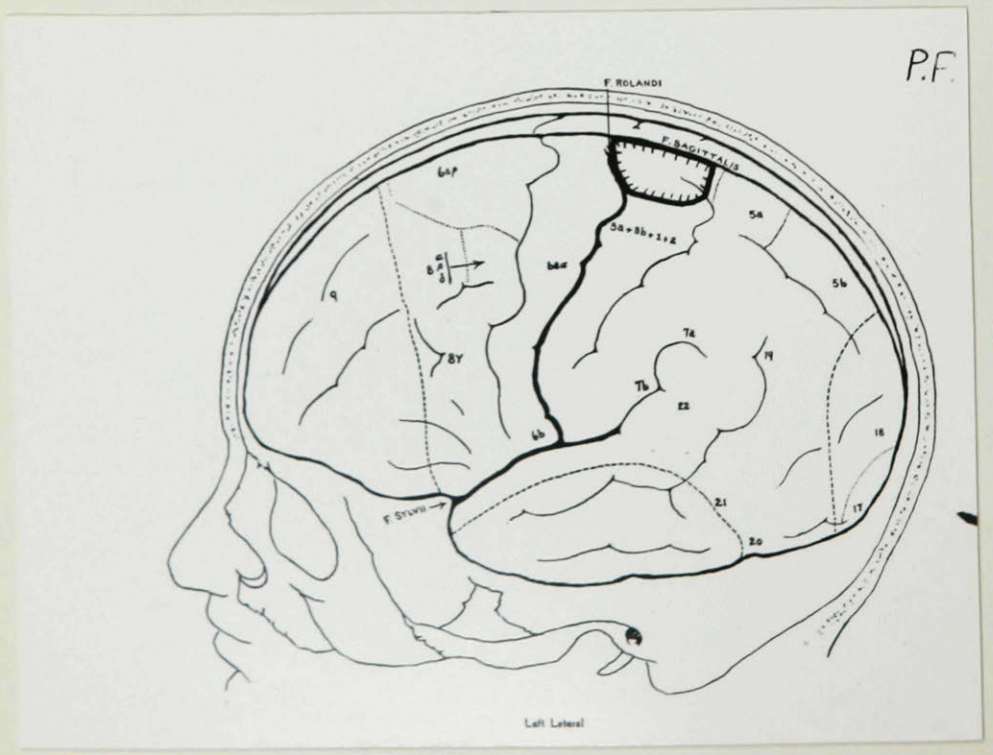

Fig. 39. Case P.F. An area excised from the left postcentral region near the midline in a right-handed patient without producing aphasia.

Case H.F., male, aged 16 was admitted on January 11th, 1939 because of seizures which had been present since birth. He was right-handed. A left osteoplastic craniotomy was performed and a small area was excised from the temporal lobe. He developed a mixed type of aphasia on the first postoperative day which lasted one hundred days. The postoperative period was complicated by an infection which may have been a factor in the prolonged aphasia.

Case P.F., male, aged 20 was admitted on March 13th, 1940 because of seizures of 13 years' duration. The age at the time of injury was not known. He was right-handed. A left osteoplastic craniotomy and excision of an abnormal gyrus from the postcentral region, near the midline, was performed. There was no postoperative aphasia.

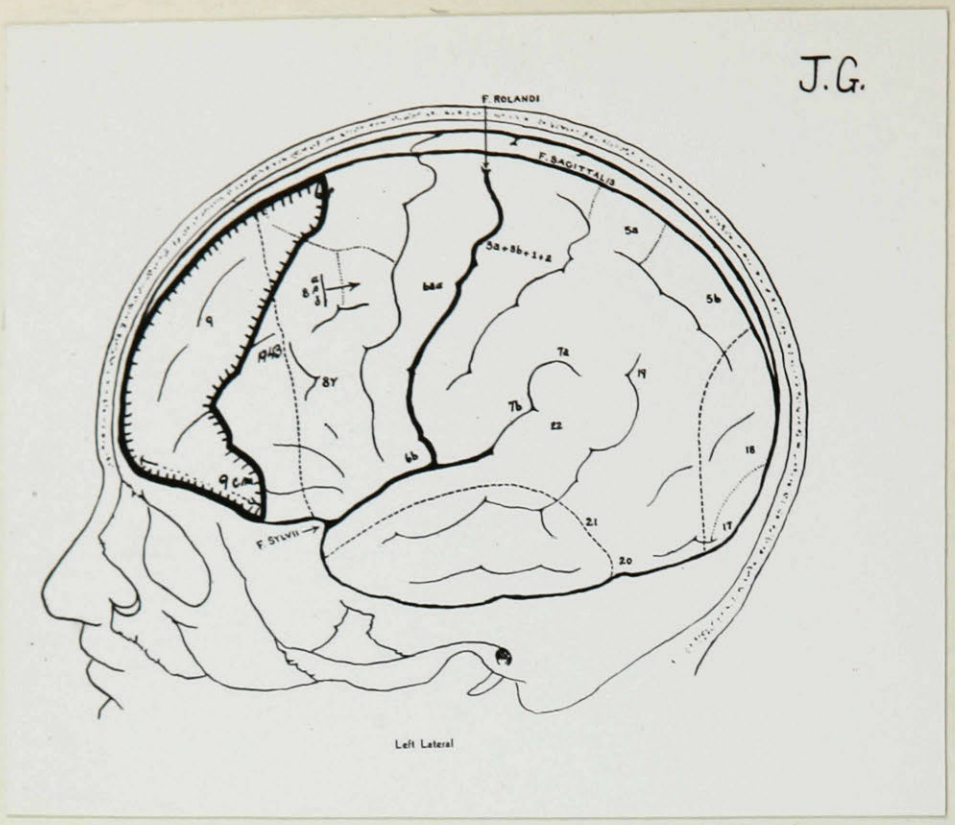

Fig. 40. Case J.G. Excision of part of left frontal lobe in two stages in a right-handed patient without producing aphasia.

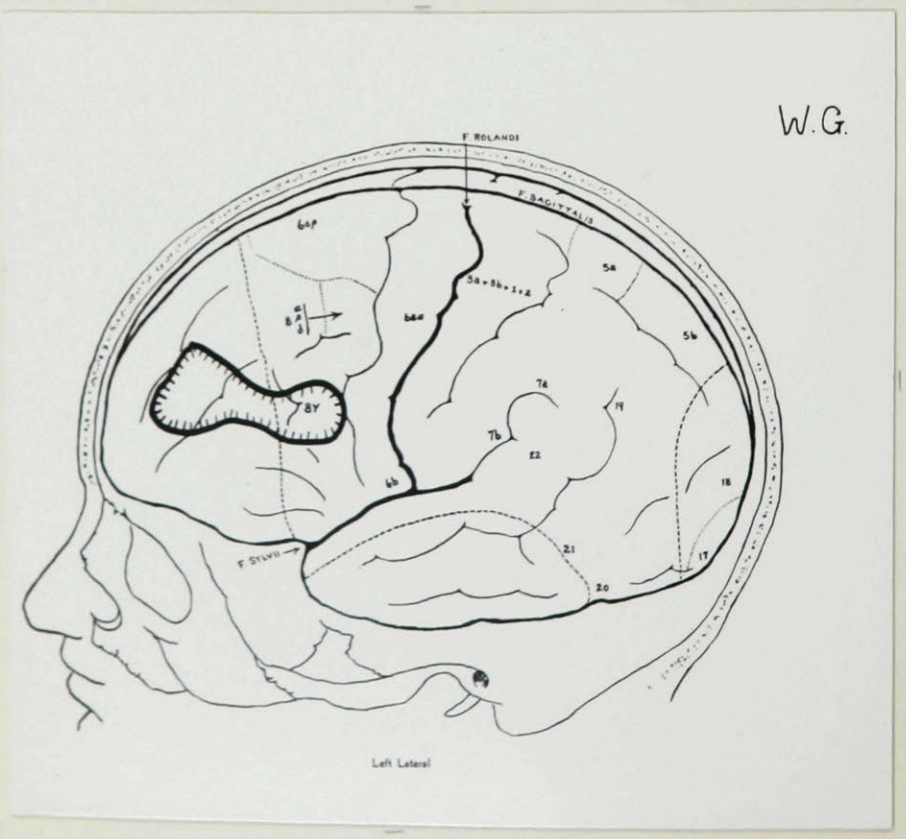

Fig. 41. Case W.G. Excision of area from left frontal lobe in a right-handed patient without producing an aphasia.

