AGNOSIA, APRAXIA, APHASIA

Their Value in Cerebral Localization

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PREFACE TO THE SECOND EDITION

THE first edition of this little monograph appeared at a time when interest in cerebral localization as applied to aphasia had lagged greatly. Especially in English-speaking countries the studies which were in progress were on the so-called psychologic basis, except for isolated contributions by neurologic surgeons.

It is, of course, difficult to arouse enthusiasm in a dying subject and I had no interest whatever in being a prophet or in any heroic salvation of localization in aphasia. I merely felt constrained to place on record what seemed to me a matter of great scientific interest. However, publishers must be practical if they are to survive and their balance sheets must show at least a little profit. They cannot proceed on an emotional basis and publish a work with monetary expense for materials and labor and with martyrdom as revenue. And inasmuch as exceedingly few men were interested in my subject in 1936, publishers consulted could see no prospects of anything but financial loss in publishing the monograph, nor could I. I decided to publish the work myself.

To my agreeable surprise the work was generously received in scientific circles. There was only one criticism, namely that I had not offered incontrovertible proof of all my statements. Certainly not. The proof would have required a monograph as large as Henschen's great work and that work had remained unread because of its bulk. Few neurologists would acquire an expensive monograph written, as Henschen's was, in four languages, whose application seemed extremely limited. Fewer would digest it.

Dr. Mandel Cohen, at the instigation of Dr. Stanley Cobb, came from Boston to study the subject. He spent months on the wards of Los Angeles General Hospital and he saw that our opportunity was unique because all cases of cerebral vascular lesions were admitted to the neurological, not the general medical, service. He became convinced that with few exceptions the statements in the first edition of this work were correct. He urged me, and many others followed suit, to publish a second edition and offer proof in some concise way. I was hesitant because I did not care to appear in the light of a champion defending a cause.

However, as the study of the subject continued it became possible to publish isolated crucial cases which proved one point after another and slowly I realized that the sort of monograph suggested by Dr. Cohen could be written and still remain small.

Many crucial cases became available through the work of the local neurological surgeons, especially Dr. Rupert B. Raney and men trained by him. World War II gradually removed from our midst one valuable man after another until at this writing (1944) our service is greatly handicapped. However, by drawing on the literature and including all available material I have now finished this second edition. I hope it will prove worth while.

I am grateful to all my friends who have encouraged me to produce this little work and to my publisher, Paul B. Hoeber, Inc., for undertaking the task.

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PREFACE TO THE FIRST EDITION

THE value of agnosia, apraxia, and aphasia in cerebral localization is a subject that is greatly in need of condensed description. With the present trend of thought on the subject distinctly in favor of the psychologic viewpoint, there is a feeling that localization in aphasia had its heyday from 1861 to 1906 and that from that time to this the medical world has had time to realize the errors of its ways and to abandon its naïve idea that aphasia could be of diagnostic value in cerebral localization.

However, the author believes that, because of gross errors and unwarranted conclusions in the old teaching, the entire doctrine has been prematurely discarded. The advancement of neurosurgery has enabled successful attacks on cerebral pathologic structures which before were impossible. Some of these achievements have given evidence confirmatory of the old doctrines. There are now several cases on record of successful removal of a complete cerebral hemisphere. These have shown that an individual can talk with utilization of only the right side of the brain. There are at least 2 cases on record of removal of the major temporal lobe. These have shown not only that a patient can understand with the minor side of the brain but also specifically what his handicaps are. These cases have also been confirmatory of the old doctrine.

The application of animal experiments to human physiology has been broader than the facts warranted. The early studies based on localized decortication of dogs, especially the removal of parts of the occipital cortices, which at the time seemed to show that localized function was a fallacy, must now be completely discarded in view of the proof that completely decorticated dogs can both hear and see (in a very crude way) and can even be conditioned to both sound and light. Focal cortical lesions in the human being do in many instances leave signs which can be recognized clinically, and diagnosis can be based on these signs.

The writer is interested in the philosophy of cerebral function on the one hand and in cerebral localization in neurologic diagnosis on the other. In favor of the latter he feels that a diagnostic method should not be permitted to fall into discard merely because it is difficult.

The method of approach used here is an outgrowth of the study of Pötzl's valuable monograph of 1928. The author showed that in many instances focal lesions of the occipital lobes could be responsible for certain symptoms (which, however, could easily be overlooked unless the case was studied in detail).

Since localization was already known for the sites of primary perception, and Pötzl indicated localization in the second stage of cerebral integration, the thought occurred to me that this idea might be extended. The present work presents this theme as far as it has been possible to carry it.

As the subject of aphasia is technical, and also tedious to the reader unless one is intensely interested in it, the work has been divided into three parts. In Part I a general account is given. In Part II methods of examination and case reports are gathered for him who wishes to study more intensely or examine the work critically. In Part III the terms and their synonyms, which are absolutely necessary because of the illogical and varied terminology in use, are listed alphabetically for ready reference.

The writer's major debt is to Dr. Samuel D. Ingham, his teacher in neurology. For years we have had conferences on every phase of the subject, and practically every point discussed in this thesis has been considered with Dr. Ingham. Besides, most of the cases here reported are from the wards of the Los Angeles County Hospital on his service.

The author's indebtedness to Dr. J. P. FitzGibbon, resident on the service, can be stated properly only by an acknowledgment of all that he did. Dr. FitzGibbon took an early interest in aphasia and demonstrated an insight into the problem such as is rarely met. He located the patients, made the first physical examinations, furnished the writer with abstracts of these, made basic examinations for aphasic disturbances, assisted in the detailed study of agnosia, apraxia, and aphasia, followed the patients as long as it was possible, and was directly responsible for the high percentage of autopsies obtained on those who died. It was through his efforts that nearly 200 cases were studied in the two years of his service. Since that time Dr. Elinor Ives has ably carried on the work.

The writer is also indebted to his many associates in the Los Angeles County Hospital for direct and indirect help. Dr. William Edler and his staff, Drs. Cullen W. Irish and Clarence W. Olsen of the College of Medical Evangelists kindly allowed freedom of their ward and access to all cases. Dr. Carl W. Rand, Chief of the Neurological Service, referred a considerable number of cases and rendered accessible all cases on his wards. Dr. Cyril B. Courville, Director of the Cajal Laboratory of Neuropathology, gave freely of his assistance in illustrations.

The writer assumes responsibility for all statements of opinion, all theories, and for all errors.

As the work is completed it is based on a clinical study of 240 cases with 25 autopsies, 13 surgical verifications, and 2 roentgenologic corroborations.

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AGNOSIA, APRAXIA, APHASIA

CHAPTER I

HISTORICAL INTRODUCTION

IT IS virtually impossible to give a complete historical account of disturbances now termed agnosias, apraxias, and aphasias, but an effort is made here to give a skeletal outline, to name the pioneers, and to mention briefly their contributions in this field, one of the most difficult in neurology and psychiatry.

At the beginning of the nineteenth century cerebral localization had not been discovered. The great masters of the time, particularly Flourens, taught that all parts of the brain served the same function, that one portion might take over the function of any other portion in case of injury or disease. He taught physiologic though not anatomic uniformity.

On the scene appeared Gall of Vienna (1805) with the postulate that persons with prominent eyes and gift of language owed these qualities to the fact that the portion of the brain mediating the faculty of speech was located immediately above the orbital plates, that in such persons this portion was larger than in others, that it pressed the eyes forward and downward and caused a bagginess of the lower eyelids. It is said that Gall arrived at this postulate by observing that certain fellow students who opposed and vanquished him in debate had prominent eyes and baggy lower eyelids. At any rate, an anatomic foundation for the teaching did not exist, and in spite of much propaganda, the alliance with Gall of Spurzheim, and subsequent wide popular enthusiasm for the doctrine, it fell into disrepute.

In the years which followed, a Frenchman named Marc Dax observed that persons afflicted with loss of ability to express themselves in language and who also suffered with hemiplegia were nearly always afflicted with a right and not a left hemiplegia. These observations were apparently not published at the time and nothing appeared in the literature until 1825 when Bouillaud of Paris, who had collected a great many specimens post mortem, announced that he believed the faculty of language to be situated in the frontal lobes (without regard to laterality).

Lordat, also a Frenchman, who in 1823 had written a book on speech in which he expressed the opinion that alalia (now called aphasia) was due to

asynergy of the muscles of speech, himself in 1828 became aphasic and later wrote an account of his experiences. While afflicted he "knew" words but could not use them properly; the speech of others came too fast for him to comprehend it; he used the wrong words, and in trying to correct himself, again spoke unintended words. He could not read what he himself had written. It is clear from this that Lordat recognized "sensory aphasia."

In 1836 Marc Dax wrote an article on aphasia presenting his views and cases gathered for thirty-six years in support of his conviction that the anatomic structure on which speech was based was located in the left side of the brain.

Bouillaud continued to gather instances and specimens in support of his contention. He was opposed by Cruveilhier and by Paul Broca. After some time a case appeared which was studied clinically by both Bouillaud and Broca, and it was agreed that when, as seemed probable, the patient should come to autopsy, the issue should be settled. Autopsy showed Bouillaud to be correct; whereupon Broca was convinced, withdrew his opposition, and became a supporter of the view of cerebral localization.

Time passed, and in 1861 Broca presented his now famous report of 2 cases to show that the faculty of language was located not only in the frontal lobes as Bouillaud contended but at the base of the third left frontal convolution. This statement was startling and was all the more remarkable in that in both of the cases presented the lesions were large, and it was only by deduction that Broca determined the lesion to have started in that area. He called the loss of function in his cases *aphémie*. His doctrine, then, was not only one of cerebral division of labor but one of extreme localization.

Trousseau, another student of the subject, was closely associated with Broca. He at first accepted the doctrine of Broca but departed from its narrow confines on the basis of several cases in which patients with aphasia were found at autopsy to have lesions in the temporal and parietal lobes of the left side. He also found fault with Broca's synthesized term aphemia to mean speechlessness. Trousseau learned from a well-educated Greek named Chrysaphis living in Paris that while *a* meant without and *phemia* meant speech, the term aphemia did not mean speechlessness but infamy. On the other hand, *aphasia* (which, according to Skwortzoff, had been used in 200 B.C.) meant speechlessness. Broca accepted Trousseau's correction, and since that time aphasia has withstood all attempts to change it in spite of the obvious impropriety of the term since the enormous expansion of its meaning has been accepted.

Wyllie in 1866 (eight years before Wernicke's epoch-making article) published the observation that "word ideas of associated motions which form the faculties of speech are supra-motory, while the situation of associated

sensations which form the faculty of word comprehension is supra-sensory.' Here was the foundation for the anatomicophysiologic concept of sensory aphasia.

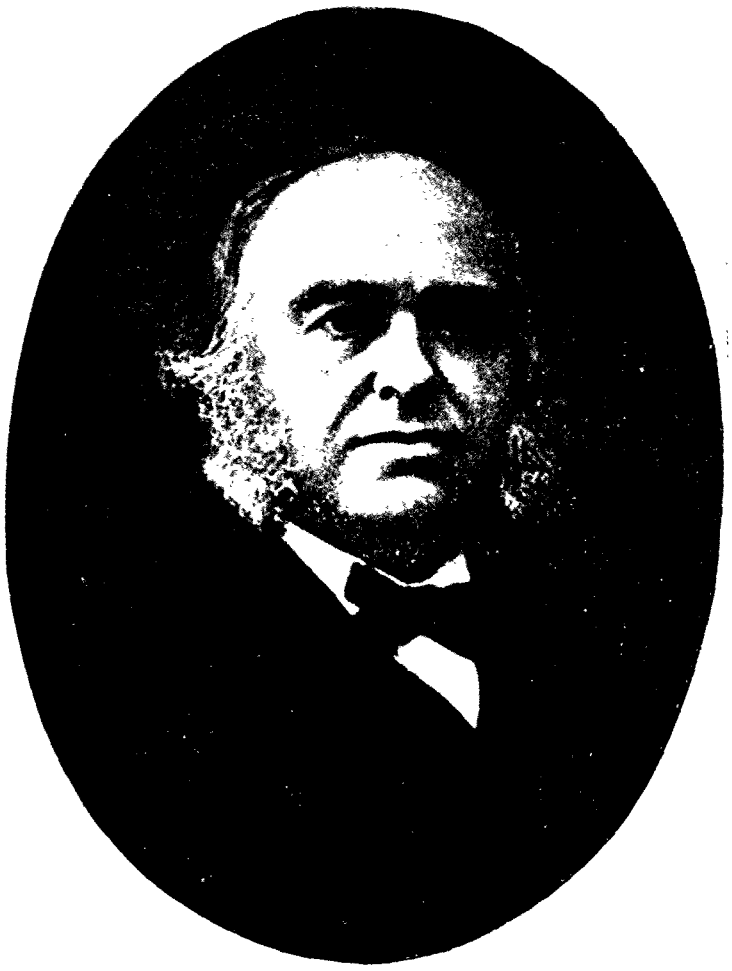

FIG. 1. Paul Broca. (From F. H. Garrison's *Introduction to the History of Medicine*, Philadelphia, W. B. Saunders Company.)

While these developments were going on in the subject of aphasia, another important step was taken by an Englishman named Moxon, although at the time it was not so regarded. He pointed out that the various centers for movement located on either side of the brain were not of equal importance in per-

formance of voluntary acts but that the centers of the left side guided those of the right, i.e., had a governing influence over them. Here was the foundation for the most important principle of modern apraxia, but no superstructure was as yet in sight. (Even the foundation was buried and had to be uncovered in 1900 by Liepmann.)

Following upon this work came that of Hughlings Jackson, largely ignored at the time only to be exhumed by Arnold Pick of the German University of Prague years later and now found to be so fundamental, important, and uniformly correct that it has been republished in more available form (1932). One of the most important of the great number of articles written by this famous Englishman was the one on left hemiplegia and *imperception*. Nothing similar had been reported (except for clinical descriptions of alexia by Schmidt in 1673), but this "imperception" of Jackson was clearly what we now all recognize as agnosia, and to be more specific, visual agnosia. Another of Jackson's contributions was the observation that patients with right hemiplegia and aphasia were often unable to protrude the tongue on request (apraxia of the mouth).

The term *agraphia* was introduced in 1867 by William Ogle to characterize inability to write. The groundwork was now well laid for practically all forms of aphasia except those pertaining to music, which were to come much later.

While sensory aphasia was described by Wyllie, this author did not present cases. The scientific presentation with anatomic material was left for Wernicke of Breslau (1874). One is much impressed with his article, not only because of its originality but because of the youth of the author (he was only 26 years of age), his modesty and self effacement, and his genius. He persistently refused credit for the work, explaining repeatedly that he was only enlarging upon and expounding the teachings of Meynert. One therefore loves the man and his work in spite of the fact that many principles enunciated by him have been disproved. His monumental work of establishing sensory aphasia on an anatomic basis remains. Almost immediately after the publication of the article, Kussmaul suggested that Wernicke's term sensory aphasia be changed to word deafness. It is now known that Wernicke's aphasia consists of auditory verbal agnosia, visual verbal agnosia, and agraphia—a syndrome of three elements.

While these clinical studies were in progress, investigators working in the experimental field and possessed of great ingenuity and keen sense of observation brought forth knowledge in this work concerning psychic blindness. The work is the more admirable because it was done on dogs. It is difficult enough to identify visual agnosia in the human subject who can talk. The

work referred to was that of Munk and of Hitzig in Germany and Luciani and Seppilli in Italy. The first two did their work on dogs alone while the last two applied the results to the human subject and made clinical correlations. Psychic blindness (transient) resulted from extirpation of portions of the occipital lobes.

Charcot in the meantime had noted psychic blindness in the human subject, and his report was followed by cases by Wilbrand in Germany. Charcot localized the lesion causing alexia in the left angular gyrus.

Exner in 1881 postulated (on insufficient evidence it is true but nevertheless his idea was correct) a writing center at the foot of the second frontal convolution on the left side. With this Charcot in France and Bastian in England agreed while others denied such a center.

While these research workers were discovering "centers" and studying cases, synthetic thinkers were correlating these discoveries. Baginsky presented the first diagram in 1871 (Fig. 4). As soon as Wernicke's work had appeared (1874), Lichtheim began to make diagrams of Wernicke's centers. These schemes Wernicke accepted, and there seems to have been hardly a dissenting note between the two men. Bastian also presented a scheme which differed from that of Wernicke and Lichtheim (motor, sensory, and concept centers) in not having a concept center but, on the other hand, having four centers (glossokinesthetic, cheirokinesthetic, visual word, and auditory word centers). Lichtheim's scheme provided for many undiscovered types of aphasia. Some of these have since been discovered, others not.

In this age of diagram makers Hughlings Jackson continued to add valuable data (nearly all of which was ignored by other students). For example, he brought forth the idea that paraphasia, which Wernicke had explained on the basis of impaired connections between the auditory and motor speech centers, was due to the function being taken over by the right side of the brain. He also pointed out the difference between propositional and emotional speech and showed that emotional speech was probably mediated through the right side of the brain. These ideas have been hard for us to accept, but they are probably correct. They have been accepted by so ardent a localizationist as Henschen and a psychologist as Goldstein. Next Charcot stressed the importance of individualization in language showing that symptoms of lesions in certain cases differed from symptoms of identical lesions in others. Some persons are visual-minded, others auditory-minded, and a lesion in the guiding center causes far more aphasia than a lesion in a minor center.

The term apraxia is commonly ascribed to Gogol, but Bonvicini points out that it was borrowed by him from Steinthal who used it in his work *Abriss der Sprachwissenschaft*. It was thought that the wrong use of objects was due

to faulty recognition of the object. This misconception persisted to the time of Liepmann in 1900 who showed in his famous patient (the Regierungsrat) who had apraxia of one side only that in the apractic, while the function was

FIG. 2. J. Hughlings Jackson. (From *Selected Writings of J. Hughlings Jackson*, edited by Dr. James Taylor, London, Hodder & Stoughten, 1932.)

comprehended, the performance of the intended act was still impossible. Apraxia was therefore a motor phenomenon. Later it was also discovered that there were various forms of apraxia depending upon whether or not the idea itself was clear, whether it was clearly conveyed to the motor area,

or whether the fineness of the movement as a purely motor phenomenon was affected. Pick (1905) showed that apraxia might be the result of a purely functional condition, as toxemia or a postepileptic state. Later Sittig (1931) confirmed some work of Mingazzini and demonstrated that various localizations in the corpus callosum by destruction may give varieties of apraxia so that these may be of localizing value. Heilbronner (1906) pointed out that unilateral agraphia may be due to apraxia and is not necessarily aphasic in origin.

As has been stated, it was thought for a long time that apraxia was due to lack of recognition. This was what Jackson had called imperception and Meynert and Finkelnburg had called asymbolia. In 1891 Freud proposed the term agnosia for this sort of disturbance, and it has come into general use. The acceptance of the term was a landmark, because psychic blindness, psychic deafness, and astereognosis were all then recognized to be of the same functional class. Pötzl, Wolpert, and Liepmann all suggested extensions of the term until (it seems to the writer) it has lost much of its usefulness. Pötzl's suggestion of including lack of recognition of pictures is good, but Wolpert's suggestion to include lack of recognition of sequence (simultanagnosia) and Liepmann's to include ideational agnosia seem to me to go too far.

To return to aphasia, up to about the year 1890 the trend of thought was all in the direction of strict localization. Then Freud (1891) in his monograph suggested that there was actually no such thing as a center (Fig. 5). He said all aphasia was conduction aphasia. Dejerine declared against the autonomy of the so-called centers, and M. Allen Starr (1889) stated that he had failed to find any case to substantiate that a focal lesion in Broca's convolution could cause Broca's aphasia. Maudsley had already (1868) declared that there were no centers but that certain areas had been adopted by speech. In 1906 Marie threw a figurative bomb into the field by declaring that the third left frontal convolution played no special role in the function of language. He stated that there was only one type of aphasia—that of Wernicke, that Broca's aphasia was Wernicke's aphasia plus anarthria. He stressed what many others had pointed out before him, that in all aphasics there was some trouble in comprehension of speech. Now, while it is admitted that there is always difficulty in comprehension of speech, it is not conceded that that fact proves lesions of all portions of the brain to be equally potent to produce incomprehension. Anyone who will take the trouble to investigate Marie's own description of his interpretation will have no difficulty in understanding the method by which he arrived at his peculiar conclusion. He simply redefined aphasia. He said, "D'après ce que nous avons dit plus haut des caractères de l'aphasie de Broca et de l'aphasie de Wernicke, on voit que ce qui, au point de vue

d'une saine classification nosologique, doit constituer en somme *l'aphasie vraie*, ce n'est pas le fait de parler mal ou de ne pas parler du tout; ce qui constitue l'aphasie, c'est le fait de comprendre insuffisamment la parole, de

FIG. 3. Jean Martin Charcot. (From *Minnesota Medicine*, January, 1926.)

presenter cette déchéance intellectuelle particulière sur laquelle nous avons insisté dans la première partie de cet article et, enfin, fait très important, d'avoir perdue la faculté de lire et d'écrire." It is seen immediately that Marie defined

aphasia as sensory aphasia plus agraphia. He also (in the same article) redefined anarthria as equivalent to the classical subcortical motor aphasia. He overlooked the fact that an anarthric cannot properly use his muscles for any

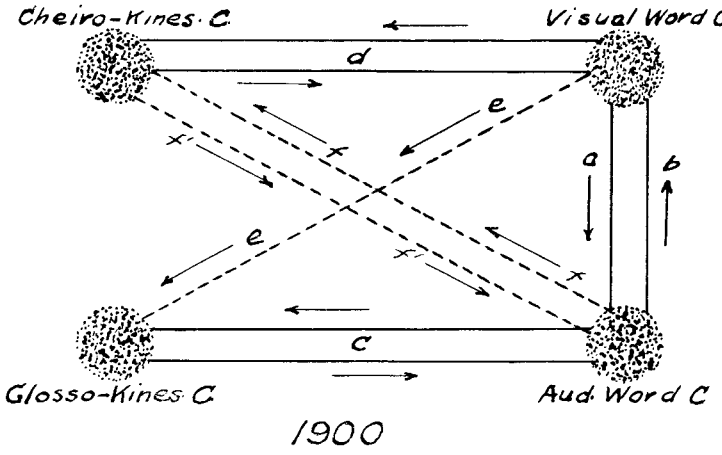

FIG. 4. Bastian's diagram with four centers.

purpose. An aphasic may use them for any purpose but speech. His statements therefore entirely lose their force. He has merely changed definitions and this makes his statement sound startling. The work had, however, the value of reopening the whole subject, which really needed reconsideration.

When apraxia had been thoroughly established and was understood, it was only natural that the theory that aphasia on the sensory side was an agnosia and on the motor side an apraxia should be promoted. This was done by the French writers, especially Laignel-Lavastine, Foix, and Claude. This question is purely a matter of interpretation. The writer is unable to see any difference between motor aphasia and apraxia limited to language. On the other hand, agnosia does not cover the subject of sensory aphasia. The type described by Head as semantic aphasia is a striking example of the difference between aphasia and agnosia. Liepmann's ideational agnosia is an example of how the two types of disturbance nearly merge.

The history as presented here has been considered largely from the standpoint of anatomy and physiology. It is, however, possible to view the matter largely or even entirely from the point of view of psychology and thus largely to ignore these basic sciences. The foremost promoters of the psychologic side of the subject have been Hughlings Jackson and Head in England, Pick in Czechoslovakia, Goldstein in Germany, and Weisenburg and McBride in the United States. It is not implied that these men were not anatomists; they certainly were, but they stressed the psychologic side of the subject. That side

is, in truth, an important matter, as there are many phases which one is even yet unable to place on an anatomic footing. Perseveration (a purely psychologic term) is always to be considered in the analysis of a case. The past life

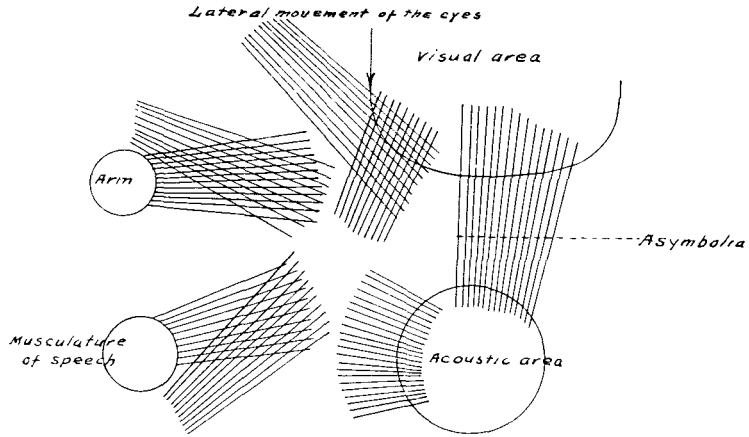

FIG. 5. Freud's scheme of 1891. The anatomic centers are indicated by circles, the association pathways by lines. Freud's idea was that all aphasia was conduction aphasia.

of the patient is of great importance in determining the type of reaction obtained. The same is true of concentration of normal attention and matters affecting it, such as conversation about the bed during the examination and depression or elation of the patient. Charcot's contribution in emphasizing the great part played by individualization in cerebral function and in aphasia must never be lost from sight. When the writer decided to approach this problem from the standpoint of cerebral localization, it was not with the idea of ignoring psychology but, while taking it into full account, to stress anatomy and physiology.

In the last twenty years there have been two outstanding workers in the field of aphasia who have taken diametrically opposite views—Head and Henschen. A third, S. A. Kinnier Wilson, considered all sides of the question and was, in the opinion of the writer, one of the sanest of authors writing on the subject.

Head simply discarded all that had previously been done and started with a clean slate. He used none of the old terms, worked largely with living material, made exhaustive examinations, and based his study as far as the limits of the material permitted on anatomy and physiology, but was

obviously greatly limited in this regard. He assumed, for example, that the lesion in the brain corresponded mainly to that of a depressed fracture, where such was present, seen in roentgenograms and on plaster casts. As the German

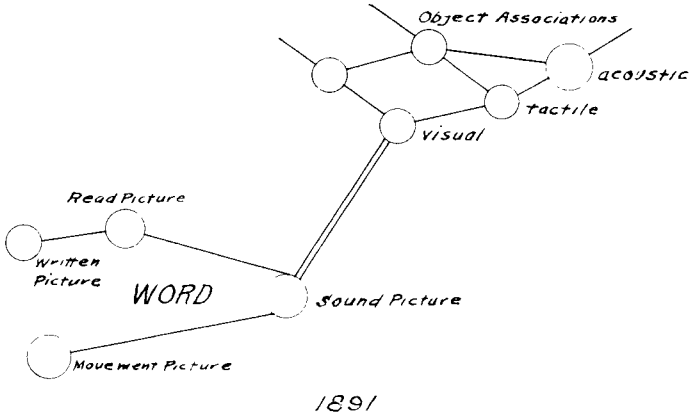

FIG. 6. Freud's diagram of "the word." This illustrates well its complex nature.

workers have pointed out, no matter how carefully the clinical work may have been done, this method could never solve the problem of aphasia. Head did, however, call attention to one important phase of the subject, that there are hierarchies in the physiologic sense in cerebral function, that it is possible to determine these and to study aphasia on the higher levels and thus find defects when the usual studies would lead to the conclusion that the patient was not suffering from aphasia. His exposition of semantic aphasia makes his work well worth while.

Henschen, on the other hand, discarded all psychologic factors and based his studies in localization entirely on autopsy material. Further, he used all the autopsy material in the literature of the world. While this had the disadvantage of necessitating the acceptance of the clinical studies of the men reporting the cases, there was obviously far less source of error in this than in confining his work to his own 60 autopsies on aphasics (a large number for any one man). He thus analyzed about 1,500 cases and presented a work unparalleled in this field. His monograph consisted of three large volumes of presentation of abstracts and excellent analysis. In this was included a study of amusia and acalculia not previously attempted. Many premature conclusions previously quite firmly fixed in the minds of students of aphasia were dispelled (for example, that the inner language was dependent upon the intactness of the heard word). Anatomic localization in the brain was carried to the *n*th degree in relation to speech.

The monograph of Wilson (1926) takes all three approaches into account, recognizes the functional hierarchies, and recommends study based upon these facts. Wilson's great contribution is his revival of Jackson's ideas and in-

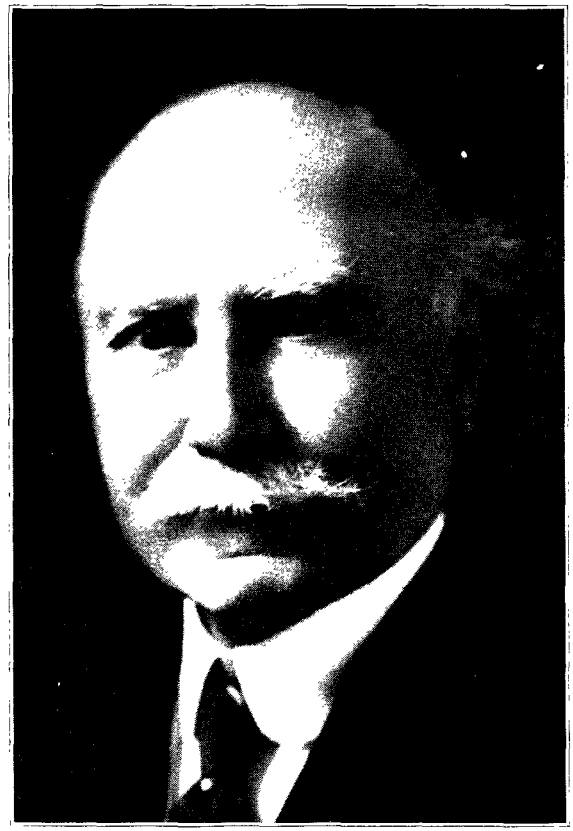

FIG. 7. M. Allen Starr. Dr. Starr and Dr. Charles K. Mills became the most authoritative Americans on the subject of aphasia.

sistence upon the separation of the anatomic, physiologic, and psychologic classifications of terms: that all three types of approach are absolutely necessary for a complete study, but that terms in the one sphere cannot be applied to structures or concepts in the other if one wishes to avoid error. It is the confusion of the spheres of thought and avenues of approach in the past which has led to the drawing of a psychologic concept center, an anatomic foot of the third frontal convolution, and a physiologic writing center all on the same diagram of a cerebral cortex, and the drawing of lines from one to the other as though association fibers connected them and impulses passed along these

lines. If workers in this field will give full consideration to Wilson's recommendations, years of valuable time will be saved in the study of aphasia.

In the course of development of the subjects of agnosia, apraxia, and aphasia

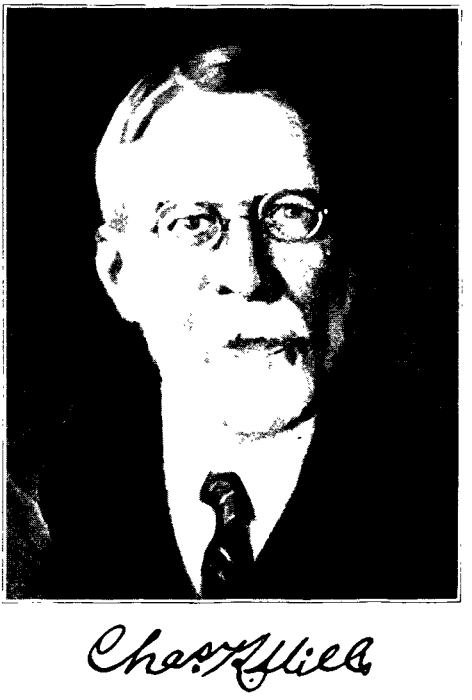

FIG. 8. Charles K. Mills. Dr. Mills was the outstanding authority in the United States in the field of cerebral localization in aphasia.

there have appeared certain literary contributions on the subject whose authors deserve special mention whether or not they can be classed as pioneers. Among these are the following: (1) Ballet, (2) Banti, (3) Bastian, (4) Bateman, (5) Bianchi, (6) Bonvicini, (7) Broca, (8) Brun, (9) Charcot, (10) Claparède, (11) Dejerine, (12) Exner, (13) Feuchtwanger, (14) Finkelnburg, (15) Freud, (16) Freund, (17) Froment, (18) Goldstein, (19) Grashey, (20) Grasset, (21) Head, (22) Heilbronner, (23) Henschen, (24) Hinshelwood, (25) Hitzig, (26) Jackson, (27) Kussmaul, (28) Lichtheim, (29) Liepmann, (30) Lordat, (31) Luciani and Seppilli, (32) Niessl von Mayendorf, (33) Mills, (34) Mingazzini, (35) von Monakow, (36) Moutier, (37) Osnato, (38) Pick, (39) Pitres, (40) Pötzl, (41) Quensel, (42) Sachs, (43) Saint Paul, (44) Sittig, (45) Skwortzoff, (46) Starr, (47) Storch, (48) Thiele, (49) Trousseau, (50) Weisenburg

and McBride, (51) Wernicke, (52) Wilbrand, (53) Wilson, and (54) Wyllie.

Of these a certain few should have additional mention because of special features about their work. Bastian gives an excellent philosophical account, presents many original ideas, and considers all phases of the subject in a good-sized monograph. Bonvicini presents a relatively recent article (1929) and gives in addition to the 270 pages of excellent discussion also 30 pages of references. Henschen gives us three volumes in which there are abstracted about 1,500 cases, each in the original language of the author reporting except Russian and Scandinavian which are reported in German. This covers all cases in the literature of the world with autopsy up to 1922. Jackson's work is now available in his *Selected Writings* and contains unsurpassed original thoughts written from 1873 to 1900. His philosophical discussions are worth the while of anyone interested in aphasia. Kussmaul wrote an article of 300 pages, complete at the time in every respect. It is fifty years old but strangely "modern" yet. He did much to standardize nomenclature.

Clinical, pathologic, and surgical contributions of the last ten years have enormously enhanced our knowledge of aphasia. The ideal of an anatomico-physiologic nomenclature now seems to have been attained.

C H A P T E R I I

AUTHOR'S CONCEPT OF EUGNOSIA, EUPRAXIA, AND EUPHASIA

Their Pathologic Counterparts, Agnosia, Apraxia, and Aphasia

MEMORY is the capacity of the individual to keep sensory impressions and their associations catalogued in the brain and have them ready for future reference. The loss of this capacity is amnesia. Memory or amnesia may be general or specific, general amnesia resulting from psychological causes or diffuse cerebral disease and specific amnesia resulting from psychologic causes or focal cerebral lesions. Agnosias and aphasias are all types of specific amnesias due to focal cerebral lesions. Some apraxias are due to diffuse, others to focal lesions. When superficially similar clinical manifestations result from functional causes, they are not properly classified under those headings, a different terminology being used. Thus complete inability to emit sounds may be a type of motor aphasia or an hysterical mutism; inability to recognize what one sees may be visual agnosia or hysterical amblyopia; inability to recall names of objects may be amnesic aphasia or simple forgetfulness. In all such cases one must exclude a functional state by satellite symptoms which accompany such states or must show by collateral symptoms that aphasia is the basis.

From this discussion it is clear that when general cerebral function or general memory is affected, one frequently is prevented from showing that specific agnostic, apractic, or aphasic disturbances are present. It is unsafe to ascribe apparent specific defects to focal cause when general cerebral dysfunction is clearly present.

Symbolization is not confined to the human race but is clearly seen in animals. A cow will scratch its companion where she herself wishes to be scratched, and her companion understands and complies with the request. A dog will go into ecstasy when his mistress puts on a certain garment which the dog has learned means a trip to the market or go into a depressed state if she takes a different one which means a long journey leaving the dog at home. All conditioned reflexes in animals or in man depend on symbolization.

However, in the human race symbolization is elaborated to a high degree of

efficiency as a labor-saving or economizing device for keeping track of objects and persons which cannot themselves be catalogued. Language is a type of symbolization, though not all symbolization is language. Language is symbolization which is used to exchange thoughts with one's peers.

The human brain has developed in its cortex (probably *with* its cortex) a highly complex system of language symbolization, the anatomical structures of which are neurons with cell bodies situated in the cerebral cortex, the complex function of which when comprehended by the student is called physiology, and when conceived functionally but not anatomically is called psychology. Various portions of the cerebral cortex are variously constructed and have different functions. The different areas have been elaborately studied and mapped by Brodmann, Campbell, Elliot Smith, the Vogts, Economo and Koskinas, and physiologically by Foerster. Certain of these areas of the cortex may be rendered functionless by natural experiment, and the resultant disturbance of function may be recognized as the direct result of such localized destruction. By correlation of certain functional defects with certain specific anatomic defects, signs of agnosia, apraxia, and aphasia may be valuable in cerebral localization. Thus it comes about that a patient who has been able to read and is rendered incapable of reading while still relatively unimpaired in other functions of symbolization is known to have a lesion in the major (usually left) angular gyrus.

One must be careful to say "relatively" unimpaired, because there is always some degree of impairment of general cerebral symbolic and other functions when the defects make possible a diagnosis of agnosia or aphasia. This is obvious, as the deleted portion of the brain is part of the area used in general symbolic mentation, and the structure as a whole does not function so well after the deletion of a part as it did before that deletion.

Language is a highly complex cerebral function requiring for its use one side of the brain behind area 6 of Brodmann. This may readily be realized when one considers the multiplicity of associations one has with each idea and the necessary complexity of anatomy and physiology to render this possible. The fact that the portion of the brain anterior to area 6 is not essential to language is shown by cases of bilateral prefrontal lobectomy.

Areas of cortex having apparently certain specific functions have been called *centers*. There is frequent reference to centers in the litrature in the anatomic sense, the physiologic sense, and in the psychologic sense. What we are concerned with here is the definition of a center in the clinicopathologic (anatomicophysiologic) sense. From this viewpoint, a center is a cortical area the functional removal of which causes a definite deficiency syndrome which can be recognized clinically. Such a center is not a functionally independent area

devoted to one certain function exclusively as it is often conceived of. It is an area essential to normal performance of a certain function. It may be an area where a certain group of impulses pass or where synapses occur in the ordinary process of certain functions.

The concept of an impulse travelling once from one area to another and thus giving rise to adequate cerebral function is too naïve, and such an impulse can in no wise represent an actual state. In the performance of even the simplest language function impulses travel in many directions from a given point or from several areas toward a given point to many different centers and back over the same or different gross routes many times. Further, the route normally traversed by impulses in the function of language, while the best in the interest of efficiency, is not compulsory; a different route may be taken when a region which would normally be traversed is out of function. The functional capacity will then be reduced but not destroyed.

To illustrate how the routing of impulses may be changed, a case of visual agnosia for objects may be considered. The patient fails to recognize objects seen and hence does not get the concept of the object. After he touches it or hears it (if it is audible, as a watch by its tick), he gets the concept and then revisualizes the object in his mind. The mental image is now practically complete, but the impulses reach the area for revisualization not by starting from the calcarine area as normally would be the case but by starting with hearing or touch. Or, consider the case of a man with the so-called transcortical motor aphasia. Such a man is unable to say what he wishes, but if someone says "Say 'New York'," he immediately says it. Instead of the impulse arising in his concept area, it arises in the acoustic area, but it reaches the same end point—the site necessary for vocal speech.

A person often has concepts without corresponding symbols. Illiterates have relatively few symbols corresponding to their innumerable concepts. The concept center or area can therefore not be the same as the naming center. In the study of any foreign language in which the fundamental symbols differ from one's own, one learns to recognize the symbols while still unable to name them. During this stage the characters are to the students not symbols but objects. For example, he who does not know Hebrew may soon learn to select from a Hebrew text all the alephs (א's) without knowing anything about their symbolic significance. Or after a cerebral lesion one may be able to "read off" written or printed words without having any idea of their significance. This in aphasia is an important distinction: the loss of recognition of the thing as an object is agnosia; the loss of its symbolic significance is aphasia.

From the historical summary given above it is evident that even in recent

times radical differences of opinion have been expressed by prominent investigators. It is in no wise possible to state dogmatically that this or that is the "modern" or "present" consensus of opinion or concept. Each writer has his own concept, and I shall give mine, the reasons for it, and the points in which it differs from that of other writers.

As stated before, it is impossible for any single approach to the problem of aphasia to be successful. All three methods of approach are simultaneously to be considered: psychologic, physiologic, and anatomic. I shall first outline the most important psychologic factors to be considered in *any* case.

PSYCHOLOGIC FACTORS

There is hardly an element of agnosia, apraxia, or aphasia established as resulting from organic lesions that has not also been noted in purely functional states. Fright may cause a temporary mutism just as thorough as that seen in motor aphasia. The inability to understand spoken words and the inability to perform purposeful acts as seen in postepileptic states may be as complete as acoustic verbal agnosia or apraxia due to organic lesions. The psychic blindness and deafness seen in hysteria and the inability to recall names of familiar persons so common in normal individuals may be as great in degree as similar symptoms in organic disease. If such symptoms can result from purely functional states, one cannot deny that functional states play an important role when organic lesions are present.

Psychologic states have a large part in production of aphasic manifestations. For instance, in general disease of the brain which affects intellectual function and consequently speech, the patient will usually lose first the ability which he has most recently acquired. There are perhaps modifications of this in the sense that capacity often practiced may be more automatic in spite of being more recently acquired than one never practiced though early acquired. However, as a general rule writing (acquired with greatest difficulty) is first lost in general disease of the brain. This statement is generally accepted but may be illustrated by two observations.

In the first instance (case 90) the patient had a severe toxemia in the form of uremia. When she manifested difficulty in speech, the physician in charge suggested that her "aphasia" be studied. There was an isolated paragraphia. With much interest the subsequent autopsy was awaited. Because of an intercurrent cardiac lesion, it came the next day. There was no lesion anywhere in the brain. I studied with care the foot of the second frontal convolution microscopically but found no lesion.

In the second instance (case 88, referred by Dr. Nicholson) the patient was seen four months before death, at which time she had paraphasia and para-

graphia. She had at the time severely choked optic disks of 4 diopters on both sides and was vomiting badly. As she had had a carcinoma removed from the breast several years before, it was concluded that she had a metastasis to the brain. Because of the euphoric mood, the paragraphia, and the absence of localizing symptoms, it was thought that she had the major lesion in the left frontal lobe. At autopsy, four months later, there were found four lesions. One was in the left frontal lobe but was only 2 mm. in diameter. The second was 5 mm. in diameter and was in the left thalamus. The third was like the first and was in the right occipital lobe. The major lesion was 2 cm. in diameter and was located in the right cerebellum. This last was undoubtedly the oldest lesion in the brain and the one which was causing the symptoms when she was first seen. In other words, the right cerebellar lesion was mistaken for left frontal. The tiny lesion seen in the left frontal at autopsy could not have been responsible for the severe symptoms seen four months before. The marked hydrocephalus seen at autopsy and the toxemia were undoubtedly responsible for the paragraphia.

It seems evident, also, from material available that semantic aphasia, acalculia, paraphasia, and ideational apraxia may be caused by nonspecifically localized lesions of the brain or even by diffuse pathologic physiology. I have an interesting case on record in which a careful study of aphasia was made and in which the patient came to autopsy. This patient (case 64) had semantic aphasia, perseveration, and ideational apraxia. Autopsy revealed a large subdural hemorrhage severely compressing the entire left hemisphere. The hematoma was 4 cm. thick at the greatest dimension. (See Fig. 9.)

In the study of a case consideration of the associations which may still remain to the patient is important. For example, if the patient is told "put your finger on your nose," he may fail entirely, not because he cannot do it but because it is an unusual proposition. If we take another course and ask "Have you a nose?" he will say "Yes." Then we ask "Where is it?" and he will immediately put his finger on it to show us. The stimulus thus reaches his "eupractic area" by a two-stage but much easier route.

The psychologic factors of interest and attention require constant watching on the part of the examiner. In some cases of aphasia it is almost impossible to get or to hold the patient's attention. One finds it necessary (if one *must* have results) to shake him or call loudly to him from time to time to arouse him. (This is hardly advisable; it is better to wait, if possible, for a better time or to examine him by stages.) In other cases the attention is easily held, but it is rarely so alert as in the normal individual.

At times or in certain cases when the attention has been obtained, it becomes fixed on a letter, a word, or an idea. This fixation is called persevera-

tion. Thus, the patient correctly replies to the question, "Where is the Empire State Building?" by saying "New York." If we then show him a pencil and ask him what it is, he may again say, "New York." Unless one is on one's

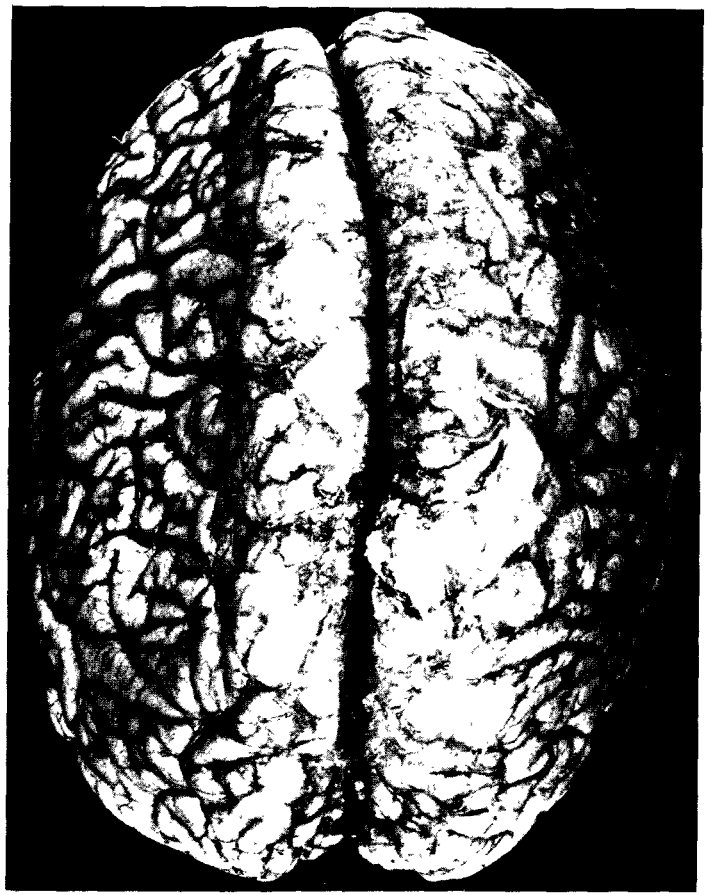

FIG. 9. Brain of the authors' case 64, B. Eld. The entire left side of the brain is severely compressed by the large subdural hematoma. The patient had semantic defects, perseveration, and ideational apraxia.

guard, one may conclude that the patient is entirely confused. But a change of subject or a little rest will produce another correct answer and perhaps two or three correct answers, after which the perseveration may return. The same precaution applies to writing. One patient who was named S. K. Davis began to write spontaneously (in one test) but produced only "S.K.SKSKSSSSSSS." Perseveration of ideas may also occur so that the

patient continues to talk about the first subject mentioned, and special effort is necessary to change his train of thought. Sometimes it cannot be changed.

Charcot taught that individuals vary a great deal so far as the relative importance to them of the auditory or the visual centers is concerned. Thus a visually minded individual who suffers a destruction of the occipital lobe will be much more affected by that lesion than one who learns best by hearing. In our clinical studies these factors (early training) are usually entirely unknown; we take the patient as he is and attempt to determine his lesion, but if the personal habits and traits were known, our analysis would undoubtedly be more accurate.

Hughlings Jackson in his numerous writings stressed repeatedly that patients with motor aphasia who were unable to say voluntarily more than a few words (and usually the same ones over and over) were nevertheless able to swear exceedingly well. He first called attention to the fact that there are two kinds of speech, emotional and propositional. The relatives of patients often stated that the aphasic could say almost anything "by accident" but could not say the same thing when he wished. S. A. Kinnier Wilson (1926) cites the case of one of his patients who could not say anything propositionally except the usual few words but who, on seeing a German Zeppelin fall in flames on the outskirts of London during World War I, exclaimed, "Hallelujah!" He could not say the same word again when asked. There is no doubt that the anatomic and physiologic patterns for the utterance of any word are still present in the brain after destruction of Broca's convolution of the major (left) side. (Von Monakow believes motor aphasia to be due to a disturbance of evocation.) The patterns are located anatomically in the right Broca's convolution as Jackson thought and need only the proper stimulus to bring them into action. They can be stimulated through the emotions, though the mediation is unknown. Jackson believed that the right side of the brain functioned in emotional speech. Henschen, fifty years later, stated that if the right side of the brain could not be placed into our scheme for explanation of facts in aphasia, the entire structure of aphasia would have to fall to the ground. Wilson (1926) recited the interesting case of a woman who had a lesion of the right side of the brain and who had lost all intonation and emotional speech. He did not consider this at all conclusive, but it is certainly interesting.

Closely related to these questions are some interesting facts about transcortical aphasias. In transcortical motor aphasia the patient is unable to say anything spontaneously but able to repeat anything asked of him. I followed for over a year a patient of this sort (Paul Sch., case 87) who had suffered a right hemiplegia and had Broca's aphasia which gradually gave way to a perfect transcortical motor aphasia. If we asked him to say "Sacra-

mento" or "Schenectady" or even "I have been in California for 30 years" he would say it perfectly. He did not repeat anything he heard unless specifically asked to do so. Now when we said "Say 'Sacramento'" he did not say "Say 'Sacramento'" but only "Sacramento." This was invariably true. In other words, he was not compelled to repeat what he heard; he was only through our command rendered capable of saying it. He listened to our request, decided what portion of our speech to say, and then said it; there was considerable mentation in the process. This is another way of saying that the impulse did not go directly from the hearing center to Broca's area on either side, but up to his "higher psychic area" (wherever that is; probably it involves a large part of the brain), and then the stimulus went to Broca's area. But if it went from the higher psychic area of the brain, where analysis occurred, to Broca's area, why could it not go when he independently decided to send it? Why was it necessary to have the outside stimulus to enter the area for hearing and start the impulse on its proper course? The answer is that the function was performed by the right (untrained) side of the brain.

The question of a "concept center" has been under discussion ever since Lichtheim first inserted such a center into his scheme of aphasia. Henschen (1922) has exhorted us not to forget that such a center is entirely imaginary, and Wilson (1926) has shown how ridiculous it is to place on the same drawing such a psychologic concept center and a physiologic center for movement of the hand in writing and then to connect them with a line. Yet, with all this in mind it seems certain that, in talking, impulses do not go directly from the word-hearing center to the word-speech center. In ordinary speech one hears what one has already said, "checks up" on it, analyzes it, and corrects it; and this often causes one to change one's diction or even one's phrases in the course of a sentence. The portion of the brain which thus holds the thought and checks up on the speech to determine whether the speech mechanism properly expresses the concept is the attention or the concept center. It would seem that the reasons for the impossibility of placing the concept center in the diagram are that the center is constituted of much of the cortex and that we know practically nothing of the multitudinous paths traversed in the elaboration of a thought. The thalamus is included in the circuit. Goldstein (1915) attempts to outline these paths to some extent.

To be sure, Broadbent and later Mills believed that a naming center (which Mills considered identical with a concept center) existed in the temporal lobe. Mills placed it in the third left temporal convolution. I am unable to conceive of the identity of a naming and a concept center because we have unnamed concepts. It is true that lesions of this area cause loss of memory of names and nouns in general, but so do lesions in the surrounding area.

and the center for the correlation of sensory impressions appears to be centered about the angular gyrus. An anatomic concept center seems an impossibility, a concept area a reality.

One often hears of word-sound deafness and word-meaning deafness. The distinction in concepts is good, and the distinction stresses the fact that in disturbances related to understanding of the spoken word the defect may be on the level of hearing *or on the level of comprehension*. However, while there is such a thing as deafness for sounds in general and, in the sense of agnosia, for words in particular, there certainly cannot be such a thing as deafness for word-meanings. The meanings of words are determined by the correct functioning of much of the brain.

The method of teaching the art of reading has changed (in certain parts of the United States) in recent years. This change has brought new types of reading disability. Children formerly learned to read first letters, then syllables, then words. Now they learn words as a whole, with the result that they learn to read words which they do not know how to spell and without recognizing the individual letters. In other words, it is the Gestalt idea applied to reading. By this method children certainly learn to read more quickly but less accurately. Later, when they develop aphasia they cannot be made to analyze the word into its elements; they guess at the entire word and attempt to determine what it is by the context which, when aphasic, they perhaps cannot read. In such cases asyllabia does not develop.

The psychologic factors in writing are also important. The present writer has had a personal experience with writing which throws light on the psychologic factors of the art. He has all his life been a good speller. Any word which he has known in any one of five languages he has been able to spell. It has seemed to him that spelling was an unnecessary subject, since it came automatically. Then at middle age he decided to learn the use of the typewriter. To his great chagrin he found that he could not spell on the machine. Such common English words as *success, business, bowels,* and *keys* were misspelled. The errors were always recognized, but for the first time in his life spelling had to be learned. He could always get words correct if he spelled them quietly to himself before writing, but this method was too slow. The reason for the difficulty was that the old set of hand movements had been subtracted from his mechanism. Gradually a new set of cheirokinetic movements have now been developed and engrams formed so that spelling of words which he has previously written on the machine is easy. Yet each time a new word is to be written he has to pause to think.

Now, he has no cerebral lesion. But suppose in an illness he were suspected of being aphasic, examined for his ability to write, and furnished with blocks

of letters to build words. He would be found unable to write. Is it not probable that one would assume a lesion of either the angular gyrus or the frontal lobe or both?

Many normal persons tend to think faster than they can possibly write and hence have trouble in spelling because of "condensation." They write "His pelling" for "His spelling," etc. When this is encountered in aphasic studies, one must be cautious about deciding on an anatomic lesion as the cause.

In certain cases it is indeed difficult to distinguish between general mental dysfunction and aphasia. The verbigeration, perseveration, and palilalia of dementia praecox may easily be mistaken for jargon aphasia. Certain otherwise normal adults do not know right from left.

It is the misinterpretation of such cases as have been cited which has caused endless confusion in the anatomic study of aphasic manifestations.

In concluding the discussion of the importance of the psychologic factors, it is admitted beyond question that speech is a mental process. The difference of opinion centers about the degree of divisibility of the psyche for various elements of speech. Certain writers have taken the stand that there is a disturbance of the entire psyche in every case of aphasia. They have correctly pointed out, for example, that in every case of motor aphasia the patient does not comprehend spoken language as well as the normal person does and that in every case of acoustic verbal agnosia the patient fails to speak spontaneously as well as the normal individual. These statements are true. How, indeed, can the brain function as well after the functional deletion of a portion as it could before? On the other hand, there are 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 motor aphasia, forgetting of names in amnesic aphasia, loss of comprehension of spoken words in acoustic verbal agnosia, or loss of recognition of written words in visual verbal agnosia, the remainder of the psyche is often comparatively little affected. The general loss which is seen in every case seems to correspond to the form well named by Head—semantic aphasia. We grant, then, that there is an element of semantic aphasia in nearly every case. With the psychologic factors which have been described here carefully taken into account we can proceed to a fruitful study of cerebral localization based on agnosia, apraxia, and aphasia.

PHYSIOLOGIC AND ANATOMIC FACTORS

1. Perception, eugnosia, and agnosia.

The newborn infant hears many sounds about him which at first are without significance. He soon learns that certain ones are associated or accompanied with certain experiences, and these in turn acquire an emotional tone, i.e., they

are pleasant or unpleasant. He probably does not distinguish words for a long time but does distinguish intonation, pitch, rhythm, and probably in general timbre. After a time sentences become separated into words in certain instances, and these entities seem to him to recur. Up to this stage, then, the infant hears sounds (utilizes the function of perception) and stores memory pictures (develops engrams) of certain words (utilizes the function of auditory verbal eugnosia). Physiologically speaking, there are two levels of integration: primary perception * and eugnosia. Destruction of the first results in deafness, of the second, in agnosia, acoustic or auditory agnosia if complete, acoustic verbal agnosia if recognition of words only is lost. These are mere matters of definition.