Case J.G., male, aged 32 was admitted on October 14th, 1943 because of seizures which followed damage to the frontal lobe when a craniopharyngeal pouch was being exteriorized. He was right-handed and was blind. Two removals were carried out through a left osteoplastic craniotomy. The first in 1938 and the second in 1943. A large part of the frontal lobe was excised. There was no postoperative aphasia.

Case W.G., male, aged 29 was admitted on August 17th, 1945 because of seizures of one year's duration. This followed a compound fracture of the left fronto-parietal region. He had had difficulty in speaking following the injury. He was right-handed. A left osteoplastic craniotomy and excision of a cicatrix from the left frontal region was performed. Nupercain was injected into the brain without effect. There was no postoperative aphasia.

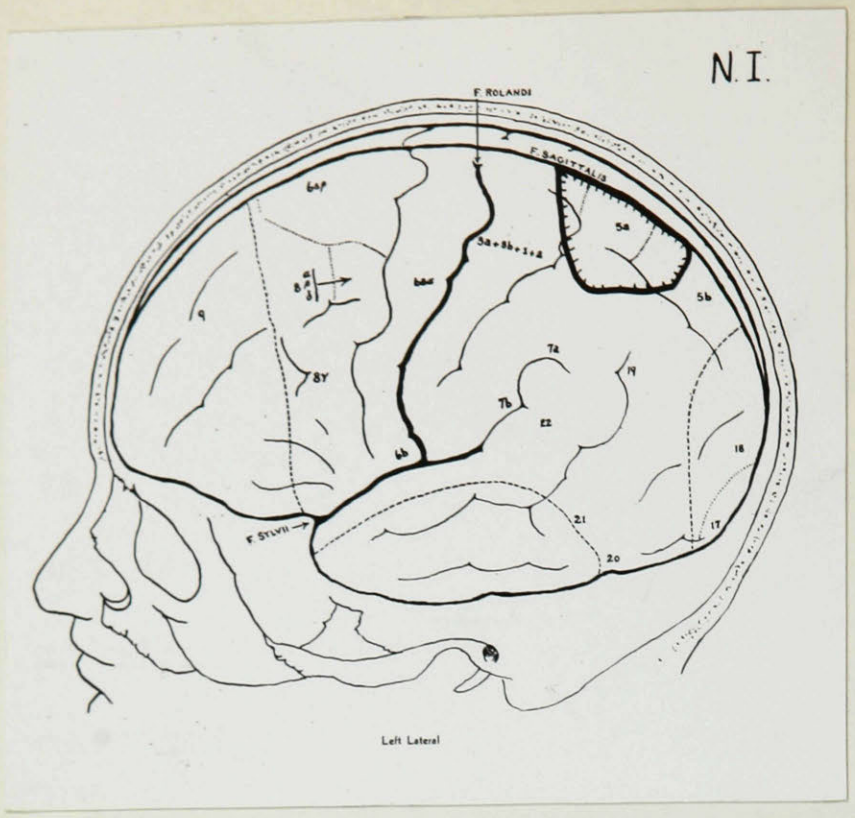

Fig. 42. Case N.I. Excision of area from left parietal lobe in a right-handed patient producing a transient, secondary aphasia.

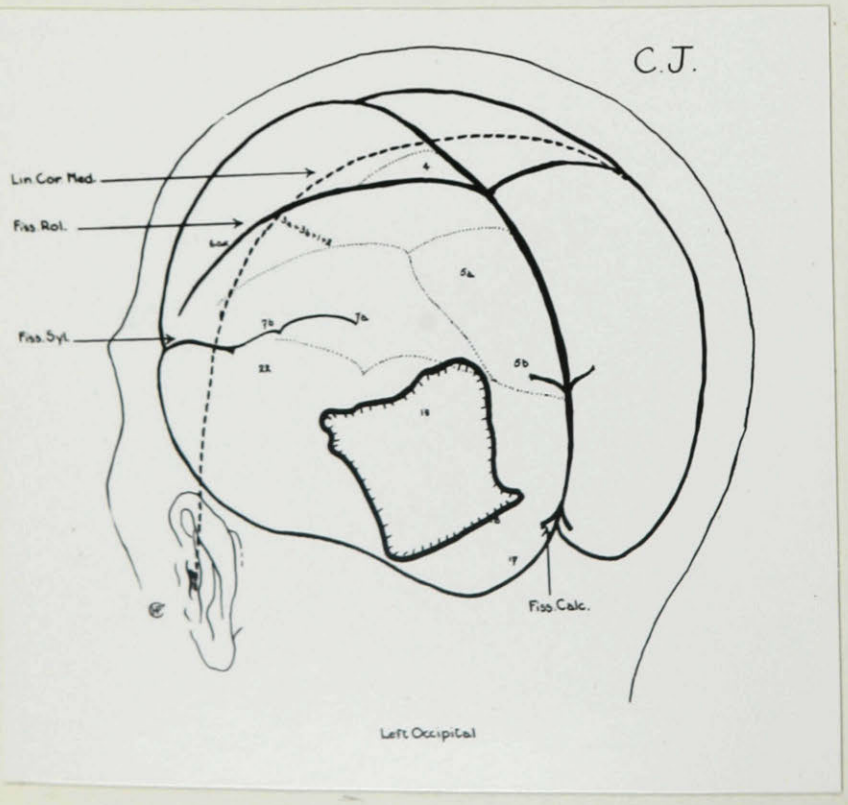

Fig. 43. Case C.J. Excision of a normal appearing area of the left occipital lobe in a left-handed patient who developed a secondary, transient aphasia.

Case N.I., male, aged 16 was admitted on May 29th, 1938 because of seizures which followed a febrile illness at the age of a year and a half. He had originally been left-handed but he changed to the right when he started to school. He wrote, ate, and played games with the right hand. A left osteoplastic craniotomy and excision of an abnormal convolution from the parietal region was performed. He developed a mixed type of aphasia on the second post-operative day which lasted twenty-seven days.

Case C.J., male,,aged 18 was admitted on July 14th, 1942 because of seizures of six years' duration. He had had a birth injury with an associated hemiparesis. He was left-handed, the right being smaller and weaker than the left. A left occipital craniotomy and excision of a large part of the occipital lobe was performed. The cortex removed appeared normal, but was producing abnormal electrographic waves. A mixed type of aphasia, chiefly in the field of written language developed on the third postoperative day and lasted thirty days.