The anatomic sites concerned in the storage of these memories (the development of engrams) are not the same for both. A patient may become sound deaf so that he does not hear anything; he then has lost primary perception. He may, however, retain ability to hear sounds but lose the ability to recognize that he has ever heard them before. Or he may retain memory of having heard general sounds before but not the memory of having heard sounds of words before. The anatomic site of formation of engrams of words is therefore different from that for other sounds.

The first transverse gyri of Heschl, one on each side, are the sites for perception of sounds. An area immediately adjacent to these (paratransversal area), in the superior temporal convolution (Wernicke's area) is (according to many authors) the site essential for recalling memory pictures of the sounds of words; while the tip of the temporal lobe (according to Henschen, 1922) is the site for musical sounds.[1] Under normal conditions the minor side cannot function correctly for very long. (See also the works of Hughlings Jackson and of Henschen.) If the minor side does function as in aphasia, it acts inadequately and incorrectly. The mishearing which occurs is called paragnosia. In the case of music the minor side takes over the function more readily and completely than in the case of words (Henschen, 1922, and the case of G.O.R. in the author's series).

So far only the recognition of sounds has been considered. We must now consider the anatomy and physiology of significance of sounds. In most important languages the same sound in certain instances has various significances according to the context. The infant in our previous illustration has no

* While the function executed by the first transverse gyri of Heschl is called primary perception, it is quite possible that some sort of sound perception occurs in the subcortical stations, but this is of no practical importance in the human subject.

[1] In view of the case of Fox and German (1935) and the case of G.O.R. (under Temporal Lobectomies in Chapter X) in which the major temporal lobes were removed, there is no doubt that memory pictures of sounds heard (engrams) are formed on both sides. These patients understood much of what they heard.

context except the pleasant and unpleasant associations. In the English language of the adult the sound *right* may be written *right, rite,* or *write.* The significance of the sound cannot be determined by just the sound itself; the associations must be taken into account. The associations are composed of other auditory memories, of visual memories, kinesthetic memories, and usually many other types. This means, then, that before the significance of the word can be determined, the greater portion of the cortex must be consulted, i.e., impulses must travel over it. In other words, there cannot be such a center as a *word-meaning center.*[2]

This descriptive picture may be carried a little further. The portion of the cortex necessary for the determination of the meaning of a word is not nearly so great as that necessary for the determination of, e.g., a point in law. Indeed there may be, and often is, insufficient cerebral substance, function, or training in one person to determine a given question.

Disturbance of function in the realm of significance of words is no longer agnosia; it is aphasia. If we wish to stretch the definition of the term agnosia to include loss of recognition of significance of words and propositions, etc., we may do so but the term agnosia was very properly suggested (by Freud, 1891) to distinguish between various functional levels, and if we call disturbances on the higher level agnosia, the term becomes useless. We, therefore, in harmony with fairly general custom, call disturbances in language above the agnosia level aphasia.

What has been said of the mechanism of hearing and interpretation of hearing applies similarly to vision and interpretation of visual impulses. The primary visual center is in area 17 of Brodmann[3] (about the calcarine fissure). Here visual impressions are received. In the areas about the occipital and occipitoparietal cortex of both sides are stored memory pictures (engrams are formed) of what has been seen. Complete lesions of the area 17 about the calcarine fissure cause total blindness. Lesions of the area about the occipital and parieto-occipital cortex cause various types of agnosia according to the specific portion involved, but unilateral lesions cause agnosia only if they are on the major side. (The variations are discussed in detail under the various types of agnosia.) The major occipital lobe is not necessarily ipsilateral to the major temporal lobe. As for the portion of visual agnosia concerned with

[2] From our study of case of G.O.R. in which the entire left temporal lobe was removed we are forced to conclude, however, that the vertical portion of the major temporal lobe between Wernicke's center and the angular gyrus (chiefly area 37 of Brodmann) is of much more importance than other parts of the brain in determining the significance of a written or printed word. Many more recent similar cases render this impression certain.

[3] There is possibly a lower form of visual perception, only light perception, in the subcortical centers (see the work of Mettler, Mettler, and Culler on visual perception in decorticated dogs), but this is of no practical importance in the human subject.

language, the memory pictures necessary for the recognition of written or printed words utilize the region of the major angular gyrus. A lesion of the specific area (or certain paths leading to it) causes visual verbal agnosia. The cortical area for mathematical figures is located, according to Henschen (1922), about the borders of the interparietal sulcus.

As pointed out in the discussion of the auditory function in language a lesion above the functional level where it must be to cause agnosia, causes aphasia. In the visual sphere we may have recognition of objects or words seen but with loss of their significance. So far as words are concerned this is the difference between word blindness and word-meaning blindness. Thus the word *dog* in a book of general literature means a carnivorous quadruped of a certain variety. The same word in a technical description of machinery may mean a small metal contrivance used to keep a toothed gear from turning. For the differentiation between the two much of the cortex is required,[4] as the context must be interpreted. Loss of ability to recognize the word constitutes agnosia; loss of ability to determine the significance of the word (sufficient intellect being retained) constitutes a form of aphasia. It will be clear that the term alexia is too broad for anything but descriptive purposes, as inability to read may depend on many different focal cerebral defects.

Tactile sensibility is perceived but not accurately localized in a subcortical center, the thalamus. This subcortical center is comparable to the subcortical centers for vision and hearing but is much more important clinically than those. A lesion in the thalamus may abolish all contralateral sensation. The cortical area for recognition or accurate localization of sensation seems to be large and to include the post central gyrus of each side and additional parietal cortex. Destruction of the postcentral gyrus does not abolish but only reduces the tactile sense. Destruction of the parietal cortex or severance of connection between the parietal cortex and the subjacent thalamus causes tactile agnosia (commonly called astereognosis). There is no major or minor side for tactile agnosia, each governing the opposite half of the body. Tactile aphasia is loss of ability to name objects or symbols felt while ability to name them when seen or heard is retained. Such cases have been recognized clinically. I have observed one.

Olfactory and gustatory agnosias are entirely hypothetical; it has not been possible to distinguish between those and peripheral lesions. Even for normal persons the identification of substances smelled and tasted is usually markedly defective.

[4] As mentioned before, however, it is probable that area 37 of Brodmann is far more concerned with interpretation of written verbal symbols than are other parts of the cortex. Historically this seems to be the first sign of localization of a sensory aphasic symptom. Little is here said of it because the knowledge rests on a few cases.

2. *Eupraxia and apraxia.*

Purposeful movements. An impulse "originating" in the Betz cells of the motor cortex travels to the intercalated neuron at the motor cranial nerve nucleus or at the anterior horn cell of the spinal cord. One often conceives of this as the motor mechanism. More attention should be given to the association cells of area 4 of Brodmann and the prefrontal cortex, particularly area 6. These association neurons form the anatomic basis for the mechanism by which movements and not mere contractions result from stimulation of the motor cortex.

Now, when any movement is made frequently, engrams are formed in and anterior to the precentral gyrus as also in the brain stem and spinal cord. These engrams must not be thought of as structures differing from immediately neighboring structures; they are pathways exactly similar structurally to those not used but which have been trained by repeated use. In other words, they are functionally educated structures over which impulses pass with much greater facility than they did the first time impulses traveled over the same route. When an idea is to be executed (by motor act) an impulse travels from somewhere in the brain to the precentral gyrus where the proper group of association cells is stimulated to effect utilization of the desired engram.

The clinical application of this mechanism is that when a group of Betz cells themselves is affected by disease, paralysis of the corresponding fibers results. This is an upper motor neuron lesion. On the other hand, when the association cells of or anterior to the precentral gyrus are affected by disease, apraxia results. There is not any paralysis in the ordinary sense of the word. If, for example, the right hand is affected in this way the patient cannot thread a needle so well as formerly, because he has forgotten how to make the movement. He now does it, if at all, as though he were doing it for the first time. If the lesion were to affect the area immediately anterior to the Betz cells of the major side which are utilized for movements of the head, especially of the mouth, the patient would forget how to make accurate movements with the mouth. If those still farther forward which are necessary for speech were affected, motor aphasia would result. Or if the area just anterior to the group of cells used normally for movements of the hand in writing (of the major side) were affected, he would be unable to write. While unable to write, he might be able to thread a needle nearly as well as ever. This selectivity depends upon whether the specific structures trained for one or another certain purpose are involved. It is thus possible to find clinically apraxia for certain movements only. This type of apraxia is the so-called kinetic apraxia of the limbs of Liepmann, the cortical apraxia of Heilbronner,

and the innervation apraxia of Kleist. (I propose to call it cortical motor pattern apraxia because it may affect axial musculature as well as limbs and it is clearly due to destruction of a cortical motor pattern.)

It was thought for many years that the two cortical areas for movements of the hands were so related that the one on the major side (usually the left) not only governed the movements of the corresponding hand but also governed the function of the similar area of the right side. This was supposed to be the reason that a lesion of the hand area of the left side had the effect of causing an apraxia not only of the right hand but of the left as well. This was Liepmann's sympathetic apraxia.

It now seems certain that such apraxia occurs only in persons who are strongly left-brained, not only for speech but for ideational plans of motor acts. Such a state is relatively rare for which reason apraxia is much rarer than aphasia and is usually transient. The minor side of the brain soon learns to make its own ideational motor plan.

So far there has been presented the mechanism of apraxia of the type called kinetic apraxia of the limbs. There are other types. For an understanding of the other types, it is necessary to introduce another concept. One has in one's memory not only kinetic pictures of movements made but also visual memories, i.e., memories of how the movement *looked*. There are also doubtless memories of how the movement *felt*, gained through the fibers of deep sensibility and recorded in the parietal lobe.[5] These memories all seem to be synthesized about the region of the supramarginal gyrus or at least about the parieto-occipital cortex. The left side is usually the major. It thus comes about that when after due reference to these memories a decision has been made to execute a certain movement, the impulse must travel from the parieto-occipital cortical region to the area about the left precentral gyrus governing movement so that proper synthesis may be made. If this impulse is interrupted by a lesion of the left parietal lobe an *ideokinetic apraxia (of Liepmann)* results. The patient has the idea intact and knows what he wishes to carry out, but he misses connection, as it were, and does something else. This form of apraxia, it will be observed, is on a higher functional plane than the form first described. This form also has a definite relation to writing, as the patient will be unable to connect his ideation with motion, although both are intact.

[5] Bastian believed that the memory picture of how a movement as in writing was made was recorded in the motor hand area (chirokinesthetic center). This meant that the motor strip was sensory. He explained that motion was not produced by stimulation of the Betz cells; the only effect was that of passage of an impulse to the anterior horn cell. If one wished motion, one must stimulate the anterior horn cell which therefore was the motor cell. With this explanation he was free to call the area in the frontal lobe chirokinesthetic. This bizarre idea later received some support from the work of von Monakow (1914) who extended the sensory cortex across the motor strip to the frontal cortex anterior to it.

A third type of apraxia is met with in diffuse lesions of the cortex—the ideational apraxia of Liepmann. In this form there is so much interference with the synthesis of the idea as well as its execution that disjointed acts result. Each element is correctly carried out, but the various parts do not collaborate to produce a purposeful act. This form of apraxia is on the highest plane.

In either ideokinetic or ideational apraxia another anatomicophysiologic factor plays an important role. This is the fact that the corpus callosum is the anatomic structure by which impulses are carried from one side to the other. And Sittig (1931), following Mingazzini, has shown that the anterior portion of this fiber system functions especially for the upper part of the body (the mouth), the middle portion for the upper limbs, a portion farther back for the lower limbs, the splenium not at all in apraxia. He has also discovered that apraxia of the trunk not only occurs but may be isolated. This is referred to an intermediate portion of the corpus callosum. These facts are of additional localizing value.

3. Speech and aphasia.

Motor speech. When the newborn infant has heard certain words or expressions a number of times, he attempts to reproduce them. In the average individual the attempt is made long before success is to be expected. In a few instances an infant has said nothing until he has produced complete words at the first attempt. In either case it is clear that the memory of the sound he has heard must be re-experienced, must be "heard" silently before he can say it. His vocal effort is then guided by an (indirect) association between the hearing mechanism and his speech mechanism, the principal part of which is located in the convolution of Broca in the foot of the third frontal convolution.

In Broca's convolution are engrams laid down by efforts to make the movements necessary for production of vocal sounds. When these engrams or the neurons which constitute them are destroyed, the patient still knows what he wishes to say, i.e., he still has clear thoughts; he can still hear the sounds silently in his head, but he has forgotten how to make the movements necessary to produce the sounds. But as the area of Broca with its association neurons to and from the center of Wernicke are part of the mechanism of speech, there is imperfect comprehension. He can still move all the muscles for chewing, clearing his throat, expectorating, etc.; it is only movements to make sounds constituting symbols of speech that he has forgotten. This is apraxia, a very specific apraxia. Motor aphasia is then a type of apraxia. But since it pertains to language and symbols, it is by definition a type of aphasia.

One must keep in mind that a complete sensory aphasia may so blot out

speech concepts that the patient cannot talk. Also, if the patient has aphasia on the level of formulation of language he may not be able to correlate his thoughts for expression in symbols and for that reason not be able to speak. One must, therefore, be careful about concluding that the aphasia from which a certain patient suffers is necessarily motor in character. When the language formulation area is gone the patient fails to talk because he has nothing to say. Furthermore a severe sensory aphasia may simulate very closely a motor aphasia by producing mutism (which, however, is usually temporary).

Just how Broca's area of the major side receives its impulse to speak is not entirely clear. It certainly cannot alone initiate impulses. When Broca's area on the left side is destroyed, the patient, as Hughlings Jackson has so clearly pointed out, can nevertheless say *something*, usually the same thing over and over again. It seems from the reported cases that he can really say, or rather utter, *anything* provided the proper or necessary stimulus is found. We are indebted to Jackson for our earliest knowledge on this subject. He it was who showed that the patient could swear after he became aphasic (and not neces sarily always with the same terms) as well as he could before the aphasia developed. Jackson classed all expressions used for emotional relief as swearing and arrived at the generalization that emotional language as a whole is not particularly curbed in motor aphasia. Jackson believed that emotional expressions were spoken with the right side of the brain, and subsequent evidence has borne out that contention. He thus reached an anatomic fact through a psychologic approach. Zollinger's patient with the entire left cerebral hemisphere (pallium) removed had ability to say a few words. The impulses must have originated on the minor side.

Wernicke believed that the area now known as Wernicke's center in the superior left temporal convolution guided functionally the structures of Broca's convolution, that interruption of the pathway from the former to the latter caused paraphasia. But in the series collected by Henschen there were 5 cases in which in spite of auditory verbal agnosia the patients had intact internal language. This was evident by their ability to write. Wernicke's center, then, is not necessary for internal language. In the concept of the writer, the internal language takes place by proper impulses passing from the language formulation area to and from Broca's convolution. Broca's area is thus normally stimulated from a considerable area of cortex, but by routes which no one has established. The external capsule is essential to this mechanism. Broca's area of either side may in some cases function in speech if stimulated from the language formulation area but this is not true in all individuals.

When the patient swears in spite of destruction of the major Broca's con-

volution, he swears by impulses going not from the intellectual concept area centering about the left angular gyrus or the language formulation area but probably from similar but poorly trained areas of the right side which function only for the crudest language and thus to Broca's convolution of the same side.

In the case of a patient with transcortical motor aphasia in which he cannot say anything spontaneously but can repeat what he is told to say, the impulse apparently goes directly from the intact area of Wernicke on the left or the right side to the untrained right Broca's convolution with only incomplete analysis in the higher psychic areas of the brain, i.e., without all the normal association with the remainder of the cortex. The anatomic area here called higher psychic area probably covers a large part of the cortex. In normal speech, however, the functionally higher psychic areas supervise what is said and also act as a supervisory body where abstract thinking goes on and concepts are stored. The mechanism of this supervision is that of impulses traveling back and forth from all sites where memory pictures are stored to the correlating center and vice versa a great many times. There is a double defect in transcortical motor aphasia. Not only does the patient say whatever he is told to say, but he cannot initiate speech. It seems as though impulses entering through auditory channels are shunted to Broca's center because of a crippled language formulation area.

Transcortical sensory aphasia presents a peculiar problem. In this form the patient can repeat what he hears without understanding it. He does not understand it when he himself has said it parrot-like. Here we are dealing with a short circuit, as it were, the impulse going on a physiologic basis apparently directly from the word-hearing center to the motor speech center without going into the higher psychic areas for analysis. It is commonly stated that in this condition the sensory cortex is separated from that of the rest of the brain. That statement appears to be true for all but the pathways to the cortex for motor speech. Heubner (1889) reported a case in which the sensory cortex was apparently isolated from the rest of the cortex above, below, and behind but not from in front. Still it is difficult to conceive how the association fibers could get under the sylvian fissure in that direction. One must keep in mind that the auditory fibers have to get *to* the auditory cortex and association fibers *from* it toward Broca's convolution. I believe that the syndrome results when the minor side is compelled to function, crude as its training may be.

The author's concept of a language formulation area is new. Evidence will be presented in a later chapter to show that area 37 of Brodmann in association with the posterior part of areas 21 and 22 constitutes a zone in which

auditory, visual, and motor memories are synthesized to formulate concepts into finished language. Not only are the words chosen (diction results) but grammar and rhetoric are managed in that zone. Paraphasia results from the disordered function of this zone coupled with a relatively good Broca's convolution.

4. Reading and alexia.

Reading. When a child first learns to read symbols, a certain symbol is pointed out to him, and he is told that this is, e.g., an A. He perceives the A with the primary visual area about the calcarine fissure of both sides. He records the observation in the region of the angular gyri (cortex) for future use. At the same time he says A and an engram is formed, a picture of the movement of the lips, tongue, and vocal organs in general in the prerolandic area for such movements. As he pronounces the A he also hears the sound in the first transverse gyri of Heschl of both sides and records the memory picture (makes an engram) of the sound in Wernicke's center of the two sides. (However, the memory pictures thus recorded on the right side cannot function except in a crude way unless they are used from time to time.) It is probable that the child also records in the parietal lobes the positions of the muscles used in speech.

The next time he sees an A and tries to read it, if the first training has been successful, he will recall the seen image, the felt image, an image of the movements necessary for pronunciation of the sound, and many other impressions associated with the first recording, all of which makes a concept, the concept of A. When he reads the A the second time he goes through the entire performance of gathering the associations, and he strengthens the first impressions, improves his engrams, and has then laid the foundation for reading. As his training proceeds he dissociates the lip movements from the rest of the process until the engram of these no longer is a necessary part of the process of reading. Before this dissociation takes place, not only will a lesion of the visual memory engram cause alexia, but so will a lesion of the engrams of the movements of the lips and vocal organs. Thus, in imperfectly educated individuals alexia may result from a frontal lesion. After the dissociation has taken place, a lesion in that area will not cause alexia. What has been said applies to reading of letters and words. Mathematical figures, i.e., the engrams of their appearance, are recorded in a slightly different location, at the interparietal sulcus, according to Henschen.

The author feels that the term alexia, while useful descriptively, is often misleading because insufficiently specific. The symptom may result from pathologic changes on three different physiologic levels:

1. Agnostic alexia is the failure of recognition of letters, figures, syllables, and words. The lesions which can cause this type of alexia may involve only the white matter of the left occipital lobe or this plus involvement of the

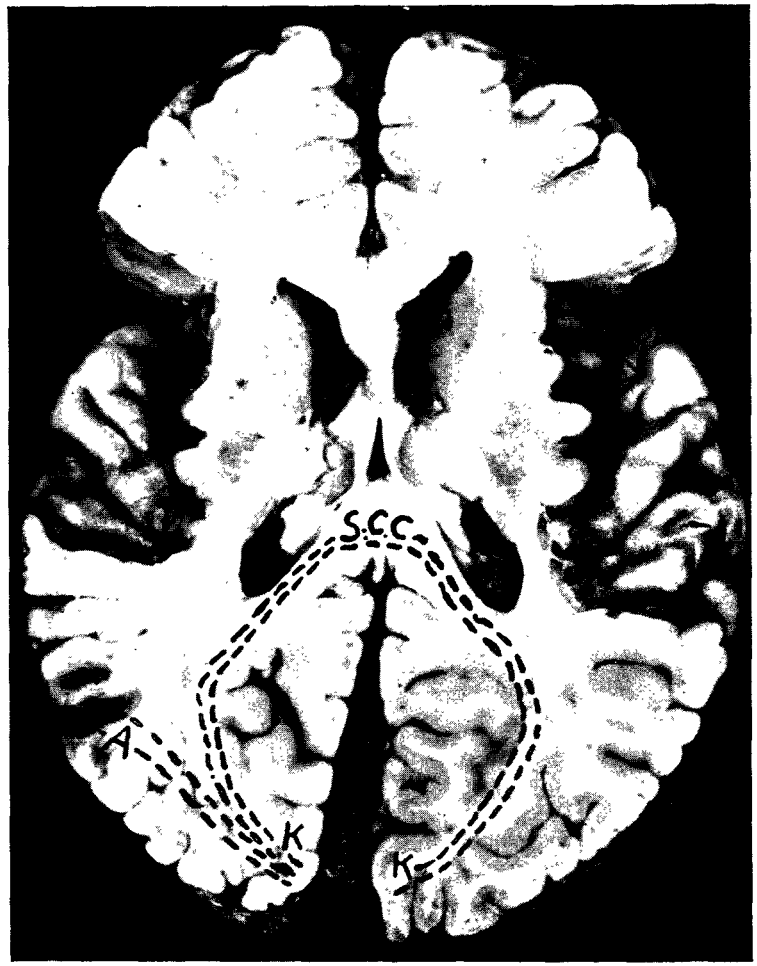

FIG. 10. Diagram (on a dissected specimen) of the simplest possible pathways for the recognition of the word seen. *A*, angular gyrus. *SCC*, splenium of corpus callosum. *K—K*, calcarine area.

splenium of the corpus callosum. This is discussed in detail under the heading of visual agnosias (Chapter IV). (Agnostic alexia is, strictly speaking, a type of aphasia but it is a more specific term than aphasia.)

2. Aphasic alexia is the loss of ability to comprehend statements of a simple

character, the words being properly recognized. Henschen believed, and subsequent investigation has proved him correct, that the angular gyrus cannot, as a rule, serve for interpretation of written or printed matter if anatomically dissociated from the area of Wernicke. For this reason acoustic verbal agnosia is usually accompanied with alexia. A lesion between the angular gyrus and Wernicke's area has the same effect.

3. Semantic alexia is the loss of ability to comprehend complicated statements, although the patient is still able to grasp simple ones involving a few words. This type, like that of semantic aphasia itself, results from a lesion of the temporal lobe involving Wernicke's area and the language formulation area.

An extremely instructive case of psychic blindness (referred to above) is recorded here to illustrate the mechanism of reading.

Case 8. Gilbertine M., a white nurse of 49 years, who had been instructor of nurses for three years at a Los Angeles hospital, was well until she was taken with acute anterior poliomyelitis in the epidemic of 1934. She had the usual course of pain in the extremities characteristic of the polyneuritic form of poliomyelitis seen in adults in that epidemic. There was no actual paralysis at any time, but the exquisite tenderness of the muscles remained for over six months, and much phenobarbital was given as a soporific, because she did not tolerate other sedatives.

The case was unique from one standpoint. After about three weeks of the illness the patient began to show mental symptoms which were at first thought to be due to a toxic psychosis. For this reason she was transferred to the service of Dr. S. D. Ingham at the Los Angeles County Hospital. There the present study was made. It was found that the patient recognized nothing that she saw. This was particularly true after the administration of phenobarbital which continued to be necessary.

She remarked to the nurse in charge that it was strange how all the nurses had taken to wearing black uniforms instead of white. (Von Monakow has reported a similar symptom in a patient.) When we asked her to read, she claimed she could not see. When an O about 10 cm. in height was written for her, she peered at it and said we were holding it too close. The position was corrected, after which she said we were holding it too far away. She kept turning the paper about and peering in various directions but was unable to read it. She then traced it with her finger (proof that she could see) and immediately read it correctly. Other letters were then tried, and it was found that she could see even small letters and could read complete sentences if she was allowed to trace the letters with her finger. She even began to complete words before she had quite finished tracing them. Here it should be noted

that she traced by arm, not finger, movements as the paper was held in the air 18 inches from her. On certain days she did not recognize colors at all; on other days she recognized some of them.

A further study of the case from the general standpoint of visual agnosia was made. While this does not exactly apply to the problem of reading, the important other findings should be recorded here for the sake of completeness and for future reference.

When shown a bunch of keys (care being taken by the examiner not to rattle them) she had no idea what they were. As soon as they were shaken, she said, "keys." A large saucepan was exhibited. She had no conception of what it was. When it was tapped with a spoon, she immediately said, "Oh, a pan." A watch was shown to her. She said it was something bright but that she could not tell what it was. It was then placed to her ear, whereupon she immediately said, "watch." She could not identify a piece of paper until it was crumpled for her. The possibility of a mere optic aphasia of Freund was excluded by the expedient of suggesting names of objects for her while she was attempting to determine by vision alone what a given thing was. This was of no assistance to her.

An orange was next presented. She did not recognize it. It was put into her hand, whereupon she said it was fruit but could not tell what kind. She was then allowed to smell it, and immediately she said, "orange." When her daily tea was about to be served, it was shown to her, but she could not identify it by sight. When she tasted it, she immediately said, "tea."

A large piece of newspaper was shown to her, but she could not identify it until it was either placed in her hand (touch) or it was crumpled for her (sound) when she immediately identified it. She identified by the sense of touch everything she handled, and she identified all visitors by the sound of their voices.

When this study had been done, her ability to write was tested. She claimed that she could not write as she was unable to see. When asked to write with her eyes closed, she wrote fluently, both spontaneously and on command. There was no sign of agraphia such as writing letters upon one another, or making large letters or sloping lines so commonly seen in persons with agraphia. She wrote perfectly normally.

How was this possible? Did she visualize the letters before writing and thus copy the visual images, or did she use the cheirokinesthetic sense entirely? To determine this point, she was asked to tell in words what a capital printed H looked like. She said it consisted of two upright lines connected across the middle with a horizontal line. Then she was asked to tell the difference between a printed capital O and a printed capital Q. She replied that the Q

had a tail on it while O was just plain round. She also described an A and a W.

This threw light on the mechanism of writing. It was clear that she merely visualized the letter and copied the visual image. In other words, her ability to revisualize letters was not lost, although she could not recognize a letter when she saw it. This was pure visual verbal agnosia.

The question now arose whether she was similarly able to revisualize objects in general. She was asked to describe the hospital at which she had taught nursing. She was unable to do so. She could not answer specific questions about it, such as where the doctors' parking space was located or how many steps there were to the main entrance. She could not even tell whether there were six or sixty. She could not tell what the lobby looked like. On the other hand, she was able on another day to describe fairly well another section of the city. She told of one building which was about twelve stories high, another smaller, etc., all of which was correct.

With regard to the method of reading by tracing the letters with her finger, it seems worthy of note that it was the examiner's handwriting she was tracing, not her own, and that she used arm movements, not finger movements. In other words, she generalized concerning the characters and still was able to identify the writing (and also printed matter) by memory pictures (engrams) in the left parieto-occipital lobe and its associations with the writing center in the left frontal lobe. She had also bilateral spastic reflexes and Babinski signs; so one may assume a diffuse cerebral involvement, but not any more on the left side than on the right. In the course of a year she learned to recognize large objects (such as a teapot) by tracing them in the air with her finger. In five years she learned to read as well as ever. Whether the left side recovered or the right side was retrained I cannot tell.

5. *Writing and agraphia.*

Writing. When the child first learns to write, he may, if left to his own resources, make marks of various sorts which he calls writing. If these are to him symbols (usually not the case), they are actual writing. As a rule, the child is not left to his own resources but is made to copy from sample. If he is asked to copy an A, he is taught at first to draw it. He finally succeeds. When he then wishes to write an A, he either hunts up the same or a similar one to copy, or he copies, if able to do so, the memory picture of the first one he saw and recognized.

If he copies from memory, the process involves the revisualization of the memory picture. This fact is important especially as it requires the function of patterns in the angular gyrus different from those required for reading. To

make this clearer, the area chiefly concerned with storage of memory pictures of letters and words is the region of the angular gyrus chiefly of the major side. By rupture of connections between the calcarine area and the angular gyrus the patient may be deprived of the use of these patterns and consequently be unable to read, yet still be able to revisualize what the letters look like (as in the case of Gilbertine M.). In harmony with this fact, Pötzl has shown that a lesion near the angular gyrus of the major side causes psychic blindness, while it is a lesion of the inferior surface of the occipital lobe and part of the cortex of the convexity of the left which chiefly causes the Charcot-Wilbrand syndrome of loss of ability to revisualize visual memories of relationships and possibly of objects. The loss of ability to revisualize words does not always go pari passu with loss of ability to revisualize objects.

This revisualization has been classified by Pötzl (1928) as an agnosia. I believe it should be classified as a cerebral dysfunction on a higher physiologic plane.

The difference between drawing and copying symbols is clearly shown by a most interesting case studied by the writer (case 4). William J. Can., aged 60 years, suffered a cerebral vascular lesion without paralysis. He was unable to talk; only unintelligible mumbling resulted whenever he attempted to answer or to talk spontaneously. He was not able to repeat any better what he was asked to say. On the other hand, he had very little if any auditory verbal agnosia or any visual verbal agnosia, as he was able to read, and no apraxia unless his agraphia was apractic. We determined his ability to read by showing him a series of words: bread, horse, wife, apple, house. He was asked to select the names of things good to eat. He drew crosses after bread and apple.

When he was asked to write something, he made a series of loops. He was then asked to copy the word California. He wrote a C and then a series of loops. When asked to copy New York, he wrote, "W. J. Cffoff" (a poor attempt at his name with much perseveration). He was then tried on figures instead of letters. We asked him to copy the figure 4. He made a scrawl. For the figure 3 he made a loop and a scrawl. There was in the marks not even a semblance to the figures. He did still less well with the left hand. (See Fig. 11.)

The question now arose whether he had lost the sense of direction and for that reason was unable to copy. He was asked to copy the picture of a small conventional birdhouse. He did it well. This involved vertical, horizontal, oblique lines, and circles, all of which were correctly copied. It was difficult to comprehend why he was unable to draw a figure 4 when he was able to copy a much more complicated figure. It seemed reasonably due to the fact that the 4 was a symbol and that he did not draw it but attempted to read it and then write it without drawing it at all.

To determine this point definitely I decided to draw something complicated but embodying the figure 4. A square was drawn with a vertical line through it thus

He copied it fairly well. He was then asked to copy the following figure:

He copied a normally written 4 which he had been totally unable to write. The right hand vertical line was then subtracted to make a perfect written 4. When asked to copy that, he made only an unintelligible scrawl. In other words, he could copy anything which could not be read, and he could make a 4 if it was not a symbol, but if the figure was a symbol, he attempted to read it and then write it without actually copying it as a drawing. He therefore had agraphia for both spontaneous and copying acts with retained ability to draw. He also had motor aphasia, but no other apraxia.

This study lead to the conclusion that the pathways used for simple drawing of a figure which cannot be read are shorter than those for even the simplest writing of a symbol. For the former there is no revisualization of a memory picture necessary. The impulse may go back and forth, to and from the visual cortex and the angular gyrus for direction, then to the frontal area for hand movements. If a symbol, however, is to be made, it is first read, i.e., classified according to previous experience. (The loss of this power is agnosia, but the present patient did not have that defect.) The impulses, then, could take the normal course, i.e., from the visual cortex they could go to the area for recognition of symbols. But upon attempting to revisualize the letter for copying on paper this patient failed. Henschen has shown that the normal course for impulses from the angular gyrus to the frontal writing center is probably through the external capsule. Since this patient had a motor aphasia, this concept would fit perfectly. The pathway for simple copying might take a shorter course, which in this case was still intact.

Unwarranted conclusions must not be drawn and the matter may not be settled, but I do want to point out that in these two cases (Gilbertine M. and William J. Can.) the first patient had a complete visual agnosia, yet was able to revisualize letters and could write spontaneously, better with her eyes closed (of course could not copy), while the second had no visual verbal agnosia

but had lost ability to revisualize letters and was totally unable to write or even to copy symbols.

To return to the account of the mechanism for writing. There is a writing

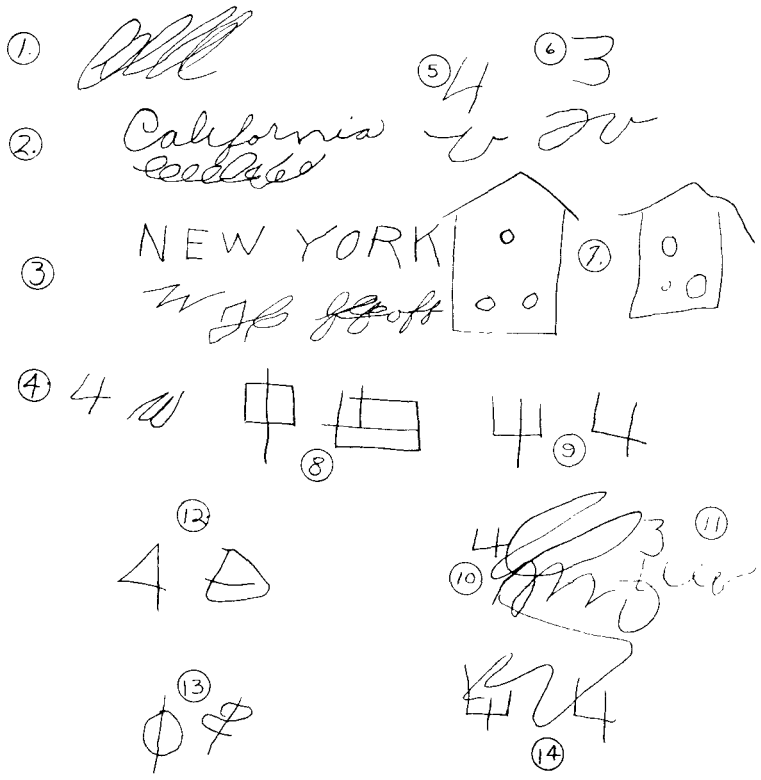

FIG. 11. Study of the writing in the case of Wm. J. Can. (Case 4) 1. Spontaneous writing. 2. Copy of the word California. 3. Copy of New York. 4. Copy of figure 4. 5. Another copy of figure 4. 6. Copy of figure 3. 7. Copy of our "conventional birdhouse." This shows that agraphia is not due to loss of sense of direction. 8. Copy of square with vertical line through it. This task was intended as an intermediate step in the copying of a figure 4 to keep out of the patient's mind the idea of symbolization. 9. Copy of a nearer approach to a figure 4. An actual 4 resulted when the sample was not a symbol. 10. Another attempt to copy an actual 4. Complete failure because the sample was a symbol. 11. Copy of figure 3. Failure when the sample was a symbol. 12. Another failure to copy a 4. 13. Copy of a circle with a vertical line through it. 14. Successful production of a figure 4 in an effort to copy something else.

center at the foot of the second frontal convolution of the major side (first postulated by Exner, 1881) which is especially trained from childhood through the formation of engrams to function as a writing center in the functional sense. In view of the cases gathered by Henschen there can no longer be any

doubt of this. From the two cases just cited it seems clear that this center if stimulated from one source will write symbols, from another, will not. It is therefore not a writing center in the sense of a center which can automatically write. It must be guided by the area for revisualization of the images of letters and words and must be governed whenever it writes by a sufficient intellect and by the areas particularly concerned with grammatical concepts (language formulation area).

If the right-hand center alone is rendered functionless and apraxia is thus caused, the patient may still be able to write with the left hand. Heilbronner has reported a case in which the patient had apractic agraphia in one hand and aphasic agraphia in the other. This is discussed more in detail under the heading of agraphia.

The writing mechanism is complicated indeed. We might approach a little nearer to a complete understanding of it by considering more in detail the syndrome of inability to read one's own writing as shown in the case of Gilbertine M. With Henschen's work at our disposal we may take his case abstracts and reclassify them to suit ourselves. By taking his cases of verbal agnosia and following them through to determine which of the patients were able to write, it is easy to find 9 cases of this kind with autopsy. These are the cases of Brissaud (vol. VI, page 143), Peters (vol. V, page 111), Hinshelwood (vol. V, page 75), Collins (vol. VI, page 145), Lissauer (vol. V, page 85), Mills and McConnell (vol. VI, page 19), Wyllie (vol. VI, page 165), Broadbent (vol. VI, page 7), and Skwortzoff (vol. V, page 122). An investigation of the pathologic findings shows that in all these cases the cortex of a large portion of the left angular gyrus had escaped. The lesions had affected in one case or another the occipital lobe except the extreme pole and the inferior and mesial surfaces of the temporal lobe in the posterior half. In some cases the lesion had approached very close to the inferior and posterior portions of the angular gyrus. It is fair to conclude that, since in all these cases the patients were still able to write, the affected areas are not essential to that function. In other words, these areas are not essential to the revisualization of visual verbal images. We must recognize that the writing of one's own name has so many associations that the right angular gyrus can practically always serve for its revisualization. I accept the dictum of Dejerine that writing one's own name is not propositional writing. One's name is almost a cliché, done almost automatically.

With this having been determined a summary of the author's concept[6] of

[6] In the first edition of this book I advanced the proposition that the posterior portion of the angular gyrus was more concerned with revisualization while the anterior portion was concerned more with recognition. I no longer believe this. The patterns are intermixed in the gyrus.

the writing mechanism may be presented. When one wishes to write, one starts activity in the neurons in the left angular gyrus and by this means revives memories of the letters and words one wishes to write. Impulses travel for

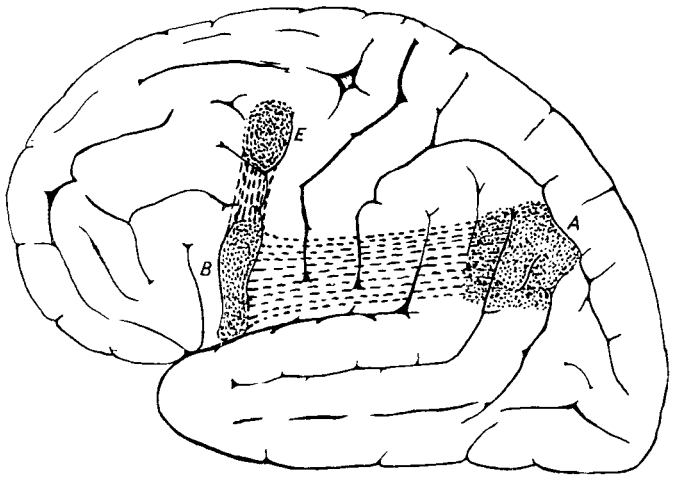

FIG. 12. Author's concept of the simplest possible writing center. A lesion in any part of this set of centers and connections may cause agraphia. E is Exner's writing center at the foot of the second frontal convolution of the major side. B is Broca's convolution A is the angular gyrus. Henschen has shown that the fibers from A to B go through the external capsule. In the majority of cases A must also have communication with the area of Wernicke in the superior temporal convolution.

association to all posterior portions of the brain and forward in the external capsule to the writing center at the foot of the second left frontal convolution. They probably go by fibers very close to the area of Broca. One passage of impulses is not enough. I believe that co-ordinated activity between the writing center and the left angular gyrus is necessary not only for the proper execution of the movements of writing but also for the proper concept and spelling of the words. Besides, the angular gyrus can usually not function for revisualization without association with Wernicke's area, though it can function for recognition without it. For this reason any lesion of either center named or of the pathways between them may cause agraphia. (See Fig. 12.)

The concept just given is strengthened by the fact that lesions of the frontal lobe near the writing center or even of Broca's convolution interfere with spelling and consequently with the concept of words. This is the reason that patients with Broca's aphasia cannot write. The reason that patients with aphemia (subcortical motor aphasia) are able to write is, according to this concept, that the fibers carrying the impulses between the angular gyrus

and the writing center either travel close to the cortex at, or are actually interrupted in, Broca's center.

If support is needed for the idea that, instead of an isolated center to do

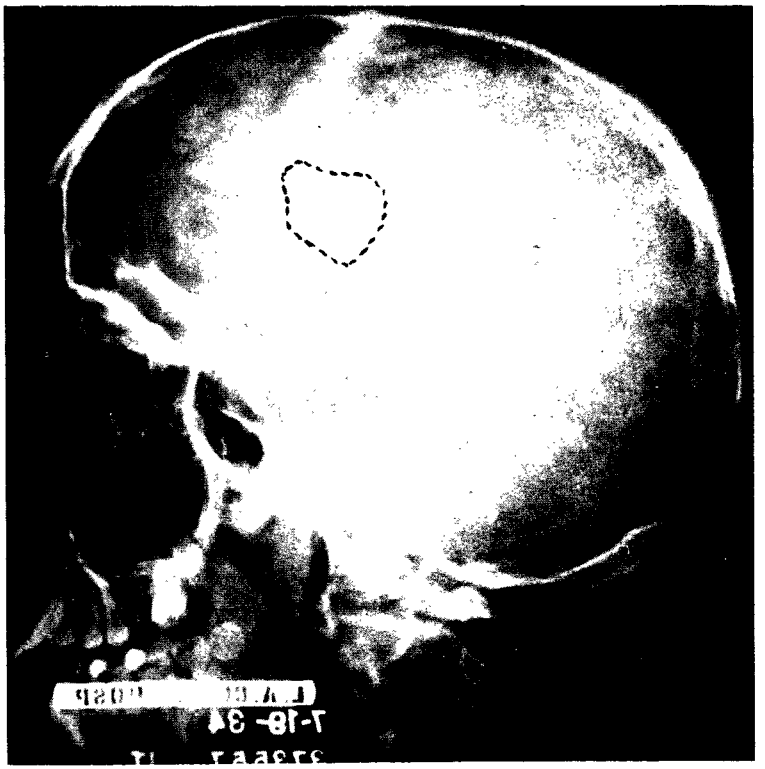

FIG. 13. Case of Laurence Gos. Roentgenogram of the left side of the head showing the fracture of the skull. The defect in the bone left by the surgeons after removal of the depressed splinters has been outlined in ink. The fracture lines extending up and down from the defect are visible. The brain has not been diagrammed in place as we wished to avoid leaving the impression that the damage to the brain is confined to the area below the defect in the skull.

work (which to me seems foolish), in reality several areas which are anatomically connected with fibers and functionally with impulses constitute a center in the physiologic sense, attention is called to the concept already expressed by Pötzl (1928) that the syndrome of psychic blindness plus visual aphasia for colors may be due to a lesion of the transverse occipital fasciculus of Vialet at either end of it. He has found that whether the lesion is at the lingual gyrus or the convex cortex of the occipital lobe, the same syndrome is obtained.

Further, Goldstein (1915) from a purely psychologic standpoint describes impulses traveling several times back and forth between areas before the function can be said to be performed.

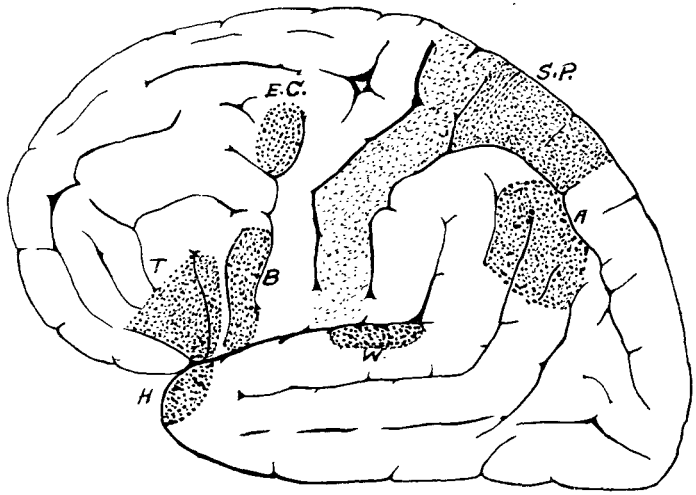

FIG. 14. Diagrammatic outline of the centers of importance in language. These are not centers in the sense that the areas function alone, but in the sense that they are far more important than the surrounding portions of cortex in the performance of certain functions. They can never function except in intimate co-operation with each other and with other portions of the brain. They exist equally on the right or minor side of the brain, and, in certain instances the minor side takes over the function easily for the major. *E.C.* Exner's writing center. *B* Broca's convolution. *T* pars triangularis of great importance in singing. *H* tip of the first temporal convolution (Henschen's music center) *W* Wernicke's area. *A* angular gyrus. *S.P.* superior parietal lobule. This with the lightly stippled postcentral gyrus is the most important area for tactile recognition in which a lesion causes tactile agnosia.

This concept further explains the old disagreement between Charcot and Dejerine about the writing center. Charcot claimed the existence of the center now proved to be a reality at the foot of the second frontal convolution. He conceived of it as a center for memory of hand movements in writing. Dejerine called his attention to the fact that the center could not be merely for storage of such memories, because the patient who suffered a lesion there was also unable to write words with blocks, which could have nothing to do with hand movements. It seems that both were right. Engrams of hand movements are stored there, but association of these with the angular gyrus area is necessary to constitute a writing mechanism. It further seems that while this function is going on it is necessary for the angular gyrus to be in constant association with other parts of the cortex in order to keep grammar and rhetoric in order.

For figures it is also necessary for the region of the interparietal sulcus to be functionally associated.

A case showing the influence of the writing center on spelling is here recorded.

Laurence Gos. (case 82), aged 17, was struck on the head in an automobile accident while he was driving. He suffered a depressed fracture of the skull (Fig. 13) in the left frontal region above the site corresponding to Broca's area. When seen on the third day he had said nothing but "No" since the accident. There was a little of the semantic defect as seen in almost all cases, but aside from this he had practically perfect comprehension of the spoken and written word. From a set of colors he selected by request those found in the American flag. From the printed words, "house, bread, horse, wagon, onions, silver, blue, rice, and oranges," he was asked to select those which indicated things good to eat. Without a moment's hesitation he selected "bread, onions, rice, and oranges." By pantomime he indicated what he wished. He was asked to sing with his one word, "No," the national anthem. He did this well. He also was able to calculate. With this background we shall present his interesting writing disability.

Because of his injury his right hand was shaky; so he was provided with embossed letters on blocks and was asked to write something. He built up, "A boy fell down." He was then asked to write what had happened to him. The following was the result:

1. "A set . . ."	Dissatisfied, he tried again.
2. "A seteer . . ."	He looked over the word carefully, dissatisfied.
3. "A srteer . . ."	When asked, "Does it look right?", he said, "No," and corrected it again to
4. "A srteecar . . ."	Still dissatisfied he separated "car" to make
5. "A srtee car . . ."	Then he tried
6. "A steed car . . ."	Then
7. "A streed car . . ."	Then as he was showing signs of fatigue I suggested that he change the final "d" to "t." He did this and got
8. "A street car . . ."	This pleased him greatly. He brightened up and finished
9. "A street car hit me."	

I asked, "Were you walking?" He wrote, "Driving." Then he built up another sentence, "There were babies with use." (Only one small error.)

The striking element in the case is the loss of ability to spell due to a lesion of the frontal lobe. He used his intact parieto-occipital lobe to check on the correctness but could not visualize the word in advance. In his case we were not

concerned with loss of the cheirokinesthetic sense, because he was not writing with hand movements; he was building words with blocks. Yet, it is clear that his writing center in the frontal lobe was badly affected.

Here we recall the case of the author, who without any organic lesion was unable to spell on the typewriter although able to spell when writing. We must grant the boy a psychologic handicap as well as a focal lesion. With this thought in mind we may have to include much of the brain in the functional writing mechanism.

CHAPTER III

METHOD OF PRESENTATION

ONE OF the greatest difficulties encountered in the effort to bring some sort of order out of the chaos in agnosia, apraxia, and aphasia is that of nomenclature. Wilson (1926) points out the desirability of having an anatomic basis but at the same time the impossibility of this at the time of writing. A physiologic basis is also impractical. A psychologic basis is easy to obtain, especially as Head (1923) has already presented one, but a nomenclature based on psychology dissolves all connection between cerebral anatomy and pathology and the subjects to be classified. I believe the acceptance of such a nosology would be too destructive to justify itself. I have here adopted a terminology which is so far as possible based upon usage, which avoids the coinage of new elemental terms but attempts a systematization on the basis of physiologic (functional) hierarchies, or levels of integration.

To clarify this last expression somewhat, attention is directed to the obvious facts that, physiologically speaking, the first cerebral activity in the reception of an impulse is the function of primary perception. For vision the anatomic site of perception is on the borders of the calcarine fissure, for audition, in the first transverse gyri of Heschl. So far as language is concerned, the first stage of cerebral function takes place mainly at these two sites. This, then, is the first functional hierarchy.

If we continue to follow the paths physiologically, the next step is, for vision, the formation of engrams of symbols seen in the region of the left angular gyrus (cortex), for hearing, the formation of engrams of sounds heard in the region of the posterior superior left temporal convolution. This is the second functional hierarchy. So far, we have localization of cerebral function. Still speaking physiologically, above this, though there are higher hierarchies of function, there is very little, if any, localization known as yet. There are at least three hierarchies in the sensory sphere: perception, formation of engrams for recognition, and higher elaboration. This higher elaboration is not all on the same level, at least two levels being definitely discernible.

The motor sphere may at present be analyzed similarly but not so accurately. An analysis of the mechanism for handling of objects in their application to

useful purposes may to some degree also be attempted. This is the basis of approach I wish to utilize.

I shall have to disagree with some previous nomenclatures, and even with concepts, in forming a reasonable classification. This is unavoidable. In the appendix, with the presentation of each term selected, the definitions and synonyms are given.

CHAPTER IV

AGNOSIAS

AGNOSIAS are disturbances of recognition or identification. Since recognition is only a small function of the sensory sphere, agnosias represent a limited loss of function. This limitation cannot be determined unless general cerebral function is fairly well retained. This statement will be further elaborated.

The earliest idea embracing the concept here discussed arose with Kant, the German philosopher. Without exact anatomical or physiological studies he developed the concept that the mind had a special capacity to deal with symbols, and he called this the "facultas signatrix." Kant's authority made this concept acceptable to subsequent investigators, and the distinction between sensory and motor functions and between concept of objects and concept of symbols was not clearly made.

Meynert (1866) and Finkelnburg (1876) recognized that loss of this capacity of "facultas signatrix" occurred clinically without insanity, and they used the term "asymbolie" to indicate that state. This term was then used by Wernicke and subsequent prominent authors and investigators. In 1871 Steinthal created the term "asemie" to cover the loss of capacity to understand or express ideas by the use of language. This was less inclusive than asymbolia and in favor of specific expression. It should have been accepted, but it never replaced asymbolia.

In 1876 Hughlings Jackson introduced the term "imperception." In his case the patient had difficulty in recognition not only of symbols but objects as well. Unfortunately the writings of Jackson were not at the time recognized as the gems they were, and both the idea and the term remained in obscurity.

Thus matters stood until 1891 when Freud in a monograph on aphasia proposed the term "agnosie" to replace asymbolia, because, ". . . to me the relation between word and object-concept deserves the designation 'symbolic' rather than that between object and object-concept." This suggestion met with immediate acceptance in many circles, but certain authors continued to use the term asymbolia in the earlier sense. Making use of Freud's distinction but not of his term these authors then began to speak of "motor asymbolia" and

"sensory asymbolia," applying the latter to the concept covered by agnosia.

For years following the introduction of the term agnosia, an extensive study of the condition continued and many suggestions were made for additions to and modifications of the original concept of Freud. It became evident to all students of the subject that even simple identification of an object was a highly complex process, i.e., agnosia was subject to much subdivision. Some of the suggested subdivisions seem to me to have been expansions. At any rate some of the suggested terms seem to have been unwise. At this point the greatest need was for accurate definition, and we shall see how salutary these definitions were even though the recommendations were not always followed.

Definitions of the term agnosia elaborated since Freud's original terse statement are selected for presentation here from two sources of particularly wide acceptance. These were both published in the same year (1914), the first by von Monakow, the second by Dejerine. Von Monakow said, "One understands by the term 'agnosia' a lessening of the power to recognize or to differentiate by their characteristic features through a sense organ otherwise easily recognizable sensory impressions (especially through vision, hearing, and touch) in the presence of relative preservation or very slight disturbance of elementary function of the sense organ concerned as well as essentially preserved sensorium and speech." Dejerine defined it as follows: "Agnosia is a difficulty of recognition. Recognition is that psychological phenomenon which permits us by the use of one or the other of our senses to identify an object under observation with an object previously observed and of which we have registered the memory picture in the form of a mental image."

There are certain elements in these definitions which require stress. Von Monakow says, ". . . through a sense organ . . . ," and Dejerine says ". . . one or the other of our senses. . . ." It would seem unnecessary to point out that the failure of recognition must be confined to one sense organ (except in cases in which one is to consider the presence of two or three simultaneous agnosias). But it is necessary to emphasize that agnosia is for one sense only because of the introduction of the terms "ideational agnosia," "dissolutive agnosia," "disjunctive agnosia," and others, which have crept into the literature in spite of the fact that they deal with disturbances of recognition through several sense organs.[1] These terms have to do with secondary identification as

[1] The author does not deny the possibility of two simultaneous agnosias occurring in one case but this is rather uncommon. When visual agnosia is coincident with tactile agnosia, as has occurred in some cases, there is nearly always tactile agnosia on one side only so that identification is possible through the other hand. In cases in which the patient is unable to identify an object through hearing, vision, or touch, it is certainly difficult to establish that he has acquired the concept of the object by contact with it. If he has also failed to identify it through the senses of taste and smell, we certainly cannot demonstrate identification. If in such cases the examiner still claims mere agnosia and not general mental incapacity, it becomes incumbent upon him to demonstrate that the patient can obtain clear concepts.

the term is used by Wernicke and by Liepmann (*1908*) and not the primary identification on the level of which agnosia lies.[2]

I, therefore, insist that agnosia is a disturbance in recognition through one sense organ as von Monakow and Dejerine have stated. The second portion of the definition, which should be stressed, is from von Monakow's, that only the three senses of hearing, touch, and vision come into play. It has never been possible to distinguish between peripheral and central disturbances of the senses of taste and smell.

Still another element needs emphasis, namely, that in object agnosia we are dealing with difficulty of recognition of the object itself, not any symbol for it. If there is agnosia for the symbol also there are two agnosias and agnosia for symbols is a subdivision of aphasia. Thus the loss of recognition of an apple is in the sphere of agnosia. Similarly, the loss of recognition of a word is in the sphere of agnosia; the loss of recognition of its significance, however, is in the sphere of aphasia. A patient may thus be able to read a word without knowing its significance; he then has aphasia but not agnosia for it.[3]

Physiologic approach to the agnosias. In tracing the physiological pathways of a sensory impulse in the spheres of hearing, touch, and vision from the site of primary perception upward, one encounters certain anatomically known stations. There are many others which are unknown. However, those which are known, if clearly understood, place at our disposal much of value in cerebral localization.

The site of the first cortical termination of the impulse is, for vision, along the borders of the calcarine fissure; for hearing, in the first transverse gyri of Heschl; for touch, in a large area apparently including even portions of the precentral gyrus and the parietal cortex in general posteriorly. These are the sites of primary perception; at these points the impulse is received, but for vision and hearing, unless it goes farther, the individual does not even become conscious of it. The area to which the impulse must reach for consciousness of it to occur may be considered tentatively to be about the gray matter bordering upon the third ventricle. However, even when the impulse reaches consciousness, this fact does not establish memory of the experience; in order for memory to occur, another set of impulses must go to another area of cortex.

[2] Primary identification is the identification of an object through one sense organ only. Secondary identification is the completion of the concept through the added knowledge gained by higher associations and with impressions obtained by means of the other sense organs. Thus alcohol to the eye is a clear liquid only. After the substance has been tasted, felt, and smelled, it is alcohol. "Clear liquid" is the diagnosis through primary identification. "Alcohol" is final after the secondary identification has occurred.

[3] For the sake of clarity it may be necessary to point out that symbols are to the child first objects. There is no distinction to the child between the concepts, "This thing is a dog" and "This thing is an A." It is only after the *significance* of the symbol has been explained to the child that the symbol becomes a symbol. A symbol must be a symbol of something.