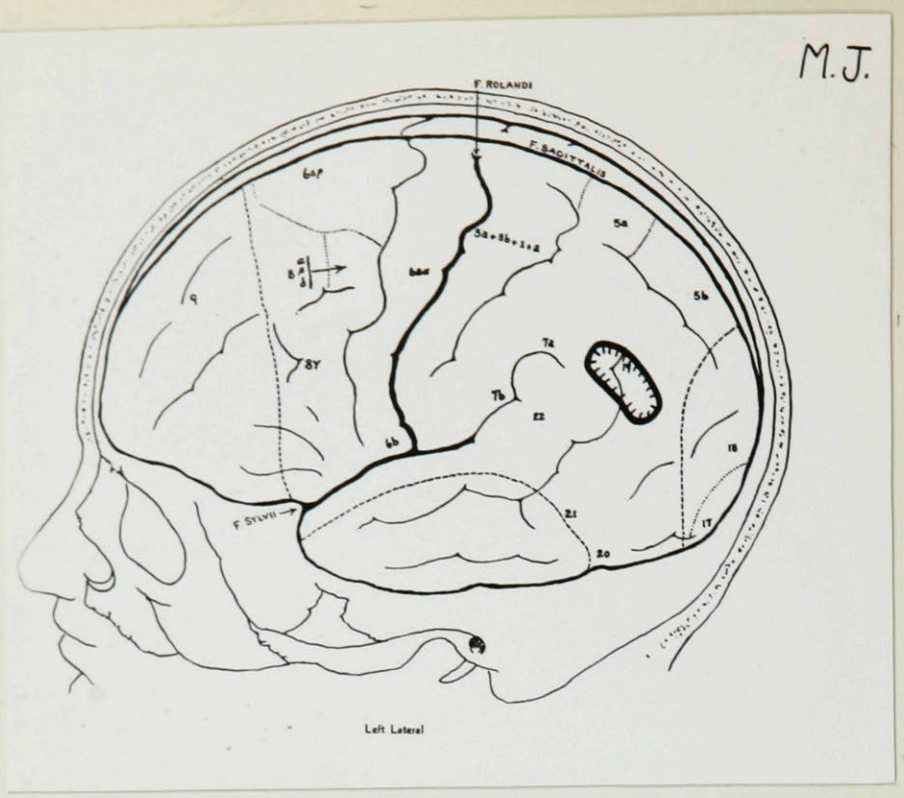

Fig. 44. Case M.J. Excision of an area from near the angular gyrus. in right-handed patient with a secondary aphasia with questionable recovery.

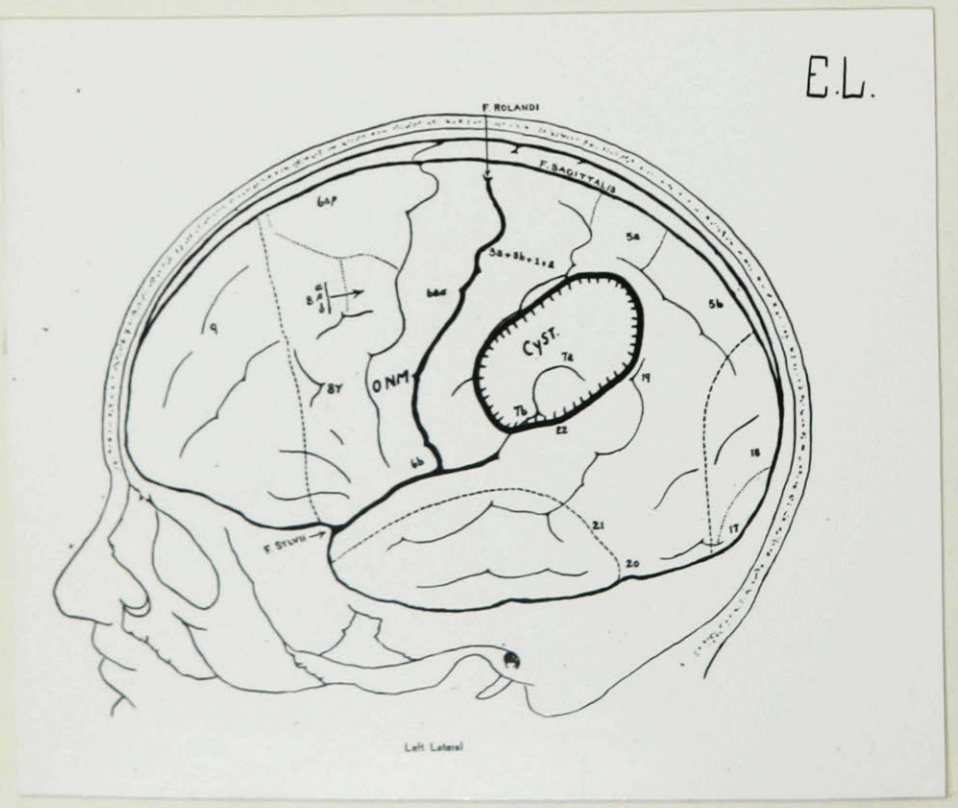

Fig. 45. Case E.L. Opening of a cyst in the left parietal area in a right-handed patient followed by a secondary, transient aphasia.

Case M.J., male, aged 30 was admitted on March 10th, 1935 because of two seizures associated with a speech disturbance and weakness of the right hand. He was right-handed. A left osteoplastic craniotomy was performed and a small area was excised from the vicinity of the angular gyrus. When he regained consciousness he was able to speak clearly in French and English. He then developed a mixed type of aphasia from which he recovered. A year later a friend wrote, as the patient was unable to do so himself. The information which we have been able to obtain on this patient is very inadequate.

Case E.L., female, aged 39 was admitted on June 2nd, 1936 because of seizures of six years' duration. At the age of thirty-three years she had probably had a vascular lesion of the brain. She was right-handed. A left osteoplastic craniotomy was performed and a cyst in the parietal region was opened. The wall of the cyst was not removed. An aphasia, not well classified, developed on the first post-operative day and lasted fifteen days.

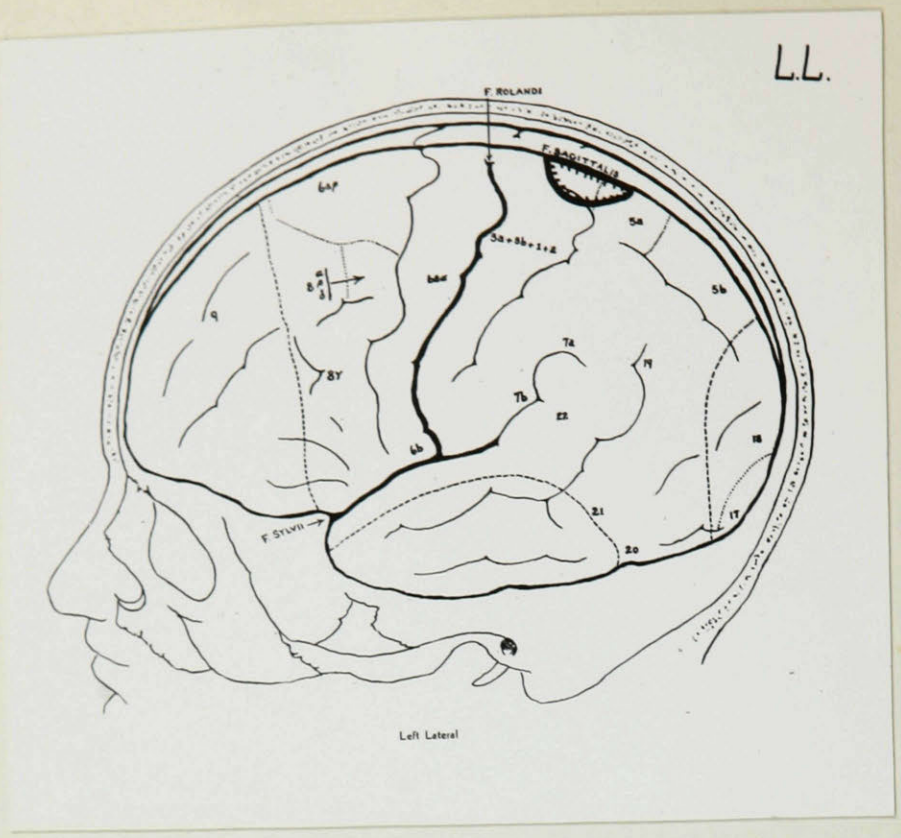

Fig. 46. Case L.L. Excision from the left parietal region at the midline in a right-handed patient producing a secondary, transient aphasia.

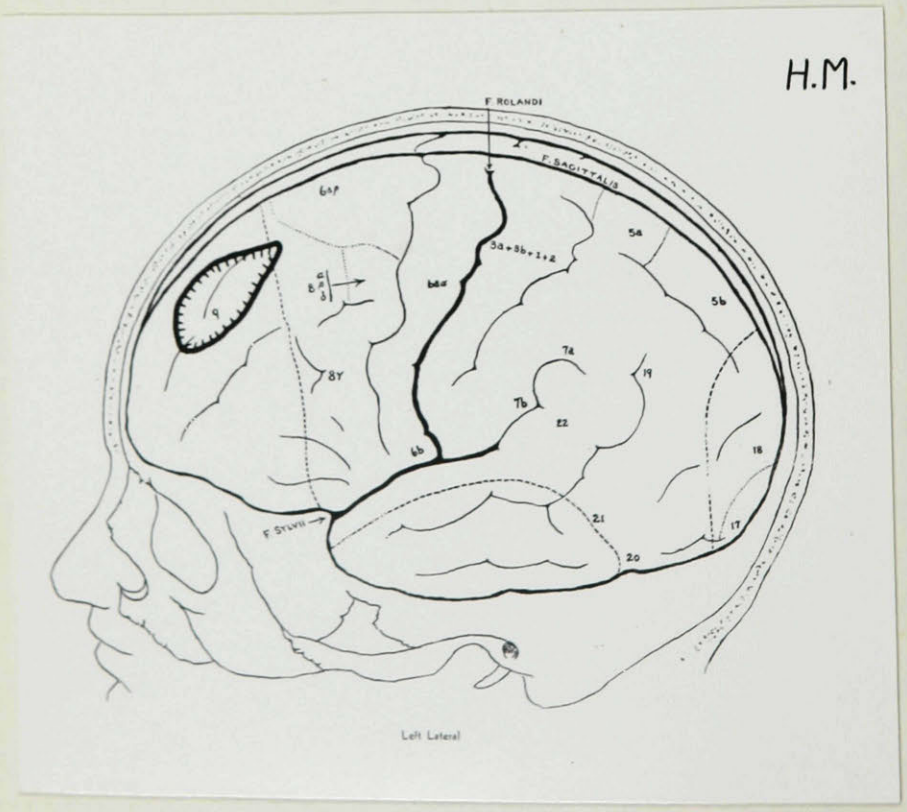

Fig. 47. Case H.M. Excision of area of left frontal lobe in a right-handed patient without producing any aphasia.

Case I.L., male, aged 11 was admitted on November 12th, 1937 because of seizures of one year's duration. The cause of the injury was not known. He was right-handed. A left osteoplastic craniotomy was performed and an area was excised from the parietal region. A mixed type of aphasia developed on the second post-operative day and lasted thirty days.

Case H.M., male, aged 17 was admitted on July 14th, 1942 because of seizures of four years' duration. The time of injury was not known. He was right-handed. A left osteoplastic craniotomy and removal of abnormal gyri from the frontal lobe was performed. There was no post-operative aphasia.

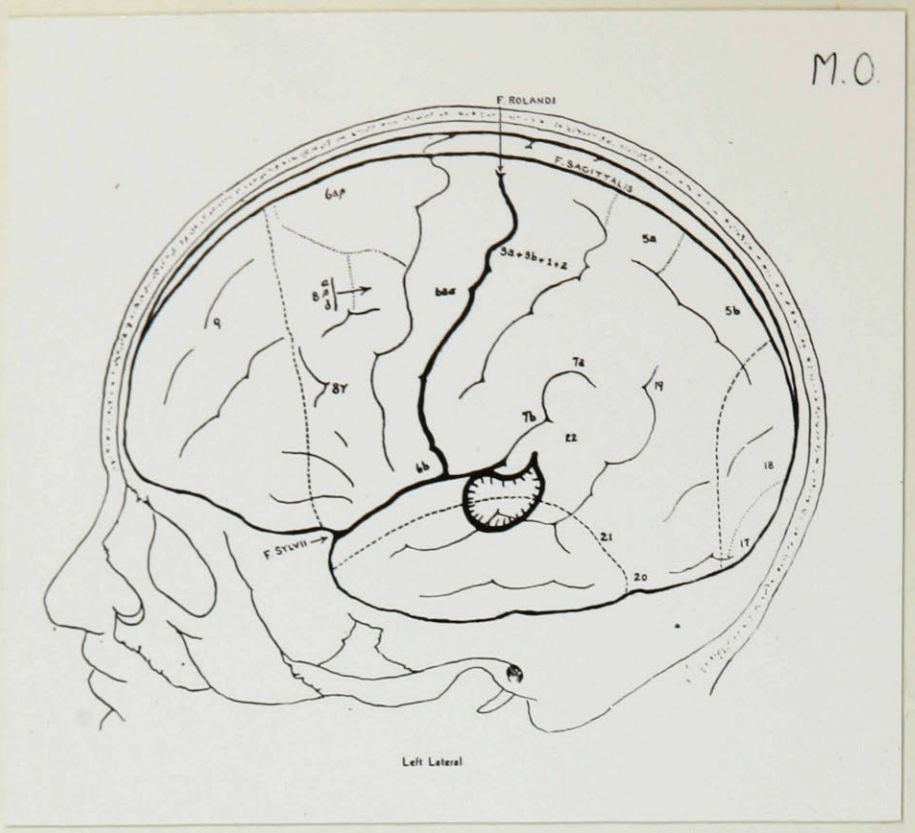

Fig. 48. M.O. Excision of an area from the left temporal lobe in a right-handed patient without producing a permanent aphasia.