The area of cortex essential for recognition to occur is, for vision, the convex occipital and parieto-occipital cortex; for hearing, a portion of the first and possibly a portion of the second temporal convolutional cortex. For general sensation an exact separation of the two areas is not yet possible. It is fairly certain, however, that for hearing and vision there is a major and a minor side; for tactile sense there is no such differentiation.

When a memory of the impulse is thus created, the individual still has no knowledge of the significance of the impulses; he merely recognizes them. For interpretation of the significance, much of the cerebral cortex is necessary and, to be sure, is often inadequate.

We thus arrive at the generalization that one limited area is necessary for primary perception and another limited area for identification of the simplest sort, i.e., primary identification. Above that stage a diffuse area is required. This generalization is made with full knowledge of von Monakow's (1914) denial of such an arrangement. The cases gathered by the author show that only the second and third occipital convolutions of the major side are essential for visual recognition of objects.

Visual agnosia (mind blindness). This is a disturbance capable of a great many subdivisions, and it is indeed difficult to be dogmatic concerning what is to be included in the term. One distinction between the various types seems definite, namely, visual agnosia for nonsymbolic entities and visual agnosia for symbols. As a basis for the former, it is possible to distinguish certain fundamental factors which will be presented first.

The most fundamental (at least psychologically) of these disturbances is geometric-optic agnosia. In this difficulty the patient has lost sense of direction of lines so that he fails to recognize objects because of their distortion. Care must be taken to distinguish it from cerebral metamorphopsia in which condition the lesion affects the calcarine cortex, and the disturbance is not a true agnosia. In geometric-optic agnosia other mental disturbances are usually present. Pötzl and Henschen have each reported cases. Closely related to this condition is optical disorientation in space, the chief distinction being that in geometric-optic agnosia the patient considers relations of objects and lines to each other, while in optical disorientation in space he considers his own relation to the parts. Pötzl (1928) has carefully considered the localization of a lesion capable of causing these disturbances and finds essentially that the occipital convex cortex is chiefly concerned.

The next most fundamental of the visual agnosias is loss of recognition of objects themselves—visual agnosia for objects and pictures—first described by Hughlings Jackson (1876) as imperception but commonly accredited to Lissauer (1889) and hence called "the type of Lissauer." In this condition the

patient may be able to recognize symbols and hence read words or music while still unable to find his way about the house or the street, or to recognize the simplest object. The writer has examined carefully (to 1935) all reported cases of visual agnosia for objects with anatomic verification in which the lesion was confined to one side. The fact was clearly established that for the syndrome to develop there must be a lesion of the cortex of the major second and third occipital convolutions, or the fibers from both calcarine areas to the major second and third occipital convolutions must be destroyed.

Closely akin to visual agnosia for objects is the condition described by Pick (1902) as apperceptive blindness of the senile. Patients develop this difficulty during senility. It consists of lack of awareness of their surroundings and lack of ability to fixate objects with their gaze. In these cases there has been found (senile) atrophy of the occipital convex cortices, the calcarine areas being spared.

Acquired cerebral color blindness has been reported a number of times. With few exceptions loss of color perception occurs before loss of object perception in cerebral lesions affecting the visual fields (hemiachromatopsia). When the lesions are bilateral, there may be a bilateral loss of that character The physiology of these conditions is not understood.

With visual agnosia for objects it is common for another defect to appear, namely, loss of ability to name colors, the patient still being able to select colors properly. This belongs to the subject of aphasia, because the loss is one of symbolization, but it must be mentioned here because it forms with psychic blindness a syndrome known as the syndrome of psychic blindness plus optic aphasia for colors. After a careful analysis of the available material Pötzl concludes that a lesion of the transverse occipital fasciculus of Vialet is the basic anatomic lesion responsible for the syndrome.

Visual agnosia for various symbols is next to be considered. Perhaps the best known and simplest of comprehension among the disturbances of this type is subcortical visual verbal agnosia. This is the "cécité verbal pure" of Dejerine. In this condition the patient has lost power to recognize written symbols (figures relatively often escaping but musical symbols lost). There is no agraphia as in the cortical variety. Thus the patient can write but cannot read what he has just written. This condition is not at all rare; the present writer has seen 6 cases in three years. The causative lesion is below the cortex of the major angular gyrus, separating its cortex from both calcarine areas. Henschen (1922) has shown that when the lesion is slightly higher, about the interparietal sulcus, ability to read mathematical figures is lost—visual agnosia for mathematical figures. Visual agnosia for musical notes usually accompanies subcortical visual verbal agnosia.

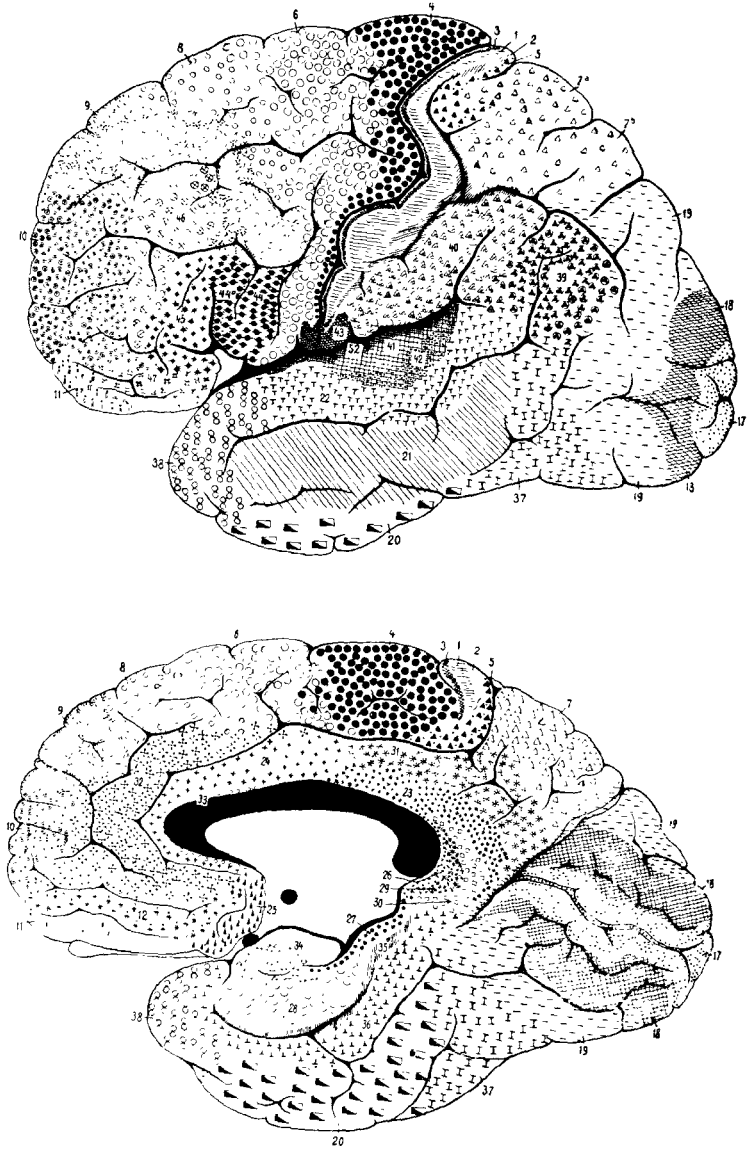

FIG. 15. Brodmann's illustrations of his cyto-architectonic areas of the brain.

Visual verbal agnosia is the cortical variety of verbal agnosia somewhat resembling the subcortical type. It resembles the subcortical type in that the patient cannot read, i.e., has the alexia of Wernicke (1874), but differs from it in that also he cannot write. The lesion may be cortical in the major angular gyrus but, inasmuch as the function of the angular gyrus may be crippled, usually for interpretation but occasionally also for recognition, by lesions separating it from Wernicke's area and even by lesions separating it from Broca's convolution, visual verbal agnosia alone does not determine the site of a lesion in a given case. The associated symptoms do determine it. If visual verbal agnosia is the only symptom present in the case a lesion subcortical to the angular gyrus must be present. There are subvarieties of this type, forms in which the patient can read letters but not words (verbal agnosia), letters or even words but not syllables (asyllabia), or words and not letters (literal agnosia). These subtypes probably depend more on the patient's early training than on the location of the lesion.

There are several conditions commonly described as visual agnosias but which correctly belong to the higher functions and will be considered later. These include revisualization of visual images, amnesic color blindness of Wilbrand, ideational agnosia, dissolutive agnosia, and disjunctive agnosia.

Acoustic or auditory agnosias. General acoustic agnosia without symbolic acoustic agnosia rarely occurs clinically. On the other hand, it is not at all rare for acoustic verbal agnosia to occur without general types, such as agnosia for sounds of automobiles, bells, whistles, crumpling of paper, etc. From the standpoint of cerebral localization there are two types of acoustic agnosia: acoustic verbal agnosia (cortical and subcortical) and acoustic musical agnosia.

"The word" or its equivalent should not, unless modified, form any part of a term used to define a function pertaining to one sense organ. A word is not heard, or seen, or visualized alone, but as Freud (1891) has clearly pointed out, it is composed of a kinesthetic written image, a visual image, a kinesthetic spoken image, and a sound image. (Kinetic might be a better term to use here.) Some words have many other images as well, all forming part of the *word*. In acoustic verbal agnosia the only portion of the word which is not recognized is the sound image. Bastian (1898) recognized all this but still considered the term "verbal" desirable.

The patient suffering with acoustic verbal agnosia is usually or frequently voluble and euphoric, the natural inhibition apparently having been removed.[4] Furthermore, as the patient cannot check up on his errors, and because the untrained inner language formulation area is the one which is

[4] The reason for the euphoria is that the patient is usually unconscious of his acoustic verbal agnosia, as Pick has shown.

functioning, he frequently uses a wrong or unintended word, and hence paraphasia and even jargon aphasia results. (Patients who can check on their accuracy and who constantly try to correct their speech may still be paraphasic.) Curiously enough, syntax and grammar are little disturbed, mostly diction. Like persons who are deaf, the patients suffering with word deafness will be satisfied with a portion of a sentence heard (the word deafness is rarely complete by reason of vicarious function of the right side) and will attempt to answer before the question is entirely grasped (paraphasia of comprehension: paragnosia). This is shown by asking two questions in succession which vary slightly. The same answer is given to both (Dejerine). There is usually associated word blindness, this making the picture of Wernicke's aphasia.

According to Mills (1898) several types of verbal deafness may be encountered: (1) The patient recognizes the voice as a voice but nothing more; (2) he recognizes language as such but does not understand what is said; and (3) (ascribed to Brissaud, 1894) he hears and can repeat but does not understand what he says. These depend on more or less complete capacity of the minor side.

Many cases are on record showing that patients with word deafness have a lesion in the middle third of the first temporal convolution on the major side. The area necessary has never been minutely determined. Starr (1909) presents diagrams of the cases of Girandeau, Wernicke, Eichhorst, Claus, Seppilli, and Hitzig which delineate the gross location clearly.

The syndrome known as pure word deafness, technically subcortical acoustic verbal agnosia,[5] is commonly described as a form of aphasia. Yet we are forced by our definition of agnosia to acknowledge that the exact difficulty with which we are dealing, that of inability of the patient to recognize spoken words, is agnosia.

Dejerine's description is here given. The patient cannot understand anything that is said to him, nor can he repeat the words nor write from dictation. Spontaneous speech is perfect. The lack of paraphasia is what distinguishes this form from cortical word deafness. Reading to oneself or aloud is normal. There is no disturbance of writing except that of writing from dictation.

Before ascribing deafness to a cerebral lesion one must of course determine that audition is present for the scale of tones or vibration rates upon which

[5] There are strong objections to both terms "subcortical" and "pure," although Wernicke accepted the term "pure." In reported cases (a good example of which is that of Dejerine and Serieux) the lesion was cortical and not subcortical. It is "pure" only in the sense that the internal language is intact. "Pure" does not mean that deafness to words is absolutely the only difficulty present in a given case.

speech depends. When this has been established we may proceed as follows with an analysis of the localization problem. The area of the cortex upon which knowledge of recognition of words depends is located close to but is not identical with the first transverse gyri of Heschl. Wernicke considered the area identical with that for audition. Wernicke's zone in the posterior half of the first and part of the second major temporal convolutions is concerned with the entire function of auditory recognition. The first transverse gyri of Heschl of both sides serve in hearing. There must then be fiber tracts from both auditory perception areas to Wernicke's center which functions correctly on the major side only. If the first transverse gyrus of Heschl of the major side is destroyed, the patient will still not have auditory agnosia, because the perception received on the minor side can still be referred to Wernicke's area and the remainder of the temporal lobe on the major side for interpretation. To produce the picture of subcortical acoustic verbal agnosia it is necessary to have interruption of the fiber tracts from both primary hearing centers to Wernicke's center on the major side or to have the major areas destroyed and have the defect produced by imperfect function of the minor side. Cases have been reported in which bilateral lesions of the temporal lobe caused the symptom, and these were so numerous that for a time it was thought that only bilateral lesions could cause it (cases of Pick, 1891; Dejerine and Sérieux, 1897; Pick, 1898; and Barrett, 1910). Liepmann and Storch (1902), however, reported a case in which the symptom was present due to a large subcortical lesion in the left temporal lobe. It is not difficult to comprehend how the compact bundles leaving the primary auditory center of the minor side can more easily be affected than the same fibers after they are spread out to traverse the corpus callosum in order to reach the other side. In the case of Dejerine and Sérieux there was found polioencephalitis affecting the primary auditory centers of both sides. This was then an interruption of the auditory fibers at their source. Briefly stated, the lesion or lesions causing pure verbal agnosia must interrupt fibers from both primary auditory areas to Wernicke's center on the major side.

Our knowledge of the localization of the lesion in acoustic musical agnosia depends practically entirely on the work of Henschen (1922). Feuchtwanger's excellent monograph (1930) on amusia was unfortunately mostly based on clinical studies.

Acoustic paragnosia is commonly seen clinically. It seems to result from lesions of the left temporal lobe whether or not Wernicke's center is exactly involved. My conception is that the major side is out of function and the defect results from imperfect function of the minor side. When patients have

acoustic verbal paragnosia, they mishear or hear imperfectly what is said very much as a deaf person does. Patients who have had surgical removal of the major temporal lobes are first-rate examples of paragnosia.

Tactile agnosia (astereognosis) was first described by Wernicke as "cere brale Tastlähmung." Many authors denied its existence, Dejerine, for example, taking the stand that there did not exist a special sense which could be called stereognosis. Dejerine also claimed that there was a disturbance of sensation in every case and that the defect found was due to that disturbance. Franz Kramer has shown how important it is to have the various types of sensibility intact before one can speak of astereognosis, but for a long time this point was not sufficiently emphasized. Kennedy as late as 1924 found it necessary to call attention to it again.

However, the matter is not settled so easily. It seems that Samuel D. Ingham is right when he claims that tactile discrimination (as determined by compass test) is practically always, if not absolutely always, impaired when astereognosis is present. The author has frequently verified this point. This would indicate that tactile discrimination is also a cortical function.

The author believes that sensations of touch, temperature, pain both superficial and deep, muscle and joint sense, and vibration sense must all be present before one can say that loss of ability to identify an object placed in the hand is due to astereognosis.

When astereognosis is present, the lesion is not thereby sharply localized. Bing (1927) places the cortical area chiefly responsible for stereognosis in the postrolandic gyrus, while von Monakow (1914) ascribes the function to a large area of cortex extending from the frontal to the posterior parietal region. As a rule the lesion in astereognosis is in the parietal lobe on the side opposite that of the affected hand. A subcortical lesion gives the same symptoms as a cortical one.

CHAPTER V

APRAXIAS

APRAXIA is a disturbance in which a patient without dementia, incoordination, or paralysis is nevertheless, because of a motor incapacity, unable to apply his powers to voluntary purposes. Henri Claude (1932) says that just as aphasia is a syndrome consisting of the divers disturbances of the complex function of language, so is apraxia a syndrome consisting of disturbance of the higher psychic functions. It is a disturbance of voluntary activity and consists of the impossibility of performing movements adapted to a purpose in spite of integrity of motility (Hollander). The apractic can often perform an act voluntarily for which he is apractic when the stimulus comes by command from another.

The term is commonly ascribed to Gogol (1873), but Heilbronner (1910) points out that Steinthal used the term in 1871. According to Heilbronner, Steinthal's definition included, ". . . not the movement itself of the limb is restricted but the relation of the movement to the object to be handled; the relation of the movement to the purpose is disturbed." However, Liepmann became the author of the modern concept of apraxia by his epoch-making work beginning with his publication of 1900. In spite of much excellent work by others (particularly von Monakow, 1914; Brun, 1922; and Sittig, 1931), the ideas of Liepmann have not been materially set aside.

The three types of apraxia described by Liepmann were:

1. Limb kinetic apraxia (gliedkinetische Apraxie).
2. Ideokinetic apraxia (ideo-kinetische Apraxie).
3. Ideational apraxia (ideatorische Apraxie).

Liepmann's concept was that the practic function of the right precentral gyrus was not subject to the person's will without guidance through the left precentral gyrus and that therefore a lesion of the major side caused apraxia of both sides. However, it now seems clear that the right side is merely not trained; after a lesion of the major side the minor one soon develops its function unless a progressive lesion is present. Liepmann's "sympathetic apraxie" must therefore be subject to a new interpretation.

Further, but in this Liepmann was correct, the region about the major

supramarginal gyrus is the area where sensory impulses are integrated, and hence impulses must pass through this area on their way to the physiologic center for movement of the hand. For this reason lesions near the major supramarginal gyrus may interrupt these impulses on their way to the precentral gyrus and thus cause apraxia in both hands. If the cortex in general is diseased so that ideation of motor function does not take place, ideational apraxia results.

The areas considered by Liepmann especially potent for the development of apraxia are: (1) the left parietal lobe, (2) the precentral gyrus, (3) the parieto-occipital region, and (4) the corpus callosum.

Later Brun (1922) from von Monakow's clinic presented a new aspect of the subject of apraxia. He divided the types as follows:

1. Unilateral motor apraxia. This was said always to be associated with crossed hemiplegia and motor agraphia, often combined with motor aphasia, without disturbance of sensibility in the apractic limbs.

2. Unilateral sensory apraxia. This was thought to be conditioned by simultaneous disturbances of the proprioceptive sensation of the higher order (as tactile agnosia) often associated with cheirokinesthetic agraphia and with sensory aphasia.

3. Bilateral motor apraxia. This was like the unilateral type except bilateral and associated with agnostic and ideational factors.

4. Bilateral agnostic-ideational apraxia. This consisted of three types not strictly separable clinically: (1) agnostic apraxia, (2) ideational apraxia, and (3) amnesic apraxia.

Correct as all this may be, it seems to the writer that the cerebral localization based upon it is of exceedingly little value. The localization as outlined by Brun himself offers very little. He finds that apraxia is often a transient disturbance and due to diaschisis. This is undeniable, but when such is the case, it is of no localizing value, and the writer is interested here in what is of localizing value.

Sittig, like von Monakow, has taken the healthy attitude of not being bound by the teaching of Liepmann to the extent of considering it practically dogma as most authors since Liepmann have done. He took into account all the literature to the date of writing (1931) and gave consideration to work of other authors, especially the French, particularly Foix who had proceeded on a purely empirical basis and had discovered valuable data. Critchley (1930) has added to our knowledge of apraxia by analyzing the symptoms of occlusion of various cerebral arteries or branches of them.

Sittig verified much of the work of Liepmann and made some additional contributions. First of all he verified the fact that lesions of the corpus

callosum produce apraxia. Further, he found a verification of some work of Mingazzini, that the anterior portion of the corpus callosum produced apraxia of the face area; farther back, apraxia of the upper limbs; and still farther back, apraxia of the lower extremities, but that the splenium played no part in apraxia. There is thus localization in the corpus callosum, i.e., certain fibers perform by reason of their connections certain functions. Sittig also called attention to the fact not formerly stressed, that apraxia of the trunk occurs, may even be isolated. If the lesion is large, the face is apt to be affected first (Sittig tries to base this on the principles enunciated in Jackson's laws which say that the areas representing greatest differentiation are most affected in a general lesion), next the upper extremities, next the lower, last the trunk.

Sittig further verified the fact which Liepmann had discovered, that lesions of the left supramarginal area produce apraxia with ideational elements. He differs from other authors in that he considers all apraxia bilateral. The hemiplegic weakness so often present is supposed to cover up the apraxia. He offers material to prove this.

The author agrees with Sittig except that it seems as though the subject of apraxia is rather crowded into the laws of Jackson. I have seen 3 cases of trunk apraxia. In the first, a man of 40 odd was unable to rise from his bed or lie down upon it. He raised his arms and legs into the air in an effort to rise but never succeeded in the attempt. Autopsy was not obtained. The second patient died of a large tumor of the left semiovale. Three months before he died he was unable to rise from a chair or seat himself upon it. He walked up to it, turned around and regarded it from all sides, made a few attempts to bend the knees, but was unable to figure out how to get himself into it. He voided and passed his feces wherever he might be, yet he walked about and understood what was said. In the third case no autopsy has as yet been obtained, but all symptoms point to the left angular gyrus. The patient has total alexia and agraphia and amnesic defects. He has no paralysis but is unable to slide down in his bed at will. He raises his pelvis in all sorts of clumsy ways and finally puts it down in the same place without succeeding in moving from place to place. He also has difficulty in drinking. He does not know how to place the bottle to his mouth. (This patient also died but autopsy was refused.)

Apraxia of the mouth (of the ideokinetic type) in the experience of the writer is the most commonly observed form. It seems to accompany nearly every case of Broca's aphasia. It is interesting that this form was discovered by Hughlings Jackson and published under the title, *Remarks on Nonprotrusion of the Tongue in Some Cases of Aphasia*, in 1878.

Kinetic apraxia of the limb is seen in practically all cases of neoplasm involving the precentral gyrus before onset of paralysis. The patient does not

have control of the individual fingers when the arm area is affected, even though he can use the hand as a whole. It is difficult indeed to distinguish between a paresis of the limb and early apraxia of this type. When the lesion is in the left cortex, there may be apraxia of the left hand, because the right motor cortex governing the left hand has been poorly trained.

Ideokinetic apraxia usually results from a large lesion of the left posterior parietal region or of the corpus callosum. In this form there is a functional interruption between an intact ideation and intact crude motor function. The patient is, therefore, unable to execute his ideas. When the apraxia is unilateral, the intact ideation is easily demonstrated; when bilateral, careful attention to the patient's behavior is necessary to distinguish this type from ideational apraxia. In ideokinetic apraxia the patient does something entirely different from what he intends. He may make a fist instead of putting his finger to his nose, may in dressing put his arms into his trouser legs, may try to write with a pair of scissors, etc. Simple acts may be executed without appreciable defect, but complex acts for which an elaborate ideational plan is required are sorely affected. The patient will often do of his own accord certain acts which he was totally unable to perform on request.

Ideational apraxia is characterized by correctness of the individual acts or elements of an act, but a failure to apply these elements to a purpose or to an ideational plan. The writer has verified that the most easily recognized characterization is that of extreme absent-mindedness. Examples which I have observed are as follows: A patient tried to light a safety match by stroking it on the sole of his bare foot. Patients strike a match to light a cigarette but forget what it is lit for and hold it between the fingers until it burns them. They seem to forget that it *can* burn. Patients have also been observed to carry food to the mouth without partaking of it. These acts do not have the unreasonable character of those in ideokinetic apraxia but give the impression that the patient has his mind elsewhere.

There are minor forms of apraxia or apparent apraxia, as Pick's pseudoapraxia due to perseveration, agnostic apraxia due to agnosia, and amnesic apraxia. The last named form is that in which the patient, while not able to carry out a request, is able to do it when shown. This is analogous to amnesic aphasia.

Amusia instrumentalis is a form of apraxia in which the use of musical instruments is especially involved. It does not differ materially from other forms of ideokinetic apraxia.

CHAPTER VI

APHASIAS

THE TERM aphasia was first applied (1861) by Trousseau to loss of speech due to a cerebral lesion exclusive of that due to paralysis of the muscles concerned with speech and exclusive of dementia. This replaced the aphemia of Broca which had previously displaced the alalia of Lordat. After aphasia had become almost universally accepted as the preferable term, other suggestions were made to render the terminology more definite and suitable. Thus McLane Hamilton (cited by Jackson, 1878) and Mills (1898) suggested the term asemasia (*meaning the inability to communicate by signs or language*), and Head made efforts to substitute the term dysphasia (disturbance of language). However, Jackson observed as early as 1878 that it was already too late to displace the term aphasia.

Definitions were formulated by Broca and by Wernicke, but this was before the breadth of the subject was realized, and hence they are not applicable at present. Kussmaul in 1885 stated that aphasia was no longer applied to speech disturbances alone but to the entire symptom complex of all disturbances of execution or understanding of signs of any sort by means of which man communicates his concepts and feelings to his fellows. Dejerine in 1914 defined aphasia as the loss of the memory of the signs by means of which civilized man exchanges his ideas with his peers. In both of these definitions it was understood that the loss mentioned was called aphasia only in the absence of dementia and if the lesion was due to disease of the cerebral hemispheres. Dysarthria was not considered aphasia. It will be observed that these definitions are broad indeed. They overlap the definition of apraxia and include some of the agnosias which are based on lesions at a far lower level (physiologically and psychologically) than are the disturbances involving the higher psychic functions or true aphasia.

In the development of the subject of aphasia the terms suggested have depended upon the leanings of the proponent. We thus have psychologic, physiologic, and anatomic terminology. At times also purely hypothetical schemes have been devised to facilitate the clinical study of aphasia, and the creators of these schemes in some instances have actually suggested terms based only

upon the diagrams. The literature is therefore burdened with many utterly useless terms, some of which it becomes our duty here at least to mention.

All efforts to base the study of aphasia purely on anatomic structures have failed because of insufficient knowledge. Physiology without anatomy to support it will always fail to provide localizing data as will a purely psychologic study. We must grant that in the present state of our knowledge of aphasia all three methods of approach must be used to solve the problem.

APHASIAS ON THE LOWER LEVEL

Aphasia is usually divided into two great categories, motor and sensory. This classification is still useful, although it is recognized that intermediate or mixed forms are far more numerous and common than purely motor or sensory ones. When Charcot first began to speak of motor aphasia, only Broca's aphasia was known. Ballet, a pupil of Charcot's, pointed out that this term was not the best, because motor agraphia was also motor aphasia. (Charcot called motor agraphia "aphasie de la main.") Under the heading of motor aphasia properly come aphasia of Broca, transcortical motor aphasia, subcortical motor aphasia, and probably motor agraphia. All these are really forms of apraxia, but since they apply to language, they are commonly classified as aphasias.

The aphasia of Broca represents a loss of more than one of the elements of speech; it is a syndrome. However, it is encountered clinically with sufficient frequency to merit a place in our classification. It is characterized by the loss of volitional emissive speech with relative retention of comprehension of language. There is usually a preservation of capacity to say a few words, as a rule the same ones over and over. (Hughlings Jackson has shown that what the patient repeats is usually the last word or group of words he spoke as he became aphasic.) While the patient has lost the capacity for voluntary speech, he still emits expletives and interjections, sometimes volubly. There is also associated with the loss of speech a loss of ability to write either spontaneously or on dictation, and to copy. The patient cannot read aloud but comprehends written language. In quite a number of cases there is a partial apraxia for use of the tongue and lips, the patient being unable to protrude the tongue on request, although protruding it reflexly or automatically with ease. The intellect is only slightly disturbed, at times hardly at all.

Hughlings Jackson's original idea (1878) that expletives and similar emotional expressions are produced by the right side of the brain has gained great support from the ideas of Moxon and the corroboration by Liepmann (1906) that the left side of the brain takes the lead and hence that the right may not be entirely passive in speech. The case of Zollinger makes this thesis practi-

cally certain. The writer at first felt that Jackson's views were not founded on sufficient data but now is convinced that his keen discernment led to the correct explanation of the fact that the patient with Broca's aphasia is "not wordless but speechless."