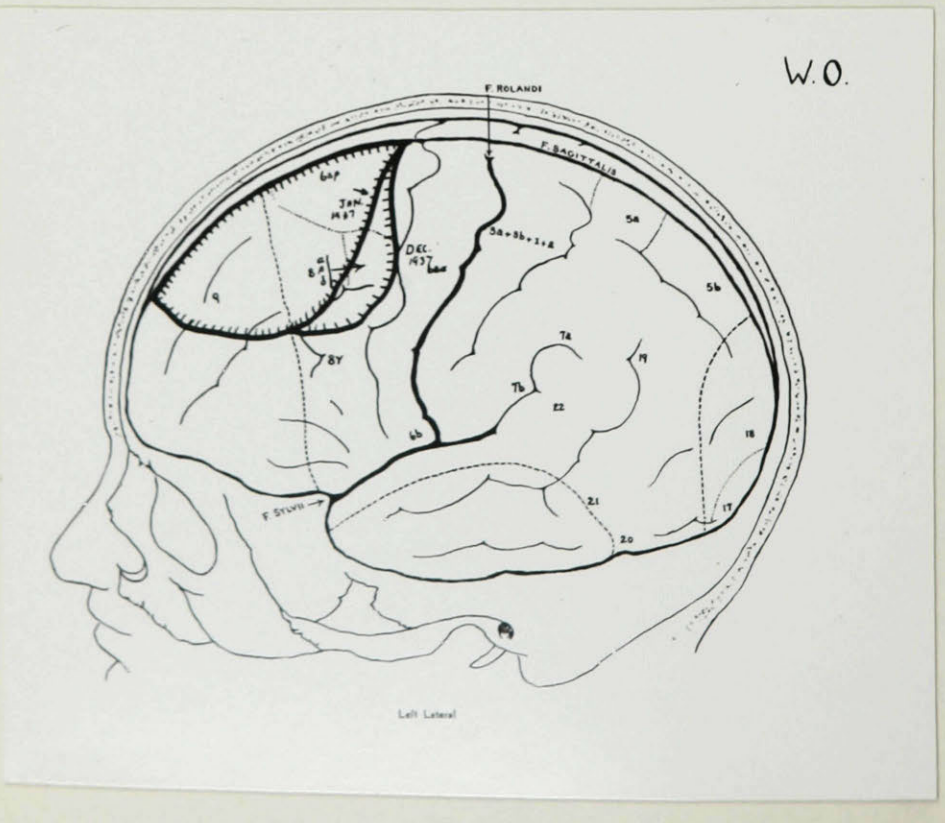

Fig. 49. W.O. Excision of area from the left hemisphere in a right-handed patient without producing aphasia.

Case H.O., male, aged 26 was admitted on two occasions, May 1937 and May 1939, because of seizures which followed whooping cough at the age of five years. He had been aphasic and hemiplegic after the illness. An exploratory craniotomy was done in 1937 and a second osteoplastic craniotomy in 1939 at which time an area of the temporal lobe was excised. There was no postoperative aphasia.

Case W.O., male, aged 22 was admitted on two occasions, Jan. 16th, 1937 and Nov. 25th, 1937 because of seizures of five years' duration. The age of the time of injury was not definite. He was right-handed. A left osteoplastic craniotomy and excision of a cyst and an arteriovenous aneurysm was performed. The attacks persisted so a further excision was carried out ten months later. Following the excision he had seizures during which he would lose the ability to speak, although he would not become unconscious. Otherwise there was no postoperative aphasia.

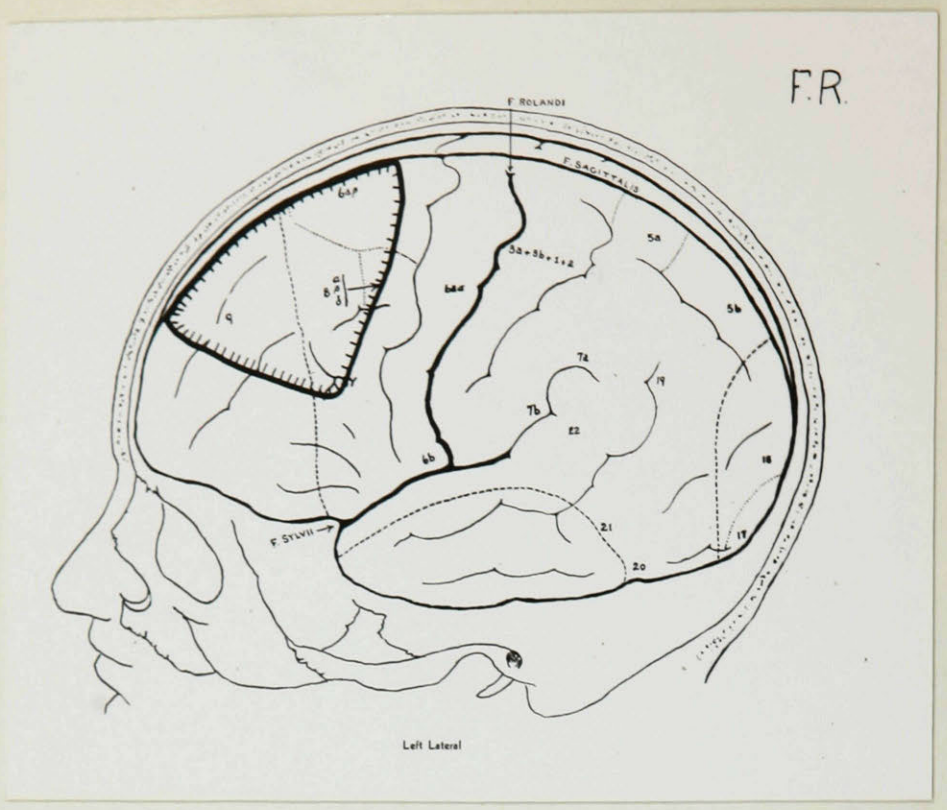

Fig. 50. Case F.R. Excision of an area of the left frontal lobe in a right-handed patient producing a transient postoperative aphasia.

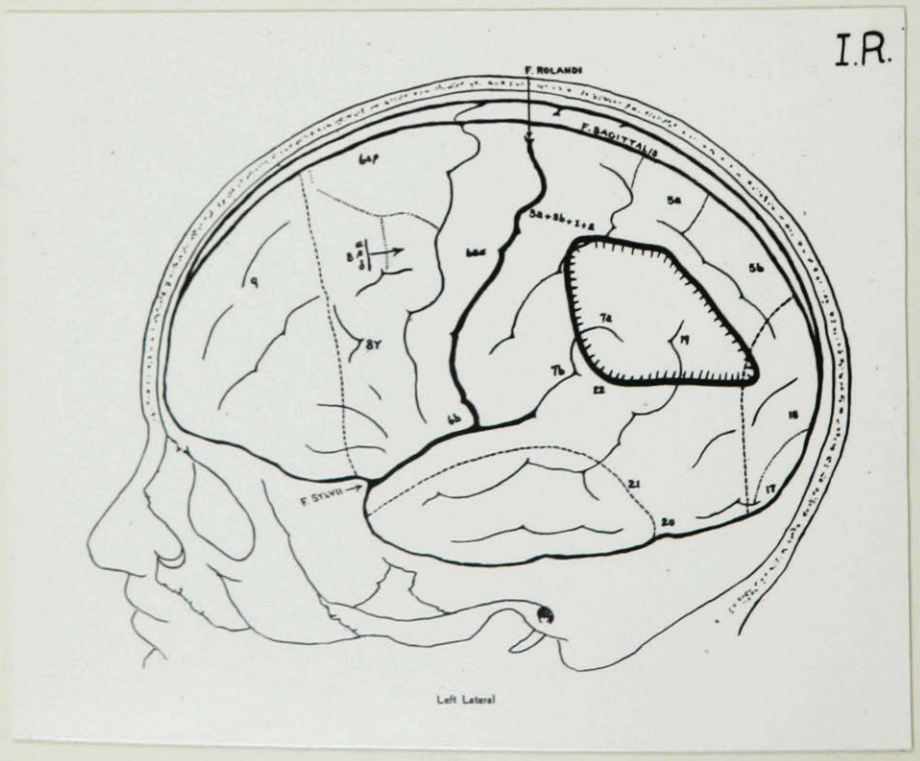

Fig. 51. Case I.R. Excision of area from the left parieto-occipital region, in a right-handed patient, in whom recovery of reading and writing was incomplete.

Case F.R., male, aged 20 was admitted on October 22nd, 1955 because of seizures. There was a history of a head injury at the age of nine years of uncertain severity. He was right-handed. A left osteoplastic craniotomy and excision of a large area from the frontal lobe was performed. Immediately following the operation he developed an expressive type of aphasia which lasted one hundred days. He had a very stormy postoperative period which probably accounted for the prolonged aphasia.

Case I.R., male, aged 36 was admitted on Nov. 9th, 1937 because of seizures of one and a half years' duration, which followed a laceration of the left parietal region. Following the accident he had complete loss of speech, and he still did not write, and read very little. He was right-handed. A left osteoplastic craniotomy was performed and a cicatrix was excised from the parieto-occipital region down to the ventricle. The postoperative period was complicated by a subdural haematoma. He developed a mixed type of aphasia on the second postoperative day, and in twenty-five days he was back to his preoperative state, but the writing and reading difficulty persisted. Eight years later he was able to read the newspaper but not as well as before the injury. He could sign his name, but wrote very little. When his son requested him to do so from overseas, he managed to write a "few lines". He was a farmer, had little occasion to write and made little effort to improve.

Case P.R., male, aged 19 was admitted on June 15th, 1939 because of seizures of three years' duration. He had had a traumatic head injury at the age of fourteen months. He was ambidextrous. A left osteoplastic craniotomy was performed and a cerebral cicatrix was excised from the left parieto-occipital region. He developed an aphasia on the first postoperative day which lasted eight days.

Case T.R., male, aged 22, was admitted on February 16th, 1944 because of seizures of four years' duration. At the age of nine years he had had a depressed fracture of the left frontal bone. He was right-handed. A left osteoplastic craniotomy was performed and a large damaged area in the frontal lobe was excised. There was no postoperative aphasia.

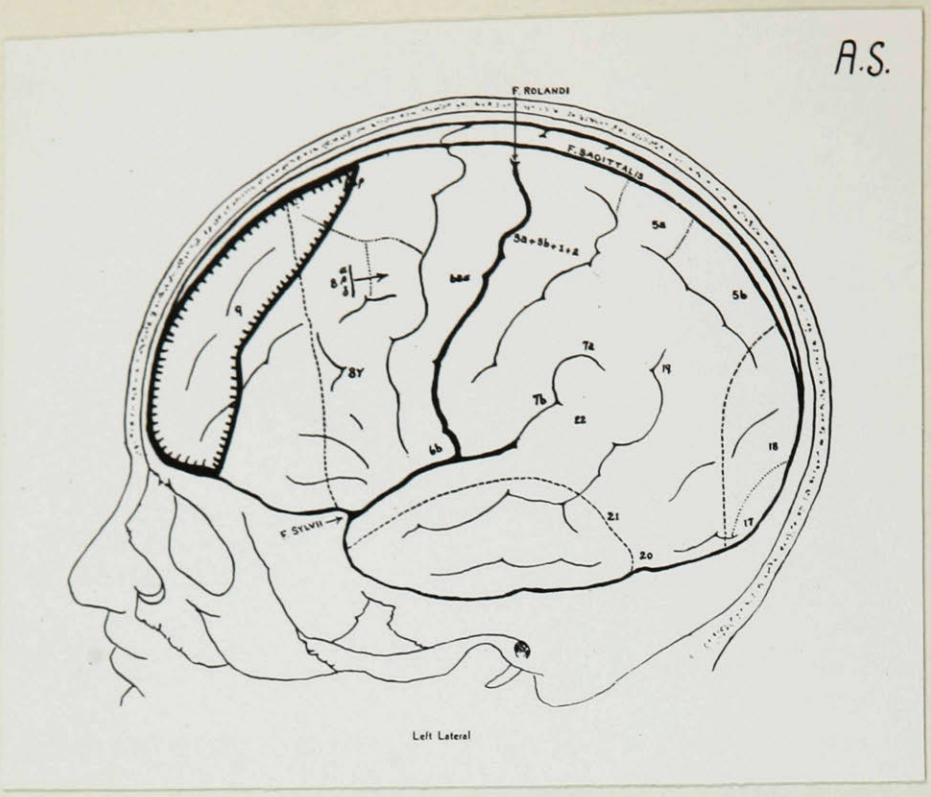

Fig. 54. Case A.S. Excision of area from left frontal lobe in a 20 year old patient who had been right-handed to the age of 13 years without producing aphasia.

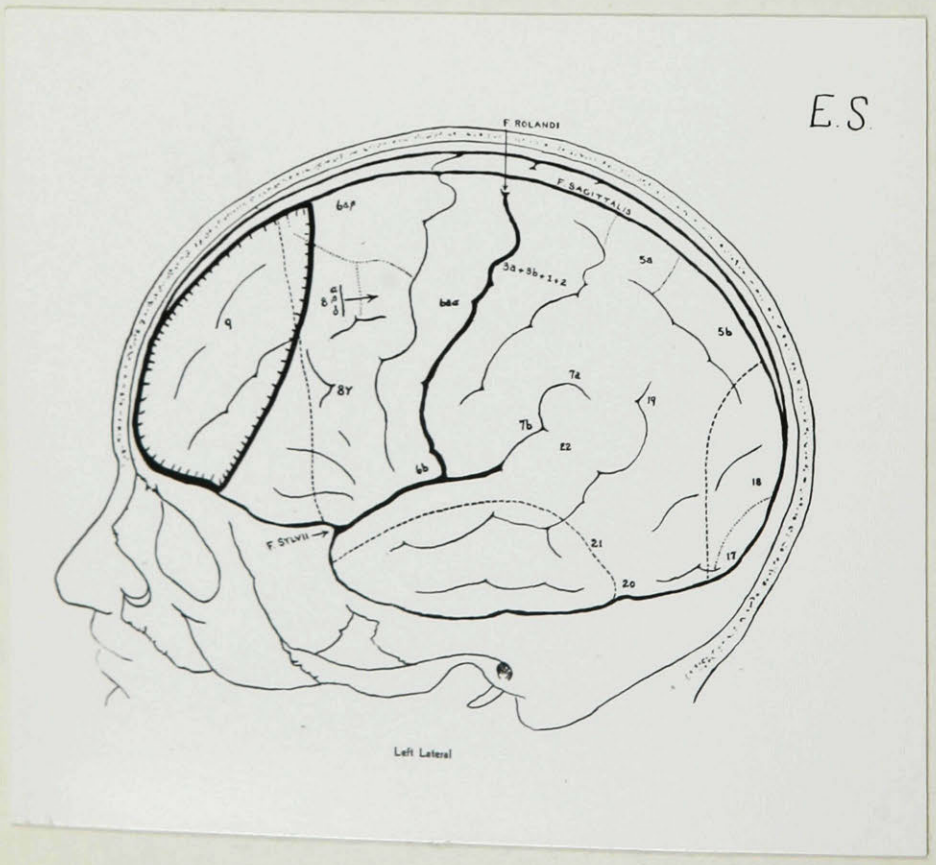

Fig.55. Case E.S. Area removed from the major frontal lobe followed by a transient, secondary aphasia.

Case A.S., male, aged 20 was admitted on Feb. 10th, 1942 because of seizures of seven years' duration. At the age of seven years he had had a severe head injury. He had been right-handed up until the accident. The brachial plexus was damaged and he had to change to the left hand which he did with difficulty. A left osteoplastic craniotomy was performed and a large area from the anterior pole of the frontal lobe was removed. There was no postoperative aphasia.