Two interesting case reports are presented here to illustrate classical cases of Broca's aphasia:

Karl Sch., aged 53, came to the hospital complaining of attacks of jerking of the right hand without loss of consciousness but with inability to speak during the attack. His story was so characteristic of a description of Broca's aphasia that it is here repeated in brief: "I am sort of a religious fanatic (he smiles) and have a little service every morning in my home. The first time the attack came on I was reading aloud from the Bible. Before the end of the sentence had been reached, I was suddenly unable to proceed. I had two kinds of trouble. One was that I could not read the next word or say anything I wanted to say. The other was that when I tried to talk, I could not avoid saying the last word I had just read in the Bible. This attack lasted half an hour and then I was all right again. In other attacks I have been talking, and then the same thing has happened; that is, I repeated the last word I had said. In some attacks I have not been reading or talking, and then I just didn't say anything. Then I could not answer at all when anyone talked to me. I have not tried to write my thoughts in these attacks, but I am sure that I can't write anything, because I don't know how to express my thoughts at all." The patient was not given to ejaculatory or interjectional speech, and such language was never heard during his aphasic periods. At operation by Dr. George Patterson an arachnoiditis was found over the cortex covering Broca's area and the motor cortex. The patient became progressively more aphasic and weak in the right hand. He also developed constant paragraphia.

In another case a woman physician, Dr. B., a victim of generalized arteriosclerosis and repeated cerebral thromboses plus pernicious anemia and diabetes, suffered two "strokes" both of which affected her speech. In one of these she was about to say that she wished horseradish at lunch time. She suddenly ceased talking but repeated "horseradish, horseradish," over and over again. She explained afterward that she knew how foolish it was, but it was the only thing she could say.

After a sleep she talked normally again.

These cases illustrate the points so carefully made by Jackson, that there are two elements in these speech errors in which the patient continues to repeat words: (1) the inability to say what he wishes, and (2) the compulsion to say what he does not wish to say.

The lesion in aphasia of Broca affects at least the cortex or subcortex of the

so-called convolution of Broca, at the foot of the third frontal convolution of the major (usually the left) side. This statement is made with the knowledge of the immense amount of work done to prove the contrary (Moutier's monograph alone is a volume of 774 pages devoted to this theme), but since the work of Henschen there can no longer be any doubt of the localization as has been stated. Proof is presented in Chapter IX of localization of motor engrams of speech in the convolution of Broca. One must keep clearly in mind that what the patient says after the major convolution of Broca is destroyed is said by means of the minor homologous area.

As to the nature of the lesion it should be said that neoplasms rarely destroy the area immediately affected but rather push aside the structures. For this reason the tissues continue to function in spite of a neoplasm at the spot, and one need not expect to apply one's knowledge of aphasia for purposes of cerebral localization in cases of tumor. There are a few exceptions. However, the usual lesion causing aphasia of Broca is vascular or traumatic. We have seen several cases of neoplasm in which Broca's aphasia was present in attacks, especially in epileptic attacks. The tumors were then under the frontal lobe (arising from the sphenoidal ridge) or in the tip of the temporal lobe pressing up against Broca's convolution.

Transcortical motor aphasia of Wernicke is characterized clinically by loss of spontaneous speech as severe as that seen in Broca's aphasia and by loss of ability to write spontaneously or on dictation, but with the peculiar retention of ability to repeat accurately anything that is said. Understanding of speech is only slightly impaired. The patient can copy mechanically and can read aloud.

There is no question of the frequent occurrence of this syndrome, but there has been much disagreement concerning the localization of the causative lesion. Many authors, among them von Monakow (1905), Dejerine (1914), and Freud (1891), have shown that the syndrome occurs in partial lesions of the cortex of Broca's area. If a lesion of the conducting fibers were the cause as Lichtheim and Wernicke believed, it would be necessary to assume an interruption of the pathway from the "ideation area or center" to Broca's area with the pathway from the auditory center to Broca's area intact. It would indeed be strange if such a lesion occurred as often as the syndrome is actually seen. Furthermore, the syndrome is encountered in the development of and during recovery from a Broca's aphasia. The author has several excellent cases on record studied clinically.

One of the most striking features is the accurate repetition of what the examiner requests. If he says, "Say 'Constantinople,'" the patient does not say, "Say 'Constantinople,'" but only, "Constantinople." If the examiner says,

"Can you say 'Yes'?", the patient says only, "Yes." In other words, the patient is "primed" but he selects the correct answer, does not slavishly repeat all he hears.[1] It will be shown subsequently that transcortical motor aphasia is due to lesions which in others would cause Broca's aphasia but that a good power of initiation of speech is present through stimulation, by the spoken word of the examiner, of Wernicke's area.

Subcortical motor aphasia is characterized clinically by the same incapacity of the patient to speak spontaneously, to repeat, to read aloud, and to sing as is seen in Broca's aphasia, but the inner language is undisturbed as shown by the practically faultless ability to gather information from written or printed matter and to understand spoken words, the ability to indicate in any word he wishes to use the number of letters or syllables (by various means), and the retained ability to write. His intonation may be absent entirely or be well retained.

The site of the lesion is indicated beautifully by the case of Ladame, widely quoted. (See the illustration in von Monakow, 1904, page 875.) A focus was found just lateral to the lateral ventricle below the F2 cortex. Posteriorly it involved the precentral gyrus. Cases much less definite even with cortical localization of the lesion are more numerous than such as the one cited.

Aphemia as "aphemie" was proposed by Broca (1861) and by him was used to designate what we now understand by Broca's aphasia. At the time there was known only the one type of aphasia. When the term aphemia was displaced by aphasia through the activity of Trousseau, aphemia gradually became the specific term for Broca's aphasia. Then Bastian (1898) of England suggested that the term aphemia be used restrictedly for pure or the so-called subcortical motor aphasia. Bastian spoke of aphasia due to lesion of the pyramidal tract. As a term to indicate motor speechlessness or pure word mutism it is still used, and this was greatly advanced by its application in this sense by Henschen (1922) in his great monograph.

Verbal aphasia (verbal defects of symbolic formulation and expression) was a term selected by Head (1923) as representing one of his four types of aphasia. It is characterized by paucity of words (in severe cases the patient can say only "yes" and "no") and dysarthria, but the patient uses all words at his command correctly. After the initial stage of shock has passed, comprehension of verbal significance is rapidly restored. As the spoken vocabulary increases, the power of writing is regained though it tends to show the same defects as articulatory speech. Simple arithmetical operations can be carried out cor-

[1] While the writer considers aphasia of Broca and subcortical motor aphasia types of apraxia, it is probable that transcortical motor aphasia is not apraxia but true aphasia, i.e., the disability is due to impaired initiation and is not purely motor in character as is true apraxia.

rectly even in severe cases. Nominal value and significance of words are perfect.

This description is taken largely verbatim from Head. The student of aphasia will recognize immediately that the description fits quite accurately Broca's aphasia.

Avocalia is the loss of capacity for vocal singing due to a cerebral lesion in absence of paralysis or incoordination of the vocal organs. This term was suggested by Henschen (1922). One must note that the term does not mean the inability to vocalize. We rely mostly on the work of Henschen for an analysis of the cases studied clinically for avocalia and later subjected to autopsy. The cases do not all agree, but after due discounting Henschen comes to the conclusion that avocalia results in most cases when the lesion affects the left pars triangularis of the third major frontal convolution.

Aphasia of intonation was described by Brissaud in 1895 (cited by Dejerine in 1914 and by Mills in 1898) as a disturbance of language characterized by loss of the power of intonation. Dejerine, while recognizing the fact that intonation is lost at times in motor aphasia, did not consider this a separate type of aphasia. Wilson (1926) thinks intonation is rather emotional than intellectual. Mills (1898) says that intonation is a form of singing. Aphasia of intonation is then closely related to Henschen's "avokalie." Henschen (1922) found in the literature 19 cases in which the patient was able to sing, not only with a single syllable, in Broca's aphasia, but able to sing words. After a careful analysis of the material he comes to the important conclusion that the motor singing center is located in the pars triangularis of the third left frontal convolution, i.e., just anterior to Broca's area. This, if the interpretation is correct, explains why intonation is often lost in Broca's aphasia. The matter, however, is not absolutely settled.

The term atactic aphasia as ataxic aphasia was ascribed by Gowers (1888) to Sanders (*Edinburgh Medical Journal*, March, 1866). Sanders used it because of the frequency with which words are wrongly formed in old or partial motor aphasia. The term is hardly used at present, Broca's aphasia being the common appellation.

Sensory aphasia is far more difficult to present and to define accurately than is motor aphasia. This is because what Wernicke originally described as sensory aphasia has been subdivided into elements which are at present not all considered aphasia and because much has been added. A brief historical account will help to clarify the situation. For this account I shall draw upon the excellent outline given by Bonvicini (1929).

From a reference given by Bernard it seems that Johannis Schmidi in 1673 gave a clinical description of a case of paraphasia and alexia without agraphia.

(It is now known that such a syndrome results from a lesion of the area of Wernicke.) In 1742 van Swieten described amnesic aphasia clearly. Also Spalding in Berlin (1872) recorded a case of alexia and another was reported by Rostan in 1823. Lordat in 1843 described his own condition of aphasia beginning in 1828. After his recovery he gave an excellent account of it, explaining that he had understood little of what was said, because he could not grasp the meaning and conversation came too fast for him (characteristic of the function of the minor temporal lobe). He knew words but could not use them properly because he had lost syntax. He was conscious of his paraphasia but again spoke the wrong word in attempting to correct his error. It is thus clear that sensory aphasia was understood as a clinical entity long before Wernicke.

Anatomically the difference between motor and sensory speech was understood by Wyllie. In 1866 (eight years before Wernicke) he said that ideas of associated motion which form the faculties of speech are supramotory, while the situation of the ideas of associated sensations which form the faculty of word comprehension is suprasensory.

However, it was left to Wernicke in 1874 (and he had delivered lectures on the subject the year before) to present the entire subject in his now famous monograph with presentation of cases, and this he did so ably that his ideas were immediately accepted. Wernicke's aphasia consisted essentially in loss of comprehension of spoken language and of written or printed matter, inability to write, and paraphasia. In present day terminology this is stated as acoustic (or auditory) verbal agnosia, visual verbal agnosia (with alexia), agraphia, and paraphasia. It is thus a syndrome, and as these elements may vary considerably, cases of Wernicke's aphasia are by no means all alike. When Kussmaul's important monograph appeared in 1885, the author suggested an essential change in Wernicke's terminology, substituting "Worttaubheit" (word deafness) for "kortikale sensorische Aphasie." Wernicke always opposed this change; the fact is mentioned to show that the heterogeneity of Wernicke's aphasia was already recognized.

When the entire syndrome of Wernicke's aphasia is present, the patient is usually voluble and somewhat euphoric. He continues to talk even though he may recognize many errors in his speech, and he is usually not so disturbed by consciousness of errors (he is frequently unconscious of them) as is the patient with predominantly motor aphasia who is paraphasic. The volubility seems to be due more to an inability to cease rather than a press of activity (Heilbronner, 1910). The paraphasia is peculiarly characterized by disturbance in diction rather than in grammar or syntax. This is especially true when predominantly sensory; if predominantly motor (then not distinctly

sensory aphasia) the *word* itself is commonly affected. Thus, "I have been here just exactly a couple of thousand years" and "There is so much of it that and then there is so many things to do and so much to do to tell it that I can't say it" are characteristic of the sensory type. And, "I ca seck the thock" for "I can say the thought" is characteristic of the motor type.

When the paraphasia is so bad that one can hardly understand the patient, it is called jargon aphasia or choreatic paraphasia, the latter being Kussmaul's term. Head called this form syntactical aphasia. The term agrammatism is commonly used, and Pick used the term "Kataphasie" to cover disturbances of syntax, grammar, etc., as being more inclusive than agrammatism. It will be shown in Chapter X that paraphasia results from the crippling of the major language formulation area and the use of the homologous area of the minor side.

The term transcortical sensory aphasia is applied to the peculiarity in which the patient is able to repeat whatever he hears but without comprehension. He also reads aloud without comprehension. A somewhat similar but nevertheless different condition is echolalia, a condition in which the patient repeats whatever he hears, sometimes with a decadence until he finally ceases when his speech is hardly audible. Echolalia is due to destruction of the major language area and good repetitive capacity of the minor side.

Wernicke thought that paraphasia was due to a disturbance of communication between motor and sensory centers and called it a "Leitungsaphasie" (conduction aphasia). This concept has proved entirely too narrow as paraphasia may be due to a variety of lesions. It is now known to be due to the activity of the right language formulation area. It occurs after complete removal of the major temporal lobe. The other essential elements of Wernicke's aphasia (acoustic verbal agnosia, alexia, and agraphia) need separate discussion. Acoustic verbal agnosia has already been explained under the agnosias. Alexia and agraphia require rather longer accounts and are given below.

Another element of aphasia which is sometimes prominent in Wernicke's aphasia is amnesic aphasia.

Amnesic aphasia, according to the experience of the writer, is met most commonly in lesions of the temporal lobe. It was a striking symptom in the author's case of G.O.R. (Chapter X) in which the left temporal lobe was completely removed. Von Monakow (1905), however, says it can accompany any type of aphasia. Mills (1898) states the term has been applied to disorders of speech from lesions on the receptive or sensory side of the brain, and this is in harmony with the impression of the writer. It is the typical aphasic manifestation in otogenous abscess of the temporal lobe. Bianchi included it in his "parietal symptom." (See also Chapter IX and the *Bulletin of the Los Angeles Neurological Society*, March, 1940, page 78).

The clinical picture is easy to recognize, although it naturally varies somewhat. In the mildest form the patient merely fails to recall proper names (anomia). Usually he fails to recall nouns in general and resorts to circumlocutions. A pen is "something to write with;" a chair is "what you sit on;" a book is "what you read out of," etc. It is always clear that he comprehends what is said and recognizes objects; he merely is unable to name them. In severe cases he also fails to recall verbs, adjectives, and possibly all parts of speech. But in all cases he immediately recognizes the word when he hears it (and is thankful for the suggestion).[2]

There is an amnesic aphasia affecting visual memories alone which, because it was discovered separately for several elements, has received different names for its various applications. To the syndrome of loss of ability to name an object until it has been perceived by some other sense besides vision Freund (1889) applied the name of optic aphasia. The loss of ability to name colors while still able to recognize them (the same as for objects) has therefore been named optic aphasia for colors but is also known as amnesic color-blindness of Wilbrand. The same condition occurs relative to hearing alone, as when one is able to recognize a tune but unable to name it. This has been called amnesic amusia. The same condition also occurs relative to sensation, inability to name objects felt until they are also seen or heard (astereognosis or tactile agnosia). Amnesic apraxia is similar and is the inability to carry out a movement until it has been seen.

The question of the location of the lesion responsible for amnesic aphasia seems to have been answered in a negative way by a large volume of material anatomically examined with lesions in all possible sites but mostly behind the rolandic fissure. Kehrer (1913) concluded that not any particular location for a lesion to cause amnesic aphasia existed. Von Monakow (1905) gave his opinion as follows: "Amnesic aphasia can appear as a general symptom in senile involution and as a portion of the picture in severe debilitating nervous affections or in dementia based on an organic lesion. It can also appear as a focal and relatively isolated symptom (without simultaneous aphasic disturbances). In the last case it appears as a relatively early symptom in disease of the deep white matter in the left parietotemporal lobe. It is also not rare in deep lesions of the angular gyrus." Mills, on the basis of a case with a localized lesion in which autopsy was obtained, concluded that a focal lesion mainly of the left third temporal convolution affected the "naming center." I do not

[2] It has been suggested that amnesic aphasia is merely a form of transcortical motor aphasia. True, in both the patient receives the forgotten word either by sight or sound and then repeats it. But there is this important difference, that the patient with transcortical motor aphasia is not looking for the word when he is told to say it. He is ordered to say it, and the concept is not in his mind before. In amnesic aphasia the patient has the concept, wants the word, in fact a certain word, and knows when the correct one is offered.

believe that this proves the point but believe that it results from a lesion of the language formulation area or of the temporal isthmus.

Simple introspection will throw considerable light on the nature and the probable sites of lesions in amnesic aphasia. Amnesic aphasia is aphasia of recall, i.e., the individual has the concept for which he wishes the *word*. It is well known that in some instances one recalls a certain word by recalling its sounds; this is particularly true in rhyming. On the other hand some words can be recalled only through association with visual memories because the sounds of them do not distinguish them from each other. In still other instances the memory of the movements made by the vocal organs during the pronunciation of the word recalls it. Now, one may forget how the word sounds, how it looks, how the vocal organs must be placed to reproduce the sound, and this element consists of two parts: the memory of the motor pattern and the memory of the pattern of the deep sensibility during the making of the movement, i.e., the memory of the proprioceptive impressions. It is therefore conceivable that amnesic aphasia may result from lesions of the temporal, parietal, and frontal lobes. I have analyzed a group of cases gathered from the literature in which amnesic aphasia resulted from lesions of the parietal lobes. In every instance it impinged upon the temporal isthmus. Amnesic aphasia resulting from frontal lesions is extremely rare; if it does occur it must be from interruption of associations between the temporal and frontal areas.

No matter where the ramifications of the discussion may go it is clear that amnesic aphasia is a disturbance in the patterns of language formulation. Disturbance of language formulation results from lesions of the posterior temporal region not involving the area of Wernicke. However, whenever language cannot be formulated without at the moment having association with the frontal speech zone a lesion of that zone might conceivably produce amnesic aphasia.

APHASIAS ON THE HIGHER LEVEL

While this division of aphasias into those on a lower level and those on a higher level has not to my knowledge been done before it seems to me particularly appropriate. Reflection will show that, while agnosias are disturbances of recognition, all of the aphasias so far described are disturbances of recall, or to use a better term, of reminiscence. The fundamental difference between recognition and reminiscence is that whenever one recognizes an object or a symbol one has the object or symbol before one; one receives a visual, auditory, tactile, gustatory, or olfactory impression of it and this impression is compared with engrams previously formed by similar impressions. If the two are essentially identical the object or symbol is recognized. The crux of the

matter is that one starts with the object or the symbol and looks for its meaning. But in reminiscence one starts with the idea or concept and searches one's cortical engrams of memory for the corresponding object engram or the symbol engram. The two processes are reversed.

The same process (of starting with the concept) applies to motor speech: one starts with an idea and looks for the motor pattern with which to express the concept. The motor pattern is found through the medium of the language formulation area in which the synthesis of concept into speech symbols is carried out and from which the motor patterns are stimulated.

There is a still higher hierarchy of language, however, higher than that of reminiscence. This is the hierarchy of semantics and calculation. Head's most valuable contribution to the subject of aphasia was the introduction of the term semantic aphasia. By it he meant one's inability to determine the full significance of speech though able to comprehend spoken or written language to some extent. In the study of cerebral localization in aphasia it is useful to draw a rather sharp line between recognition and comprehension because there are all degrees of comprehension. A physician comprehends much more of a medical article than does an attorney, but the reverse is true of a legal document. It is questionable whether anyone ever gathers the full significance of what an author writes and we must thus all have semantic aphasia if we use the old terminology. If, however, we draw the line between recognition of a word and its significance we have a valuable distinction. Loss of the first is agnosia, of the second, aphasia. Significance is still not reminiscence; it is a higher function because one can recall a word of which the significance is unknown.

Calculation is also a function on a higher plane than mere recall. In calculation one must not only recall all the symbols necessary but must keep in mind the results of conceived mathematical processes. Henschen studied calculation and called a disturbance in that sphere acalculia. The patient with acalculia may be able to read numerals but be unable to calculate within his former ability. In general a lesion to cause acalculia must be located in the angular gyrus-occipital region.

C H A P T E R V I I

MISCELLANEOUS TERMS

MISCELLANEOUS terms not classifiable according to the scheme used here include the disjunctive, dissolutive, and ideational agnosias of Liepmann. These are disturbances of secondary identification and not of primary identification as are the true agnosias. They cannot be called aphasias, because they are not concerned with language. They are really psychologic difficulties on a high level of integration. They are described in their proper alphabetical places in the Appendix.

Psychic paralysis of gaze. (Seelenlähmung des Schauens of Bálint). Up to 1928 Pötzl, reporting in his excellent monograph, had not been able to find a case to parallel the original one of Bálint's. Bálint described the syndrome in his case as follows: "The patient read from a test chart only the portion in the upper right quadrant. It was found on examination that he was not blind in the rest of the field but merely did not observe. When his attention was called to the other portions, he read them well. He merely failed of his own accord to observe the three quadrants (not strictly quadrants but roughly so). This condition remained for a time, but then other developments appeared, and the final clinical picture was, of course, the one to represent the autopsy findings which showed softening in both parieto-occipital areas as well as a small area in the left fronto-parietal regions." One must conclude that such a clinical picture may result from lesions in both parieto-occipital regions. Pötzl (1928) has observed part of this clinical picture in a case shown by autopsy to be one of Alzheimer's disease. The syndrome corresponds to Riddoch's syndrome of "loss of attention in homonymous half-fields."

Charcot-Wilbrand syndrome (syndrome of visual agnosia plus loss of ability to revisualize images). The syndrome consists essentially in the loss of the faculty of portraying in the mind's eye familiar surroundings. This carries with it the inability to draw from memory or to describe scenes formerly familiar. This is clearly an aphasic, not an agnostic, disturbance when symbols are concerned.

While the syndrome is named the Charcot-Wilbrand syndrome, Hughlings Jackson (1876) described a case (but as one of imperception) seven years be-

fore Charcot. This patient's loss was not complete. Charcot's (1887) patient was exceptionally well educated. He knew five languages well and was also an artist. He had a habit of sketching scenes which attracted him on his numerous travels. After an episode he lost the ability to draw scenes formerly familiar, and his home town of Vienna was entirely strange to him upon his return. He had also lost the ability to speak French except by translating first from German or Spanish.

In Wilbrand's (1887) case after a vascular insult a woman of 63 had a field defect and thought she was blind as did her friends. However, she was able to read fluently and to write from dictation. She was unable to find her way about her own house. Autopsy showed bilateral occipital lesions. Pötzl has reported a somewhat similar case.

After a careful analysis of the material available Pötzl (1928) believes the lesion in these cases to affect of necessity the inferior occipital surfaces and part of the convexity, a portion of the area supplied by the posterior cerebral artery.

Another term for miscellaneous classification is *simultanagnosia of Wolpert.* This is not an agnosia but a psychologic loss on a high plane. It is described as loss of power to visualize action as depicted in still pictures. In severe cases the patient cannot understand cinematography though he knows the significance of the elements of each picture of the film. Two cases on record with autopsy showed lesions of the left occipital lobe.

CHAPTER VIII

THE BODY SCHEME

BODY SCHEME (Head) and body image (Schilder) are terms used to designate the concept which a person has of his own body, its parts, and the relation of the parts to each other. The body scheme is apparently represented in the cortex of the brain by a set of engrams on each side of the brain, but not similarly placed on the two sides. These may be disturbed in such a way that the patient loses the scheme of his own body only (the lesion then being on the minor side) or that he loses all concept of bodies to include those of others as well (the lesion then being on the major side). The engrams of the body scheme may be separated from the engrams of language formulation leaving the patient with intact body scheme but unable to formulate language which involves it. Such a condition results in finger aphasia and related syndromes. The engrams of the body scheme may be separated from the visual recognition sphere leaving the patient cognizant of his body scheme by the sense of touch but unable to recognize the body or its parts through the sense of vision only. The result is visual autotopagnosia and related syndromes.

A subdivision not attempted before (as far as the writer knows) with reference to the body scheme is disturbances of one's concept of the anatomy and similar disturbances of the physiology. There is a fundamental difference between them. In disturbances of anatomic concepts the patient forgets parts of the body or he has illusions or delusions that the body is no longer normal; in disturbances referable to the physiology the patient has delusions that the physiology remains normal after it is actually disordered. An example of disturbed anatomic concepts is the belief that the left limbs no longer exist; an example of disturbed physiologic concepts is the belief that the left limbs are not paralyzed when they actually are.

The body scheme is somehow related to handedness and language; it is on the basis of handedness and language that the major and minor sides are differentiated and the disturbances of body scheme are readily separable into those of the major and those of the minor sides.

ANATOMIC BODY SCHEME AND ITS DISTURBANCES

1. Minor side.

From the material available it seems justifiable to conclude that there exists a set of engrams in or about the minor supramarginal gyrus which is concerned with what the individual believes about his minor (opposite) limbs. In rare cases (in about the same proportion as the occurrence of aphasia from a lesion on the right side of the brain in a right-handed person) disturbances of the anatomic body scheme result from a lesion similarly placed on the major side of the brain.

In an attempted analysis of the physiologic basis for delusion of absence of the minor limbs the first thought is that, as the symptom is known to result from a lesion of the thalamoparietal peduncle, the delusion is based on loss of sensation of the limbs. The following interesting case, however, which was reported by the author in 1938 shows that such a concept must be revised. A Mexican laborer had completely lost knowledge of the position and even of contact of the left upper limb. He could not locate it with the right hand and could not tell whether it was touched by the examiner, yet he had no illusion or delusion that the limb was absent. The engrams representing the body scheme must, therefore, be different from those of the cortical engrams of sensation. Just how they differ in location is not known.

The usual manifestations of such disturbances are as follows:

The patient may have amnesia for the minor (usually left) limbs, more frequently the upper limb than both upper and lower. In such a case the patient, a dentist, simply forgot the limbs periodically. He bathed the right side of the body and dressed the right side only. When his wife called his attention to the discrepancy he recalled that he had left limbs and took care of them. During the process of bathing and dressing he used the left limbs correctly and without apraxia but in such use he was not conscious of using them; he gave them no attention. In such cases of periodic amnesia the minor side of the body is merely lost from the attention. When his attention is called to the limbs by someone else he accepts them and is amused that he could have forgotten them. Their loss from his consciousness or awareness is a much less severe disturbance than delusion of their absence, to be described shortly.

As the case of the dentist progressed he became more and more definitely amnesic for his left limbs; it seemed that he would have had permanent amnesia for them if surgical intervention had not been decided upon. At operation a spongioblastoma was found in the right inferior parietal region. In another case reported by the author and Sult the patient had permanent amnesia for the left limbs.

Barkman has described the same syndrome and it has aptly been suggested that such a permanent loss of the limbs from consciousness is like a hemianopia of which the patient is unaware. It differs from *hémianopsie noire* in which the blind visual field seems to be black and its blindness is forced on the attention of the patient.

The next more severe disturbances of the anatomic body scheme referable to the minor limbs is a positive sensation of absence of the limbs—an illusion of their absence. Such an illusion corresponds in our simile with hémianopsie noire. In such cases the patients have thrown much light on the psychologic reactions and attitudes. A patient of Dr. Clarence W. Olsen claimed that her left limbs belonged to someone else. When her right hand was placed on her left shoulder and it was made to follow the arm down to the hand she was confronted with a choice between two sources of information, vision and sensation. She then said, "But my eyes and my feelings don't agree, and I *must* believe my feelings. I know they look like mine, but I can feel they are not, and I can't believe my eyes." Another patient said that as she sat in a theater her left hand suddenly slipped into her lap and she had a feeling that it was detached and did not belong to her. She traced it, by means of her right hand, to her shoulder but could not shake the conviction that it was absent. She said, "It was the eeriest feeling." That sensation disappeared on the following day.

The decision of the patient to accept her "feelings" in preference to her "eyes" recalls the statement of Flechsig made as early as 1886. He pointed out that one becomes aware of one's bodily contour and limitations through sensory perception before one forms a visual image of them. Throughout life the cortex of general sensation rules over that of visual perception and recognition as an absolute monarch and the monarchy never resigns in favor of a republic. Practicing physicians know how difficult it is to convince a neurasthenic patient that the perceived sensations do not prove serious disease. The patient insists that his bowels are diseased in spite of roentgenologic and exploratory findings to the contrary because he can still *feel* that they are ill.

Patients who have the positive sensation (illusion) of absence of the limbs usually show an associated psychologic defect, namely, the tendency to explain away the difficulty even with elaborate confabulation. One patient upon feeling her own left elbow with her right hand said, "That's someone else's knee." When she similarly felt her lower limb and her left hand she said, "That's an old man. He stays in bed all the time. I don't want any spirits in bed with me. That's my brother-in-law's hand." (Her brother-in-law had died.) On another occasion she said, "That's some old meat. That's part of my brother-in-law's arm." When it was shown to her by the tactile route, letting her own

right hand follow the left hand to her shoulder, she became convinced that it belonged to her. She then also acknowledged the foot and said, "My old, rusty foot."