Case E.S., male, aged 23 was admitted on June 2nd, 1933 because of seizures of four months' duration. The cause or the time of onset of the injury was not known. The handedness was not recorded. A left osteoplastic craniotomy was performed and a cicatrix was removed from the left frontal lobe. A mixed type of aphasia developed on the first postoperative day and lasted thirty days.

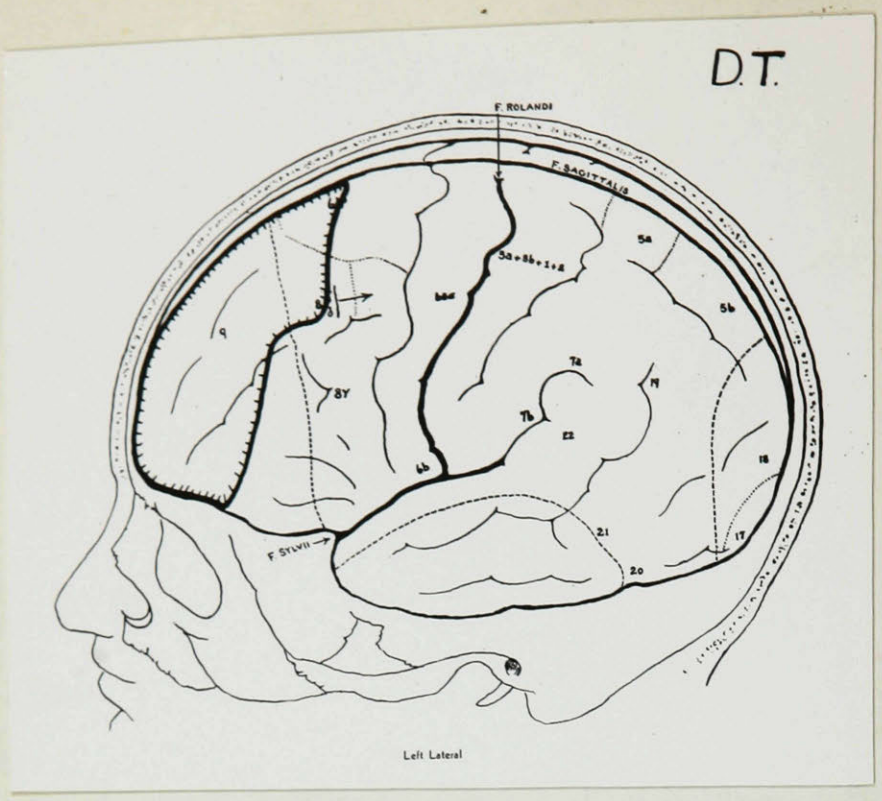

Fig. 56. Case D.T. Excision in two stages of large amount of left frontal lobe in a right-handed patient, who developed a secondary aphasia after the first operation.

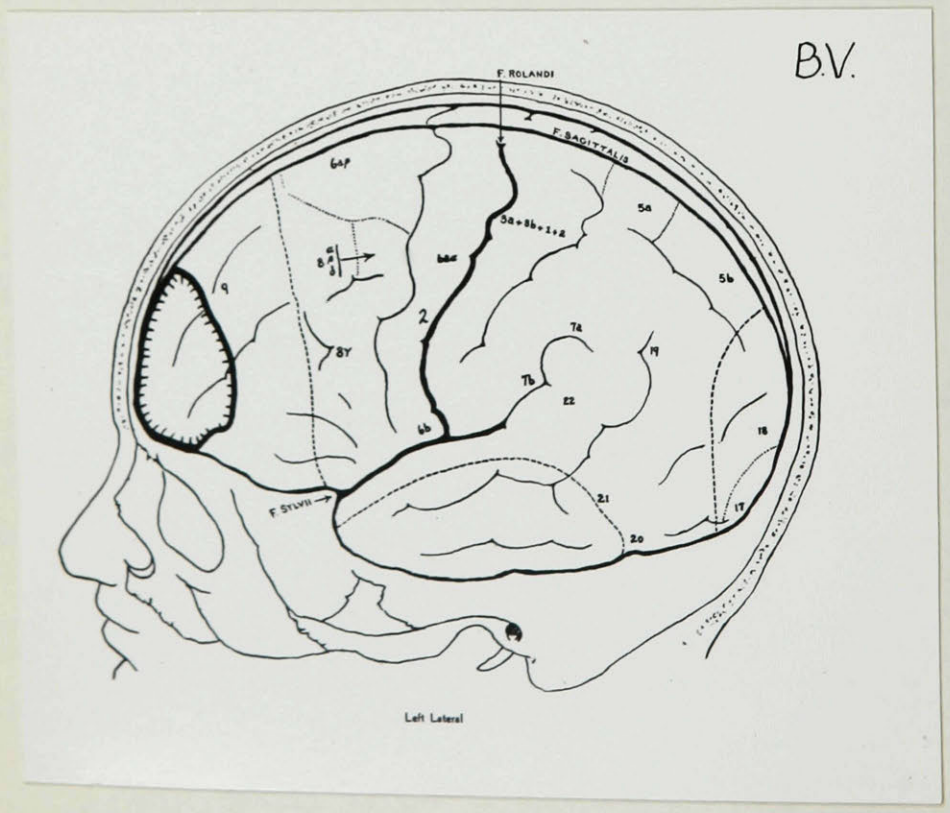

Fig. 57. Case B.V. Excision of area in left frontal lobe without producing aphasia in a right-handed patient.

Case D.T., male, aged 18 was admitted on two occasions (April 23rd, 1945 and January 14th, 1946) because of seizures which followed a head injury at the age of 13 years. He was right-handed. A left frontal osteoplastic craniotomy was performed and in two stages a large amount of the frontal lobe was excised. Following the first operation he developed an expressive type of aphasia on the first postoperative day. This lasted for eighteen days. There was no aphasia following the second procedure.

Case B.V., male, aged 18 was admitted on January 17th, 1944 because of seizures of six years' duration which followed a head injury at the age of eight years. He was right-handed. A left osteoplastic craniotomy was performed and an area was excised from the pole of the frontal lobe. There was no postoperative aphasia.