If the illusion continues for days or weeks, and occasionally even after only a few hours, some patients develop a delusion of the absence of the limbs. They become thoroughly convinced that the limbs are absent and a great deal of evidence must be presented before the conviction disappears. In some instances the delusion has become so firmly fixed that it must be classified as an insane delusion. Such patients accept the positive feeling of the absence of the limbs in preference to a positive feeling through palpation with the unaffected hand and in spite of visual evidence to the contrary. A colored patient will even claim that her black hand belongs to the white doctor. This must mean that the positive proprioceptive sensation which the patient receives in the right parietal cortex and which he interprets as absence of the limbs carries a greater conviction than the exteroceptive tactile sensation through the right hand to the contrary.

It is of importance that not all patients develop amnesia for or delusion of absence of the limbs from lesions similarly placed. The psychic makeup of the patient plays a role; some patients will insist on facts and will accept no substitute explanations.

In nearly all cases of this sort the patient recovers in time but the writer has seen one case in which the delusion persisted until death five years later.

The site of the lesion in cases of amnesia for the limbs and in cases of illusion or delusion of their absence has been grossly the same in all cases—the thalamoparietal peduncle. Either the same lesion is extended to cause the hemiplegia or there are two lesions if hemiplegia is present. In one case only, so far reported, the lesion was localized in the supramarginal gyrus. If it is safe to generalize on the basis of this material we can conclude that the thalamo-supramarginal fibers are the ones which normally convey the impulses of presence of the limbs. However, even bilateral cortical lesions of the supramarginal gyri do not produce lasting disturbance of the body scheme. Therefore, we cannot postulate that the engrams of the body scheme on the minor side of the brain are found in the cortex of the supramarginal gyri. Exactly where they are is not known; they are possibly not sharply localized.

2. *Major side.*

It has been pointed out that disturbances of the anatomic body scheme from a lesion of the minor side of the brain affect the patient's ideas of the minor limbs only. A series of disturbances of a similar, but rarely identical, nature may result from lesions of the major side. Aside from the rare instances in

which delusions of absence of the major limbs result from a lesion of the major side, all disturbances of the anatomic body scheme caused by a lesion of the major side influence the patient's conception of both sides of the body. From this we deduce that the minor side of the brain which has its own engrams of body scheme "reports to the higher authority" of the major side and that the major side of the brain has engrams representing the body scheme as a whole.

A complete disturbance of the body scheme resulting from a lesion of the major side is rare. Such a condition was described by Pick as autotopagnosia but should have been called visual autotopagnosia. It is a more limited disturbance than visual agnosia for animate objects in that it affects the patient's own body only and not all animate objects.

In visual autotopagnosia the patient is unable to recognize his own body or any part of it by means of vision alone. He cannot recognize his hand, his foot, or even his own artificial teeth, yet he recognizes all inanimate objects. In severe cases he also fails to recognize the bodies of others or even of animals. In the only case on record in which the test could be made the patient classified flowers with animate objects.

Cases of incomplete visual autotopagnosia are, as one should expect, more numerous than complete ones. Visual finger agnosia described by Gerstmann as part of his syndrome is the most common fractional autotopagnosia. This is to be expected as, in harmony with the laws of Hughlings Jackson, the most highly differentiated and evolved part of the body has the greatest extent of cortical representation. However, Isakower and Schilder reported a case in which the patient looked on the floor for her eye and other instances have been reported in which the patient visualized a body as being without a neck or with the head in the abdomen.

In Gerstmann's syndrome of "finger agnosia, confusion of right and left, acalculia and agraphia" the visual finger agnosia most frequently affects the three middle fingers of both hands. In more severe cases the patients do not recognize any of the fingers and he does not know which is right and which is left in his own body or in the bodies of others. I have studied a case of amnesia for the fingers. In many cases of finger agnosia the patients also have apractic loss of finger demonstration and constructive apraxia.

If the cortical area concerned with visual finger recognition is dissociated (by an anatomic lesion) from the language formulation area the patient develops finger aphasia. Finger aphasia may be present without visual finger agnosia. It is becoming clear that Gerstmann's syndrome should be subdivided into its elements and the elements should be studied separately. The entire syndrome contains visual agnostic, apractic, revisualization, and other defects.

So far as the body scheme is concerned we can outline and subdivide the clinical manifestations of major-sided anatomic disturbances as follows:

I. Visual agnosia for animate objects.
   1. Generalized visual autotopagnosia.
      A. Visual agnosia for laterality.
      B. Visual agnosia for fingers.

II. Loss of revisualization of animate objects.
   1. Loss of revisualization of one's own body.
      A. Loss of revisualization of laterality.
      B. Loss of revisualization of fingers.

III. Aphasia (inability to recognize names of or to name on sight) for the body or any one of its parts.

In the first of the three groups we are dealing with genuine agnosias—loss of recognition through one sense organ only. The disturbances are on the plane of the second level in the visual sphere. On the basis of what is known of cortical localization in visual agnosias one should expect to find the lesions in such cases in area 18 of Brodmann. A little modification of such a concept seems necessary in view of certain autopsy findings which show that the engrams for representation of the body scheme are somewhat diffused in the occipital lobe.

In the second group of the three we are dealing with disturbance of revisualization (reminiscence) relative to the body scheme. That function is on the next higher plane of cerebral function above recognition. From what is known in general about cortical localization in cases of disturbed revisualization one should expect the lesion in such cases to be in area 19 of Brodmann. This concept is generally verified at autopsy but, as will be outlined, there is a little evidence that one occipital lobe may be the major for animate, the other for inanimate objects.

The third group consists of disturbances in the sphere of language and are therefore aphasias. They are chiefly disturbances of naming or of comprehension of the names of parts of the body. Amnesic aphasia for objects and persons (the common anomia) when on a functional basis may possibly be due to trouble in the engrams between the occipital lobe and the language formulation area.

Our knowledge of the anatomic sites of the lesions in these various clinical manifestations has been extended during the last few years. So far as is known only two cases of generalized visual autotopagnosia have been reported, one by the author and Ives (1937), the other by the author and Sanborn (1942). In the first there was a vascular softening in a vertical strip about 1 cm. wide in the left occipital lobe. In the second the cortex only of the left occipital lobe

was destroyed by asphyxia and cerebral anoxia. Inasmuch as generalized visual autotopagnosia is almost identical with visual agnosia for animate objects another case of the author's comes in for consideration here. That patient developed visual agnosia for and loss of capacity to revisualize inanimate objects by a lesion of the right occipital lobe. That fact would indicate that ability to recognize and revisualize the animate ones was mediated by the left occipital lobe. These three cases then suggest that animate objects may be recognized and revisualized by the left occipital lobe while the same functions for inanimate objects proceed through functional activity of the right lobe. In other cases the left occipital lobe seems to be divided in the two functions, a lower area for inanimate, a higher for animate ones.

The site of the lesion for visual finger agnosia seems well established by a number of cases. Lesions on the border between the major angular gyrus and the second occipital convolution cause visual finger agnosia. The occipital lobe is more affected than is the angular gyrus in such cases. The site for lesions producing confusion of laterality is grossly identical.

The site of the lesion causing finger aphasia or aphasia for laterality and parts of the body seems to be slightly farther forward toward area 37, the language formulation area. In some cases the lesion has undermined the angular gyrus.

In most instances the lesion has also caused homonymous hemianopia, but in some instances the lesions have been so superficial that the fields of vision were unaffected.

PHYSIOLOGIC BODY SCHEME AND ITS DISTURBANCES

Disturbances of one's concept of gross neurologic physiology have been described as separate conditions but may be grouped as disturbances of the physiologic body scheme parallel with those of the anatomic body scheme. The basic unifying factor in them is that the patient's concept of his normal physiology is not altered by the development of gross alterations of the physiology. It thus contrasts sharply with disturbances of the anatomic body scheme in which the patient believes the anatomy to be pathologic while it remains normal.

Historically, the first description of such a condition seems to have been the one by von Monakow in 1885. His patient became blind through bilateral lesions of the occipital lobes, yet he denied the blindness. In 1898 Pick reported instances of hemiplegia denied by the patient. He cited some teachings of his master Anton for which reason it is probable that Anton had made previous observations of the condition. In addition in 1899 Anton described unawareness of deafness and also the important fact that patients with Wernicke's

aphasia were often unaware of their inability to comprehend spoken language. The latter is now described as unawareness of auditory agnosia. In 1914 Babinski, unaware of Pick's description sixteen years earlier, presented, as a new syndrome, denial of one's own hemiplegia. He coined a new term, *anosognosie* (a = deprivation, nos = disease, gnosis = knowledge or recognition of), and consequently the conception was widely disseminated. Babinski is commonly credited with priority.

Disturbances of the physiologic body scheme result from lesions of the minor side, of the major side, or of both sides. Following the system outlined for the anatomic body scheme the disturbances due to lesions of the minor side will be presented first.

1. Minor side.

Babinski's term anosognosia was not fortunately chosen. The term means lack of recognition of disease in general, whereas the condition itself is only lack of recognition of hemiplegia. Moreover, at Babinski's presentation before the *Neurological Society of Paris* (June 11, 1914) much discussion arose, all of which had the effect of diffusing the applicability of the term. Pierre Marie pointed out that visceral as well as somatic disease might be ignored. Gilbert Ballet called attention to lack of recognition of blindness without, however, mentioning von Monakow, and Henri Meige suggested that it might be the limbs and not the paralysis which were unrecognized. He said it was to the patient as though the paralyzed limbs had never existed. Souques expressed the opinion that the condition was a forgetting of the affected side. By the time the meeting was ended it was clear that Babinski's term for the one condition was inappropriate but no one actually summarized the situation.

Since it has become clear that agnosia is loss of recognition and through one sense organ only it is obvious that anosognosia is not merely an agnosia; there is always an associated illusion or delusion or both. Moreover, when patients have amnesia for, or delusion of absence of, the limbs one cannot speak of lack of recognition of the hemiplegia. If the patient is not even aware of the limbs he cannot be expected to be aware of their paralysis. A disturbance of the concept of anatomy, therefore, precludes a disturbance of the physiology. One can speak of a disturbed physiology only when there is an anatomy present whose function may be disturbed.

Lack of recognition of paralysis of the minor (usually left) limbs is not always present in cases of left hemiplegia but it is so common in cases of cerebral vascular accidents that it is difficult to understand how the condition escaped notice for so many years. Patients who come in with left hemiplegia do not usually have aphasia and are therefore able to give good histories.

When such a patient awakens in the morning he commonly throws his good leg out of bed and attempts to get up only to fall to the floor. He does not recognize the left-sided paralysis and will not believe that it exists. When he arrives at the hospital he states that he fell on rising in the morning but that he will soon be all right. When he is asked about paralysis he denies being paralyzed. When asked to raise the left arm he agrees to do so and proceeds to make the effort. When we ask him whether he has raised it he answers in the affirmative.

The disturbance is therefore a true delusion and here, as in other disturbances of the body scheme, we see confabulation; the patient claims he has raised his arm when he has not and when he can see that he has not. In fact the patient usually makes no effort to see for himself; his sensation has already decided the question.

Generally speaking, in a few days to a week the patients recognize the paralysis and take a normal attitude toward it. Rarely the delusion persists for months or years.

2. *Major side.*

There is only one disturbance classified as physiologic, which is concerned with the body scheme, and which is due to a lesion of the major side. It was described by Anton as lack of recognition of one's own word deafness. Just why some patients with auditory agnosia are conscious of their defect and others are not is a matter not understood. It is probably due to a lack of self-criticism on the part of some persons but it may depend on the site of the lesion. The writer is strongly inclined to believe that separation of the area of Wernicke from the thalamus causes unawareness of the defect.

The syndrome is commonly seen in association with Wernicke's aphasia: the patient talks a great deal and attempts to answer questions which he has not understood. The same defect is commonly seen on a functional basis in deaf persons regardless of the cause of deafness. The humorous replies of the deaf are an old source of amusement.

3. *Simultaneous lesions of the two sides.*

There are two striking symptoms of which the patient may be unaware —blindness and deafness. When these defects are due to disease of the pallium the lesions must, of course, be bilateral. It has never been determined why some persons deny the blindness or the deafness and confabulate to conceal the loss of function rather than acknowledge the defect. It would seem to a normal person that the difficulties which deaf patients encounter in attempting to conceal the deafness are far greater than the embarrassment of admission.

Their judgment must be defective and defective judgment is a constant accompaniment of the syndrome of unawareness of deafness. The patient deludes himself into believing that he has heard.

The author's concept of the pathogenesis of the syndrome of unawareness of deafness is that the auditory fibers are interrupted close to the thalami, just lateral to the lateral geniculate bodies.

Lack of recognition of blindness is indeed striking to the observer. The patient pretends to see. He will describe objects held up before him even though unable to distinguish daylight from darkness. The confabulation is a constant symptom in the syndrome and the answers given by the patient are characteristically vague. He will say, "The color is light, not exactly white and not gray, but sort of a light color." "That thing is small, not large like a pillow but larger than a fountain pen. It is all right. It looks something like a shawl or some cloth." When he is asked to state when a flashlight is turned on or off he will answer directly in the affirmative or negative.

The symptom does not of necessity occur from a bilateral lesion of the calcarine cortex but may apparently do so. It is more apt to result from lesions nearer the thalamus.

CHAPTER IX

METHOD OF EXAMINATION

THE BASIC principle governing examination of the patient for agnosia, apraxia, and aphasia which is followed by the authors is merely to work from the simple to the complex. In skeletal form it is: agnosia—apraxia—aphasia. For ethical reasons the physician should introduce himself to the patient and thus obtain immediately a perspective of the type of case that confronts him. As stated before, the psychologic factors must be taken into account at all times during the examination. It is impossible to make a separate or preliminary psychologic study but a preliminary physical examination is essential.

Introductory. The patient is greeted with, "Good morning, how do you do?" or something similar, to detect whether or not he is in stupor, whether he can answer, whether he is paraphasic, etc. In the cases presented here this introduction is not always recorded, because these matters had already been determined prior to the examination.

AGNOSIAS

1. Visual agnosia. We begin at this point, because it has been found that for the patient recognition of objects about him is fundamental, and unless the examiner knows that the patient has this faculty he will often be led astray into other spheres. If the patient is known to handle objects about him with understanding, no special test is necessary. If he seems disoriented, it is essential to determine whether this may not be due to visual agnosia.

It is often difficult to determine whether he recognizes objects, as he may be unable to talk, to write, or to indicate by signs, and he may have hemianopia. If he can copy, visual agnosia is excluded. If he seems confused, he is allowed to feel, taste, or smell objects to aid his process of identification. Sometimes he fails to recognize objects even after the application of all senses. This defect goes beyond the sphere of agnosia.

2. When one has determined that the patient recognizes objects in general, one tests to determine whether he recognizes letters, mathematical figures, words, musical notes, other symbols, and colors. If the patient cannot talk or

if he talks with such marked paraphasia that one cannot determine by his statements what he means, it is well to have him select letters, figures, words, etc., on command. One of the authors' favorite methods for determining the ability to read words is to write with a heavy black pencil on a large sheet of white paper a few words of four to six letters and have the patient select words of a certain type, as things good to eat or to drink. If he can select words according to meaning it is determined at one stroke that he has no profound semantic defect. In testing for colors he is asked to select certain colors from a group. If he does not understand what is said to him, the physician writes a sentence which will provoke an emotional reaction, as, "Do you want to get well?" The authors also have pictures for him to identify. He may show by pantomime what he sees. If the examiner cannot get *any* response whatever, he is asked to sort into groups cards bearing words. This tests only recognition.

3. Failure to read may rarely be due to geometric-optic agnosia, the letters appearing to the patient to be crooked or jumbled. He then probably has disorientation in space as well.

4. The patient should always be tested for his ability to recognize and to name colors. Loss of the first is agnosia, of the second, aphasia. He is asked to select colors which the examiner names if he shows difficulty in naming them. He is asked to sort colored cards into groups. Any disturbance of this type at once leads one to investigate closely the functions of the occipital lobes.

5. Next is tested the patient's ability to revisualize scenes, buildings, and colors, perhaps even letters and words. He may be asked to describe an A or an H.

6. Acoustic agnosia. In acoustic agnosia the patient is unable to recognize what he hears, words, musical notes, and perhaps all sounds. This condition is rarely complete; in fact the common finding is paragnosia, the patient mishearing or misinterpreting what he hears. Acoustic verbal agnosia is far more common, as would be expected, than complete agnosia.

7. Besides testing for recognition of words heard, the authors test for identification of other sounds as that of crumpling paper, a bell, and common tunes. The patient may not be able to state whether he recognizes words, but by his actions one can tell. The authors find that it is not safe to depend upon the usual test of asking him to put his finger to his nose. He may, because of marked semantic defects or apraxia, fail to carry out the order, although he understands the words. We have learned to ask first, "Have you a nose?" If he says "Yes," we ask him, "Where is it?" He will then point to it. By thus getting the request in two stages he comprehends with much greater facility. The differentiation from apraxia is given below.

Patients who have verbal deafness will often do just as deaf persons will do, i.e., guess at the question rather than admit that they have not understood. If two slightly different questions are asked, the same answer is given to both (Dejerine).

8. Tactile agnosia (asterognosis) will have been determined in the physical examination. Olfactory and gustatory agnosia cannot be detected. They appear as anosmia and ageusia.

APRAXIAS

1. Kinetic apraxia of the limbs (cortical motor pattern apraxia). One notes with ease whether the patient has merely lost his dexterity in fine movements —kinetic apraxia of the limbs or whether the whole limb is paralyzed.

2. Ideomotor apraxia is so striking that it has usually been discovered before the patient is examined. If tests are necessary, one proceeds to have him do various complicated acts, as lighting a match, cutting with shears, etc. To exclude agnosia (in the presence of which the patient would fail to carry out commands because he fails to comprehend what is wanted) the examiner seats himself before the patient and has him mimic all acts with both hands, both lower limbs, and face. Except in the rare cases of total imperception, in which the patient pays no attention to anything that goes on about him, this method of mimicry will demonstrate apraxia. In general imperception one depends on what the patient spontaneously does in difficult positions into which he is placed.

3. In ideational apraxia the same tests may be used, but they are usually not necessary. The patient appears extremely absent-minded, lighting a match to light his cigar but apparently forgetting to finish the act or placing the wrong end of the cigar into his mouth.

APHASIAS

1. The patient's emissive speech is for the most part at once noted without difficulty. The most obvious form is aphemia or subcortical motor aphasia, in which he says absolutely nothing. If the patient is seen some time after the vascular insult which caused the aphasia, he may be able to say a few words. If he speaks several languages, it is well to encourage him to try them all. He may possibly be able to speak one of them.

2. If the patient is found unable to speak, there are three details to be considered: (1) Can he repeat what he is told to say? If so, his minor hemisphere has good repetitive ability. (2) Is his entire speech area so severely affected that he fails to speak because he has nothing to say? The difficulty may thus be entirely in the internal language, and chiefly on the sensory side. The events

of the first week or so after the vascular accident will clear up this question as a rule. If his condition permits, his ability or inability to write may show immediately whether or not the internal language is preserved. (3) Can he sing, perhaps even words? One usually neglects to test this point when the patient is obviously speechless; yet it is important at least for the future study of aphasia to have autopsies on such cases. If the patient can talk, it is nevertheless also important to determine whether he can sing.

3. If the patient does talk, one notes the character of the speech. The most common error of speech in this category is paraphasia, which may be so severe that it is classed as jargon aphasia. Or the patient may repeat whatever he hears in a meaningless way (echolalia). Grammar may be apparently entirely ignored (agrammatism or akataphasia); telegraphic style is a form of this. These are disturbances of language formulation.

4. Special attention is always directed to amnesic aphasia. The patient is asked to name objects shown him. He may not be able to state whether he recognizes each, but he can indicate its function. He commonly uses circumlocutions, as, "It's what you comb with." Or he says, "I know but I can't say it." This may apply equally in the musical sphere and in attempts at writing.

5. One notes whether the patient can read and get sense out of what he reads. Here are several points which can be nicely discriminated. Recognition of the written or printed word is one thing; the loss of this function, as already stated, is visual verbal agnosia. The second point which is stressed at this place is whether the patient comprehends simple statements consisting of a few words. He should consider this different from recognition. The loss of ability to comprehend simple written or printed statements is alexia on the level of aphasia, a higher level than that of agnosia. The third point is the inability to comprehend complicated statements, which is on a still higher level (semantic aphasia). This will be considered in more detail later.

6. An interesting element always to be considered is the occurrence of the syndrome known as transcortical sensory aphasia. In this the patient can read fluently but understands nothing of what he reads. He may also repeat what he hears without understanding it.

7. Optic aphasia may easily be overlooked. This is really amnesic aphasia in the optical sphere. The patient appears not to recognize what he sees, although he actually does recognize it but merely cannot name the object until he hears the name. Acoustic-optic aphasia is related to this but is not identical. In this form the patient cannot comprehend propositions which have only an optic association, although he has no difficulty with other propositions. He will thus not understand, "The light shines," but will grant, "The light made by crackling, burning wood is bright."

8. The question of intonation should have more consideration than it has received in the past. Some patients can use intonation so effectually that they can, in spite of inability to find the words they want, almost express themselves. Others can apparently not use intonation even though they have words to express their thoughts.

9. Finally one should look for Wernicke's aphasia complex. In this the patient has visual verbal agnosia, acoustic verbal agnosia, alexia, and agraphia, with usually severe paraphasia. The volubility of the jargon speech should lead one to suspect it.

The time-honored test of having the patient recite the months of the year, the days of the week, and the alphabet seems to the writer to be a better test for amnesic aphasia than for motor aphasia. The words needed for such a recitation are, in the mind of the patient, all names, i.e., nouns, and are not easy to recall until a start is given.

SEMANTIC APHASIA

This is one of the most common elements observed in aphasia. When one addresses a patient suffering with aphasia, one finds in nearly every case that one must be careful to make one's statements simple, direct, and concise. If they are at all complicated, the patient fails to grasp the significance. This is because of semantic defects on the sensory side. Thus, if the patient is asked, "If you spend 10 cents out of a dollar, how much change would you get?", he may tell us correctly, "Ninety cents." But if we ask him thus, "If you go into a store with a dollar and buy cigarettes for ten cents, how much change would you get?", the increased number of words may alone be enough to confuse him. Or he may grasp the significance of each of the words in the question, "What was it that Adam and Eve did in the Garden of Eden to cause them to be expelled?" and yet fail entirely to grasp the idea sufficiently to answer the question. The discovery of such defects, however, does not help us in cerebral localization.

ACALCULIA

This does have a slight localizing value. One determines whether the patient can perform mathematical calculations of which he was capable before the aphasia developed. The class of patient with which one is dealing determines the type of mathematical problem which it is appropriate to give. Sufficient complexity (varying with the case) will cause any patient to fail.

WRITING

It would seem easy to test for the ability to write. It is actually quite complicated.

1. First, can the patient write spontaneously? He is given a pencil and invited to write. He frequently says he cannot write because his hand is weak. He is urged to use the left hand, and if he actually cannot use either because of paralysis of one and apraxia of the other, we ask him to try building words with blocks. We may utilize simple blocks so that he may build large letters with them or embossed blocks each of which carries one letter. Sometimes, if his vision is poor or he has visual agnosia and does not even recognize a pencil when he sees it, we ask him to write with his eyes closed. This may be successful. The ability to write his own name should not be taken as sufficient evidence that he can write. Ask him to write something entirely different.

2. If the patient cannot write spontaneously, or even if he can, he is asked to write from dictation. He may fail because of acoustic verbal agnosia, or he may apparently succeed but not be aware of what he has written.

3. If the spelling is wrong, one notes what sort of errors he makes. Distinguish between literal paragraphia and written paraphasia. In the former the letters are jumbled; in the latter he writes what he would say if he spoke. In other words, the second form is subject to pronunciation. He may write neologisms.

4. He is asked to copy from handwriting and from print and from print into handwriting. We note whether he copies slavishly, letter under letter, or whether he copies in his own handwriting. If he appears to lose sense of direction in forming the letters, a geometric-optic agnosia may have been overlooked. To determine this point it is useful to have him copy a small geometrical figure.

5. The patient may be able to copy figures which are not symbols while unable to copy symbols of any kind. This proves that he does really not copy or attempt to copy the symbols, but instead reads the symbols and then writes them. This is a valuable discovery as the differentiation has localizing value.

6. The patient may be able to write in one language and not in another. In polyglots several languages should be tested.

7. Care should be taken not to interpret mirror writing as scribbling. If one observes the process of writing, there will be no mistake. Much is learned by this means. It is a great mistake to leave the patient to do his writing and return later for the sample.

MISCELLANEOUS SYNDROMES

1. The condition called simultanagnosia properly belongs here. This is the inability to synthesize the elements of processes into a whole. An excellent illustration is the inability to comprehend cinematography with retained abilit

to recognize each picture. The patient should be asked to describe a picture so that one may determine whether he sees only details of it or comprehends the whole.

2. The agnosias of Liepmann called ideational, disjunctive, and dissolutive come under this heading. The essential defect in these forms is the lack of ability to correlate the various sensory impulses received through several sensory channels. One looks for such defects especially in cases where disorientation is commonly suspected. If the patient fails to recognize objects by the use of one or even several sensory channels, does he recognize them when still other channels are used? In other words, the inquiry must be intensive if defects of identification are found.

3. The Charcot-Wilbrand syndrome seems to the writer to come under this heading. The revisualization of old visual impressions is not recognition but a function of a higher order. It is through complicated associations that such revisualization is effected. One tests for this by asking the patient to describe scenes which he should know. A subdivision of this is the inability to revisualize letters, words, and figures. We ask the patient to describe what an H or an A looks like, or how a Q differs from an O. The ability to do this is the basis of the ability to write.

In concluding the discussion of methods of examination the authors wish to say forcefully that no amount of outline or preparation or following of a scheme will take the place of or compensate for ingenuity and perspicacity on the part of the examiner. The method given is an instrument, nothing more. In the application of the scheme, it must be obvious that various types of agnosia, apraxia, and aphasia are mutually exclusive. A case never occurs requiring all the tests here given.

CHAPTER X

THE EVIDENCE

MUCH OF the evidence upon which the statements concerning localization of the functions of speech and the physiology of language are based has been presented in the remarkable and voluminous monograph of Henschen (1920–22). The limited availability of the monograph and its massiveness have unfortunately combined to leave it relatively unread. Also, since its publication much has been discovered and many details elaborated; the material concerning the body scheme (Chapter VIII) is all new. Instead of following Henschen's method of statistical analysis, which is inevitably long, only crucial cases will be presented here; the necessary evidence can thus be greatly curtailed and at the same time be brought into sharper focus.

Individual case studies alone cannot demonstrate all of the facts of aphasia and apraxia. This is true because the capacity of brains of various persons in language varies enormously and a truth demonstrated on one patient may not be applicable to the next. This fact should be immediately evident when one keeps in mind that whatever function may be shown to exist in a given case depends on the function of the cerebral structures *remaining*, and not directly on the lesion. Hughlings Jackson emphasized that fact in the nineteenth century but the force of the statement was not felt or understood by many students at the time or even subsequently. Even when Henschen later discussed the matter in detail and devoted a special chapter of his monograph to the function of the right cerebral hemisphere in language it was not fully realized by many neurologists that a given function lost by a destructive lesion of the major side is compensated for, if at all, by the function of the homologous area of the minor side.

Because of the variability among cases Henschen made elaborate statistical studies by which he determined not only the rule for each function but also the exceptions. Through his work it became evident that if one author reports that a lesion of Broca's convolution produces agraphia while another reports that it does not, we need not assume erroneous observation on the part of one but rather that the two cases were different because of differences in correlation of the various speech centers in the brain. To establish any principle,

therefore, it is necessary to present a number of instances and to discover the generalizations which are valid.