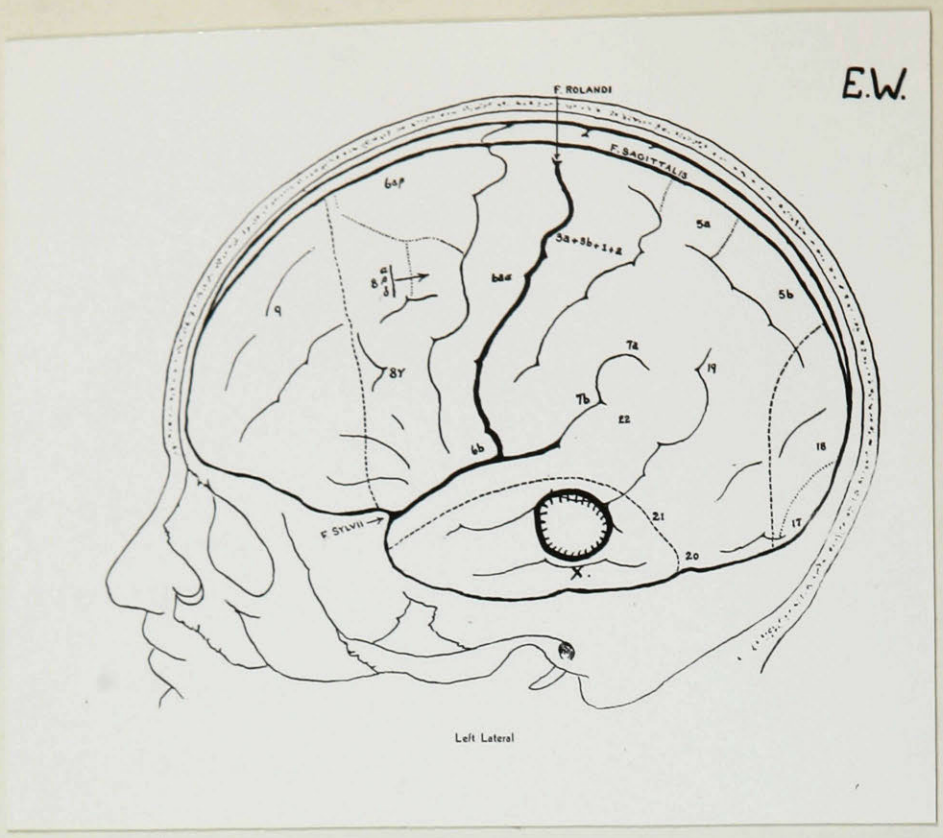

Fig. 58. Case E.W. Area excised from the left temporal lobe in a left-handed patient who had been trained to use the right, and who developed a secondary transient aphasia.

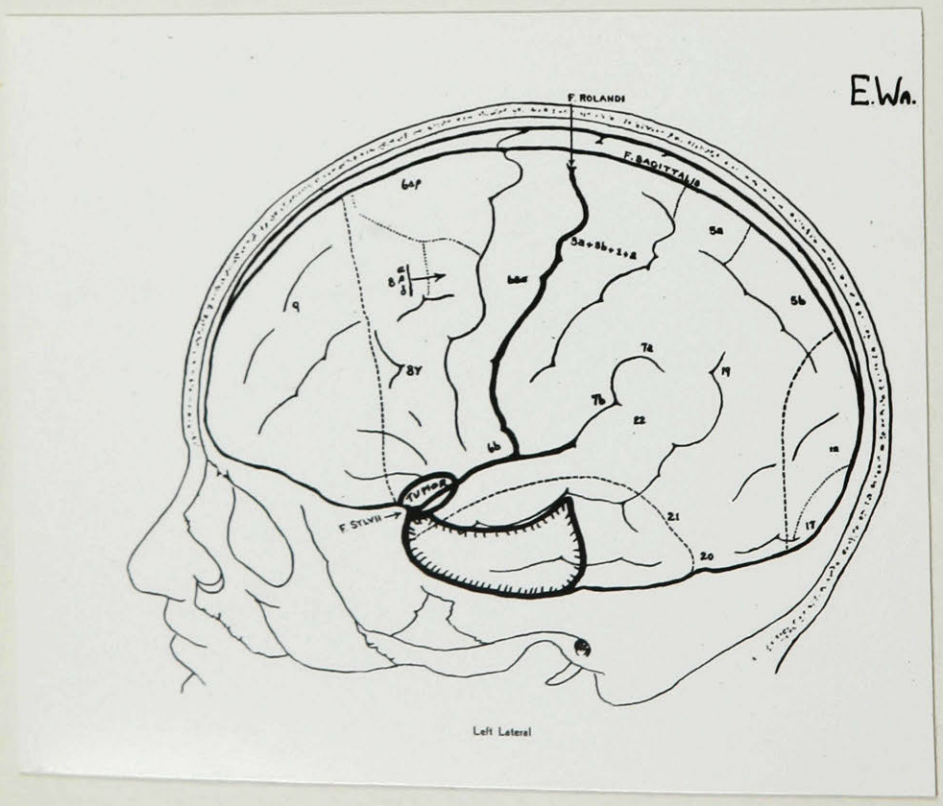

Fig. 59. Case E.Wa. A left-handed patient who had a partial removal of a tumor from the left fissure of Sylvius, and an excision of part of the temporal lobe without producing an aphasia.

Case E.W., female, aged 37 was admitted on January 19th, 1931 because of seizures of 16 years' duration. She had been left-handed at birth but was trained to use the right. She had twin daughters who were both left-handed. A left osteoplastic craniotomy was performed and an atrophic gyrus was removed from the temporal lobe. She developed a mixed type of aphasia on the second postoperative day which lasted three days.

Case E.Wa., male, aged 24 was admitted on August 29th, 1945 because of seizures of one years' duration. He wrote and ate with his left hand, and played games with his right. He was found to have a tumor in the fissure of Sylvius which was partially excised. On the first postoperative day he developed a mixed type of aphasia which lasted thirty days.

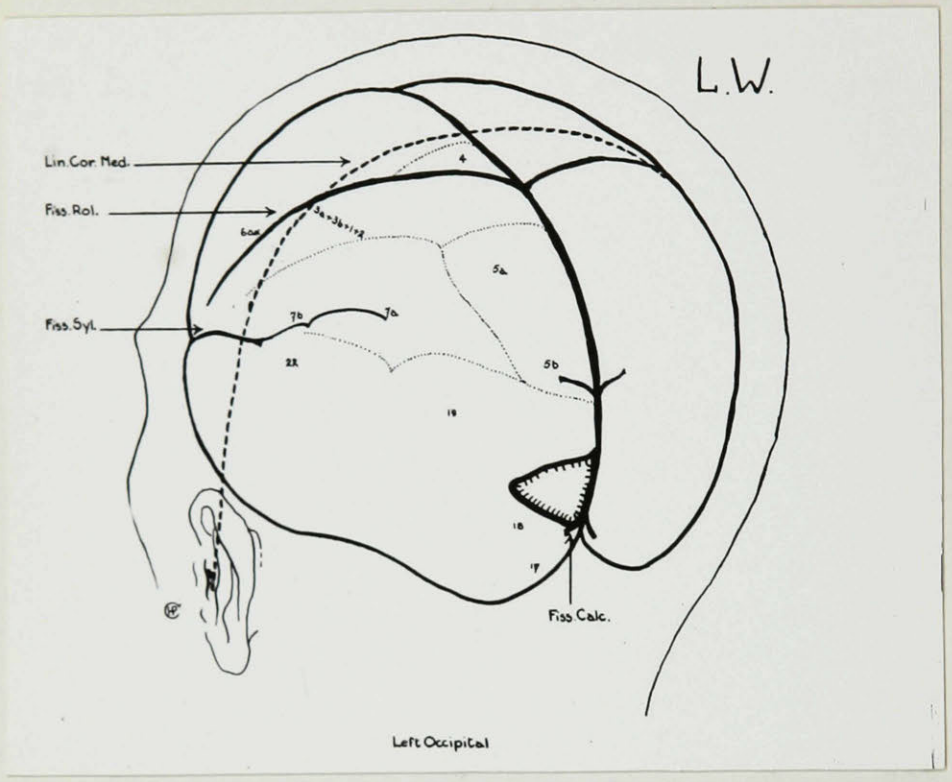

Fig. 60. Case L.W. Area excised from left occipital lobe in a right-handed patient without producing any aphasia.

Case L.W., male, aged 15 was admitted on July 29th, 1940 because of seizures of two years' duration. There was a history of a birth injury. He was right-handed. A left osteoplastic craniotomy was performed and a small collection of shrivelled gyri was excised from the pole of the occipital lobe. Following the operation he had almost a complete homonymous hemianopsia, but no aphasia.

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