The selection of illustrative cases is a matter of great importance. The patient during the study must have been sufficiently clear mentally to insure that his disturbance of language was not based upon a general mental defect. His attention must have been sufficiently good that lack of it does not explain the clinical findings. The clinical and pathological studies must have been adequate. In the pathologic study the question of location of the lesion must have received special attention. The lesion must have destroyed the area affected; compression by a neoplasm or the occurrence of sclerosis in an area does not necessarily render it functionless. Cases of neoplasm cannot be utilized except in the rare instances of well circumscribed gumma, tuberculoma, or other focal new formation which causes, at the site and not elsewhere, destruction of the tissue and not pressure. Neoplasms usually grow for a considerable time after the clinical study is made and thus render the autopsy findings useless for clinical interpretation. Besides, they frequently produce a reactive gliosis in the surrounding tissue the limitations of which cannot be accurately determined. Cases of agenesis are of no value because in such instances language is not lost; it merely is not fully acquired.

With these restrictions in mind we can proceed to a presentation of carefully selected cases to prove the points stated dogmatically in the preceding chapters.

THE UNILATERAL FUNCTION IN LANGUAGE: HEMISPHERECTOMY OR DESTRUCTION OF THE LANGUAGE AREA

Inasmuch as both cerebral hemispheres have a function in language but that one is far more capable than the other the two may be designated as major and minor. If cases of destruction of the entire major language area are gathered for study we can readily tell what capacity the minor hemisphere possesses. Destruction of the area supplied by the sylvian artery is equivalent to hemispherectomy so far as language is concerned because the occipital and frontal extremities have no function in language.

During the last ten years of autopsy of brains at the Los Angeles General Hospital I have discarded numerous brains with softening in the distribution of the major sylvian artery because the clinical records showed that the patients simply had no language function. It seemed so obvious that patients with such extensive lesions of the major language areas could not possibly have any language function that the brains were not even photographed. They would have been valuable additions to the present study as cases of hemispherectomy. However, they would merely have served to show that in cases of acute exten-

sive lesions the patients never succeeded in using their minor hemispheres at all. In the 19 cases gathered here from the literature (see the bibliography) all were verified at autopsy. These cases will demonstrate that in many instances patients who survive such lesions may be able in time to manifest some function of the minor cerebral hemisphere.

1. Zollinger (1935). A woman of 43 years with extensive tumor of the left cerebral hemisphere was operated upon and the entire hemisphere, except for the basal ganglia, was removed. A few hours after surgery she replied, "All right," to everything asked of her. On the second day she also said, "yes" and "no." On the third day she said, "thank you," "sleep," "Good-bye," and "Please." The word "sleep" was said when she was tired after an examination. She seemed to use "yes" and "no" with understanding of their meanings. The patient died on the seventeenth day after operation.

2. Moutier (1908, observation XX, page 579). A man whose age is not given survived a cerebral softening for eighteen years. He uttered syllables without meaning. He sat down when ordered to do so but when asked to protrude his tongue he opened his mouth; when asked to mimic the act he performed no better. He could not be made to read or to write and he could not sing.

Autopsy showed an enormous softening extending nearly the entire length of the hemisphere so deep that the lenticular nucleus was absent. The destroyed areas included the first and second temporal convolutions (except the extreme anterior portion), the angular gyrus, the supramarginal gyrus, and the foot of the third frontal and much of the second frontal convolutions.

3. Moutier (observation XII, page 540). A man of 40 years, ill with hemiplegia and aphasia for twenty years, comprehended all commands and executed them. He spoke spontaneously and plainly except that at times he omitted the articles. When asked to repeat long sentences or phrases he repeated only the principle words. He sang "admirably." While he was able to read separate letters and numerals he was unable to read even detached words. He was unable to write or even to copy.

At autopsy a softening was found which had completely destroyed the foot of the second and third frontal convolutions at their insertion on the prefrontal area and on the pars triangularis. Behind, the focus destroyed the white substance of the insula and the retrolenticular portion of the internal capsule. It extended into the white substance below the posterior horn of the ventricle.

It is worthy of note in this case that while the lateral cortex would appear superficially to be preserved to a considerable extent, closer scrutiny shows it to have been undermined and therefore isolated. An area of cortex isolated from all connections cannot, of course, function. The youth of this patient

and the long period available for re-learning explains his remarkable performance with the right cerebral hemisphere.

4. Moutier (observation XI, page 522). A man aged 54 years was taken with right hemiplegia and aphasia and survived eleven years. He comprehended simple commands. Attempts to speak resulted in a few words spoken clearly but rapid fatigue followed with resulting jargon aphasia. He was unable to whistle or to sing tunes. He read simple commands and executed them. He also recognized numerals and traced them with his fingers and he was able to calculate slightly on his fingers. He copied from print to handwriting. While he recognized objects he could not name them.

Autopsy showed lesions located both cortically and subcortically. The areas destroyed comprised the foot of the third left frontal convolution, the superior and the anterior portion of the second temporal convolution, the angular gyrus, and the posterior limb of the supramarginal gyrus. There was a cystic space separating the cortex of the insula from the lenticular nucleus. The posterior horn of the lateral ventricle was greatly dilated and there was a cystic cavity in the third frontal convolution.

Even though this man was 54 years old at the time of his first illness he recovered considerably in the eleven years of survival. The return of ability to read to some extent is the most striking element in the case, but his ability to speak is also noteworthy.

5. Mills (Henschen case 1068). A man, whose age I have not been able to determine, at first understood nothing that was said but gradually improved and understood partly. His only utterance was "anyone." Reading and writing were not reported.

Autopsy showed the sylvian area destroyed. The foot of the left third frontal convolution, the precentral and postcentral gyri, and the insula were destroyed. The inferior parietal region and the superior temporal convolution were necrosed.

6. Goldstein (Henschen case 934). A woman of 53 years suffered a cerebral vascular lesion eleven months before examination. She was able to comprehend simple commands as well as words but repetition was faulty. She had complete amnesic aphasia. She was able to read neither letters nor words with comprehension. The only thing she said was, "Ich kann nichts." She was able to copy a few letters and to write her own name.

Autopsy showed a cystic cavity tensely filled with fluid. This took in nearly the entire hemisphere except the temporal lobe, which, however, was isolated. The third frontal convolution, including the pars triangularis, operculum and white matter, precentral and postcentral gyri, were destroyed, as were the supramarginal and angular gyri. The corpus callosum was degenerated.

7. Pick and Kahler (Henschen case 1180). A man of 45 years began at the age of 32 years to have episodes of unconsciousness with loss of speech. Five months before the study he lost the faculty almost completely. There was some echolalia and marked amnesic aphasia. He could neither read nor write nor even copy. Spontaneous speech consisted of "yes" and "no" and combinations of these and "I am 45."

Autopsy showed an extensive encephalomalacia from the foot of the third frontal convolution to the second occipital inclusive. There were even a few small areas of softening in the right temporal gyri, corpus callosum, internal capsule, gyrus fornicatus, and in the thalamus.

8. Bernard (Henschen case 766). A woman of 80 years had for three years had paralysis of the right limbs but was still able to speak. After a new ictus she lost power of speech. There was partial word deafness; she understood a few questions addressed to her. She could not recognize words written. Speech was reduced to a neologism and a few syllables. She was unable to calculate or to indicate the value of money. Writing was not tested.

Autopsy showed the third left frontal convolution riddled with small foci. The inferior part of the precentral and postcentral gyri were similarly affected. A plaque in the pars triangularis extended in to the ventricle. An area of softening occupied the inferior part of the first temporal convolution, the angular gyrus, the anterior portion of the insula, and the middle portion of the postcentral gyrus.

9. Beduschi (Henschen case 742). A patient aged 49 years suffered an ictus three years before. Spoken language was reduced to "ma ma." Practically nothing spoken by others was understood by the patient. Reading and writing were impossible.

The lesion was very extensive. The foot of the second and of the third frontal convolutions was destroyed as well as the anterior part of the insula. The posterior part of the first temporal convolution, a part of the second temporal, the cuneus and precuneus, putamen, and the anterior part of the internal capsule were degenerated.

10. Rosenthal (Henschen case 1209). A patient aged 39 years suffered a cerebral vascular accident and survived only three months. He did not respond to questions and his spontaneous speech was reduced to "Weiss nicht" and "Mein Gott." At autopsy the entire sylvian area was softened and degenerated.

11. Touche (Henschen case 1267). The patient, aged 54 years, as a result of a cerebral vascular lesion became "a veritable deaf-mute." He could not read, write, or speak. At autopsy the entire sylvian area was destroyed. There was a separate area of softening in the angular gyrus.

12. Mingazzini (Henschen case 443). A patient aged 65 years suffered a

cerebral vascular accident as a result of which he became word deaf but responded with echolalia. His spontaneous speech was reduced to a few syllables. At autopsy the middle portion of the first, second, and third frontal convolutions was found softened. The first temporal convolution was much reduced in size and the middle and anterior portions of the second and third temporal convolutions were absorbed. The transverse gyri were gone. The lower part of the occipital lobe was degenerated.

13. Wernicke (Henschen case 1319). An apothecary aged 64 years first noticed that he was no longer able to read or to write. There was no motor or sensory paralysis but he had a right homonymous hemianopia. He was not word deaf but he had lost power of speech. At times he spoke without meaning. He could copy only slavishly and was unable to name objects. He improved in one year so that he was able to write but he did not learn to read at all. He survived twenty months without learning to speak.

At autopsy there was found softening in the posterior part of the left hemisphere. There were foci in the first and third frontal convolutions and a large focus in the postfrontal and prefrontal gyri. There was a subcortical focus extending from the second temporal convolution to the angular gyrus and a large focus subcortical to that gyrus extending to the ventricle. Still another lesion extended from the second temporal convolution to the second occipital. The anterior two thirds of the lenticular nucleus was softened.

It was noteworthy that the patient relearned to write but not to speak.

14. Maas (Henschen case 1033). A man of 33 years suffered a cerebral lesion which he survived three years. After a few months he learned to repeat what was said, to comprehend fairly well, and even to read to some extent. He became able to write though in telegraphic style and he found his words fairly well. At autopsy a large cyst was found. The transverse gyri were destroyed as well as all of the language area except a small part of Broca's convolution.

Here a relatively young man learned in only a few months to use the other hemisphere to a limited extent.

15. Gatti (Henschen case 917). A patient suffered in infancy a lesion which caused gross paralysis and atrophy of the right limbs. He grew up without any disturbance of language function. At autopsy a vast and deep lesion was found in the left hemisphere occupying a large part of the second and all of the third frontal convolutions as well as the insula, the inferior two-thirds of the precentral and postcentral convolutions, the anterior part of the inferior parietal and the greater part of the posterior first temporal convolutions.

16. Ives (*Bulletin of the Los Angeles Neurological Society,* 1937, page 36).

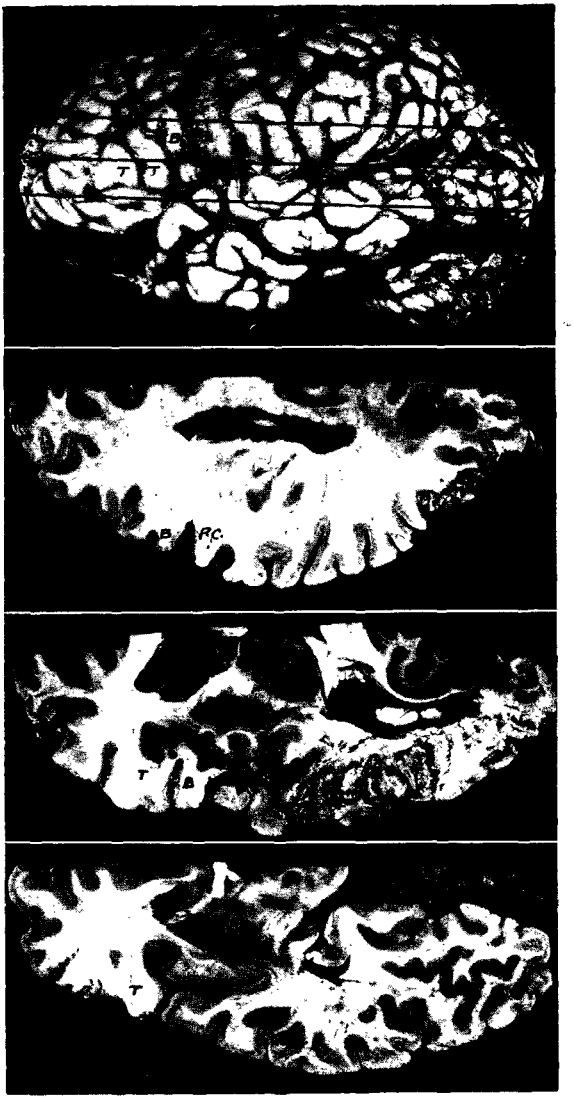

FIG. 16. Case 16 (Ives, 1937). On the left lateral view of the brain have been drawn lines indicating planes of section. The lower three views are respectively the sections from above downward photographed from above. The marked narrowing of the temporal lobe in the posterior half is due to degeneration of the superior temporal convolution, the middle and inferior temporal convolutions not having been affected. There is also superficial softening of the precentral convolution (*P.C.*). The posterior limit of softening which appears in the first section occurs in the posterior part of the angular gyrus. The extensive softening of the superior temporal convolution is graphically shown in the middle section. Superficially there appears to be little involvement of Broca's convolution (*B*) and the pars triangularis (*T.T.*) does not seem affected.

A white laborer of 50 years suffered a cerebral vascular accident. Studied after six weeks he showed ability to respond to a few simple spoken commands before he became fatigued. He correctly raised his hand on request and placed his finger on chin, nose, and ear but then failed on further attempts. He could not select colors on spoken command. Spontaneous speech was characterized by a few perfect sentences but by much paraphasia with neologisms. He was able to play cards but unable to read or to write. His general mentality was well preserved as he helped with housework at home. He recognized music and indicated a defect in pitch.

At autopsy six months later the superior temporal convolution, insula, Broca's convolution, and the occipital lobe were softened and disintegrated (Fig. 16).

17. Bastian (Henschen case 71). A man of 32 years suffered a cerebral vascular accident in 1877 and had several exacerbations after an initial improvement. He died in 1896. During several periods of remission he learned to read and to write to some extent but the final clinical picture, which corresponded to the autopsy findings, was of inability to speak, slight ability to comprehend spoken language, no ability to name objects, to read, or to write.

Autopsy showed an extensive lesion of the entire language area including the insula. The anterior one-third of the first temporal convolution was preserved but isolated.

18. Liepmann (Henschen case 427). A woman of 65 years suffered a cerebral vascular accident and survived two months. She was able to speak to some extent and able to comprehend spoken language quite well. She was totally unable to read or to write.

Autopsy showed a large subcortical cyst involving the entire language area. The temporal isthmus was completely destroyed.

19. Giannuli (Henschen case 925). A man of 51 years suffered a series of left sided cerebral vascular accidents over a period of five years. He failed to speak spontaneously except for a few syllables; he was unable to repeat spoken language. He comprehended little of what was said. Reading was confined to letters and single words; the author states that reading was impossible. The patient was unable to write, except his own name.

At autopsy there was found softening of the opercular portion of the precentral and postcentral gyri, all of the paracentral lobule, the insula, the putamen, part of the thalamus, and most of the upper three temporal convolutions.

Analysis of the preceding 19 cases.

In the 19 cases presented the question of handedness is not discussed; the fact that the patients had aphasia is proof that the major side was the one

affected in all cases except that of the infant. In that instance discussion of handedness is useless because it does not develop until the age of 9 to 12 months. It is well known that aphasia does not develop in infants from a unilateral lesion regardless of laterality. However, the infant is excluded from the following analysis:

A review of the 18 cases shows that comprehension of spoken language was easily recovered as it was excellent in two (cases 3 and 13), good in two (cases 14 and 18), fair in four (cases 4, 5, 6, and 8), slight in six (cases 1, 2, 7, 9, 16, and 17), but practically nil in four (cases 10, 11, 12, and 19).

The function next most easily recovered was spontaneous speech, which was excellent in one (case 3), fair in three (cases 1, 4, and 18), slight in eleven (cases 2, 5, 6, 7, 8, 9, 10, 12, 13, 14, and 16), and nil in three (cases 11, 17, and 19).

The functions of reading and writing were recovered only exceptionally. Other functions which it would have been interesting to compare, such as recall of words and ability to calculate, were noted too little to make comparison worth while. Paraphasia was present in only two cases.

As would be expected, the younger the patient, the better the general health, and the longer the survival period, the better the recovery.

Another view of these cases reveals additional valuable information. Since it is clear from this discussion that comprehension of spoken language may be recovered to a much greater degree than other functions, emissive speech less, reading and writing least, we have evidence that the various functional units are at least to some degree independent of one another. Either cerebral hemisphere, therefore, does not function as a whole in language but separate units function to some degree separately. One function may be assumed by the minor side without simultaneous assumption of the others.

SPECIFIC LOCATION OF NEURONAL PATTERNS OF SPONTANEOUS SPEECH

If we consider separately the elements of language as motor, sensory, reading, and writing it can easily be shown that these elements disappear individually as the result of focal cerebral lesions. We shall consider first the effects upon emissive speech of small lesions placed by nature in certain locations. Forty-three cases have been gathered from the literature in the first 26 of which the right side did little or nothing while in the subsequent 19 the right side performed to a degree which might be called from "quite well" to complete function. The series will serve to show that in cases of hemispherectomy in which there is little function of emissive speech the defect is due to destruction of the area of Broca and that what function remains is due to training of the minor identical area. It is immediately apparent that in the majority of instances with good recovery the patients were relatively young. In the

second portion of the series (of 19 cases) in which the function was fairly good, cases with large lesions have been included while such cases have not been considered valid for use in the cases without recovery of function. A review will convince any unbiased observer that Broca's convolution contains patterns of motor speech, i.e., engrams (neurograms), which constitute the anatomic basis of memory of how to articulate words.

1. Barré (Henschen case 734). A man who comprehended spoken language and wrote and read well showed a little scansion and monotony of speech and at times also difficulty with pronunciation of the first syllable or the first few syllables of words. Autopsy showed an old localized area of softening of the foot of the left third frontal convolution which had caused widening between the pars triangularis and the prerolandic fissure.

Barré thought that the case was not one of aphasia but the description fits perfectly that of other cases in which the right area of Broca was known to have mediated the manifest spoken language. (See the cases following.)

2. Charcot and Bourneville (Henschen case 836). A man was afflicted with "aphasia" without any trace of paralysis or loss of sensibility. (The term aphasia was used at that time as loss of emissive speech is used today.) Autopsy showed a small focal lesion of the opercular portion of the third left frontal convolution.

3. Petrina (Henschen case 1174). A man aged 53 years was taken suddenly with jerking of the right arm and the right side of the face, and with inability to speak except a few words. His comprehension remained good until his death after three weeks. Autopsy showed only a small lesion, a small tubercle surrounded by tiny ones at the foot of the left third frontal convolution. The lesion was the size of a hemp seed and did not affect the white matter.

4. Schulz (Henschen case 1225). A young man aged 20 years fell from a wagon and developed difficulty with speech followed by jerking in the right side of the face. He was clear mentally and understood spoken language but lost consciousness whenever the jerking occurred. At first he was able to repeat word for word slowly but finally could say nothing. He died on the fourth day after the fall. Autopsy showed exactly in Broca's convolution or in the precentral sulcus a yellow cheesy mass the size of a hazel nut situated chiefly in the cortex.

5. Simon (Henschen case 1241). A woman aged 68 years after a cerebral vascular accident became unable to utter a word. Intelligence was good and she understood spoken language. She died on the thirteenth day. Autopsy showed a hemorrhagic lesion 1 cm. in diameter in the foot of the left third frontal convolution.

6. Vanni (Henschen case 1294). A man aged 50 years fell in an attack of vertigo and remained unconscious for a short time. After this he was unable to speak but understood very well. He recovered completely after four days. Nineteen years later he again became mute but without losing consciousness. Aphasia became complete in five days except for his retention of ability to say "yes" and "no." Five years after the second ictus the status was as follows: He understood commands but was unable to say more than the two words mentioned. He could neither read nor write. Autopsy showed a small lesion in the cortex of the foot of the third left frontal convolution. There was also a small lesion in the first temporal convolution but this was anteriorly located.

7. Boë (Henschen case 779). A man aged 25 years, suffering with rheumatic endocarditis, fell while at work, lost the power of speech, and remained hemiplegic. He immediately had trouble with protrusion of his tongue and with deglutition, the former, however, disappearing in a few days. He was able to say only "yes" and "no" though he used the words correctly. In about a month he recovered the ability to pronounce a few more words. In writing he had a tendency to duplicate letters. He mimicked appropriately. He died in two months. Autopsy showed the left hemisphere to be normal except for the posterior part of the third frontal convolution where an area of softening 2 cm. in size was found extending back from the "highest" part of the third frontal to the sylvian fissure below. The point of greatest softening was not the most posterior portion of the third frontal convolution; there was a small band intact between this area and the precentral gyrus.

8. Bouillaud (Henschen case 786; excellent abstract in Kussmaul, 1881). A young man was injured when an umbrella was stuck into his left orbit with such force that the eye was brought out of its socket. He lived for eight days during which time he was totally unable to speak although he moved his tongue about freely and understood all that was said. On the other hand, he was able to write and stated that he had the thoughts but was unable to express them in spoken words. Autopsy showed a purulent softening of the left frontal lobe extending from the point of the puncture to the anterior end of the ventricle. "From the direction of the wound it was clear that the point of the umbrella must have penetrated the third frontal convolution."

9. Broca (Henschen case 812). Lelong, aged 84 years, suffered a cerebral hemorrhage eighteen months before his death and was aphasic except for ability to utter a few words which he used over and over. General memory and intelligence were good and he understood spoken language. He was able to tell time by the watch but unable to write. Autopsy showed a small hemorrhage subcortical to the second (slightly) and third frontal convolution which

had left a cavity continuous below with the fissure of Sylvius. There was no other lesion.

10. Charcot and Dutile (Henschen case 837). A woman at the age of 44 years suffered her first stroke of cerebral thrombosis with right hemiplegia and paralysis of the tongue. After a time she recovered her speech to some extent but agraphia remained. She was able to read the journal. Eleven years later she suffered a left hemiplegia with loss of speech again, but speech returned once more two years later. The third episode appeared seventeen years after the first and caused some further trouble with speech. In the seventeenth year she had also her fourth episode after which speech was totally lost and the pseudobulbar syndrome developed. Agraphia remained from the first episode. She died eight years after the last ictus, twenty-five years after her first.

Autopsy showed cortical softening of the foot of the second and third frontal convolutions. This extended slightly under the precentral gyrus but the insula and the basal ganglia were not affected. On the right side there were several small foci but none involving the speech areas. The portions affected were the basilar portions of the precentral and postcentral gyri, the anterior part of the third frontal convolution, the fusiform gyrus, and the third temporal convolution.

11. Charcot and Pitres (Henschen case 840). A woman of 67 years suffered first a mild attack of left hemiplegia from which she recovered little by little. There was no disturbance of speech. Later she suddenly lost ability to speak without losing consciousness. Her intelligence was retained but she was unable to utter a single intelligent word. Autopsy showed a small recent hemorrhage at the foot of the second and third frontal convolutions affecting also the inferior part of the precentral gyrus. The corpus striatum, the optic thalamus, and the internal capsule were absolutely intact.

12. Charon (Henschen case 843). A woman of 72 years had had hemiplegia for three years and was considered demented; she never uttered an intelligible sound. Autopsy showed softening of the foot of the third frontal convolution and of the inferior part of the precentral gyrus.

13. Chauffard and Rathery (Henschen case 849). A woman of 61 years two days before admission was taken with a slight right hemiplegia and complete anarthria without loss of consciousness. There was no word deafness. Because of shock the function of writing could not be tested. She survived only ten days. Autopsy showed red softening of the foot of the left third frontal convolution extending to the precentral gyrus. There was also a small plaque in the second frontal convolution and one the size of a lentil in the insula.

14. Duval (Henschen case 889). A boy of 5 years fell and fractured the left

frontal bone. Intellect was unaffected but he was unable to bring forth a single sound. He was drowned thirteen months later. At autopsy there was found a cyst the size of a walnut filled with fluid and located in the third frontal convolution.

15. Giacchi (Henschen case 920). An illiterate man of 47 years after a cerebral vascular accident was unable to utter a single word. He indicated with his hand that there was an obstacle in his larynx which prevented speech. He understood spoken language quite well and replied with gestures. Autopsy showed a cyst in the left frontal lobe affecting mostly the third frontal convolution and but slightly the second frontal convolution.

16. Leva (Henschen case 998). A man of 61 years after a cerebral ictus had a right hemiplegia and disturbance of speech. He claimed inability to read because of poor vision but he was able to write spontaneously. He was paraphasic but was able to comprehend spoken language. He replied poorly. At autopsy there was found a focus of softening in the foot of the left third convolution extending to the precentral gyrus and inward to the claustrum.

17. Liepmann (Henschen case 1008). Liepmann gives no information except that the patient (Ceremowitz) was mute ("wortstumm"). The foot of the left third frontal convolution was affected chiefly subcortically. There was a small lesion in the caudate nucleus, one in the lenticular nucleus, and one in the anterior limb of the internal capsule.

18. Malichecq (Henschen case 1052). A farmer was felled by a blow on the head. Slowly there developed inability to speak and hemiplegia. He understood all that was said to him though unable to reply. Autopsy on the fourth day revealed a blood clot pressing on the third left frontal convolution.

19. Raymond (Henschen case 1192). A woman aged 70 years suddenly became aphasic and weak on the right side of the face without loss of consciousness. She was unable to protrude her tongue. She understood questions put to her and tried to answer but was unable to bring forth intelligible sounds. Autopsy showed a small focus of softening in the posterior part of the left third frontal convolution affecting the lower part of the precentral gyrus.

20. Rousseau (Henschen case 1214). An old woman with right hemiplegia and aphasia of eighteen months' standing finally became clear but she remained unable to speak except for a few words. She was unable to write except her name. Two years later she suffered a left crural paralysis. She understood spoken language but toward the end was totally unable to speak. Autopsy showed an old area of softening "the size of a two franc piece" affecting the posterior part of the left third frontal convolution and the lower part of the precentral gyrus. On the right side there was a lesion occupying the superior third of the rolandic zone.

21. Atkins (Henschen case 715). An imbecile woman aged 45 years suffered a cerebral vascular accident in 1876 after which she had a right hemiplegia and aphasia. Her aphemia was absolute. She died after ten days. Autopsy showed a partial softening of the left third frontal convolution and of the insula.

22. Baldisseri (Henschen case 719). A tuberculous man of 25 years suffering with mitral insufficiency after a drinking bout developed a right hemiplegia and aphasia. He was totally unable to articulate while his intelligence seemed preserved. He was illiterate. He survived four months. At autopsy there was found a softening of the foot of the left third frontal convolution and the anterior portion of the insula. The ventricle was partly dilated.

23. De Boyer (Henschen case 795). A man was taken with a cerebral vascular accident in December of 1876 resulting in a complete right flaccid hemiplegia. He was able to utter only monosyllables. By February 1877 the hemiplegia had become spastic and anarthria complete. He lived three months. Autopsy showed a superficial area of softening about the opercular area affecting the posterior third left frontal convolution, the anterior portion of the insula, and the anterior upper portion of the superior temporal convolution.

24. Petrina (Henschen case 1172). A young woman of 20 years suffering with endocarditis suddenly lost power of speech. She improved somewhat but remained unable to find single words. There was no motor paralysis except for the right side of the face. She was able to say a few words, mostly interjections. Comprehension of spoken language was good but she was unable to write. Autopsy showed a cortical lesion with sinking in of the surface of the opercular area of the left third frontal convolution and the foot of the precentral gyrus. The second and third insular convolutions were similarly affected.

25. Archambault (Henschen case 712). A man of 70 years with incomplete right hemiplegia showed no signs of aphasia except a little scansion, monotomy of speech, and difficulty in pronunciation of the first letters or syllables of words. He understood spoken language and could read and write. Autopsy showed softening of the posterior third of the left third frontal convolution. The precentral gyrus was slightly affected.

26. Barlow (Henschen case 733). A boy aged 10 years after a fall immediately talked normally but the next morning he had lost his speech and had a mild right hemiplegia. By the tenth day he had recovered his speech except for occasional paraphasia. He subsequently suffered an attack of left hemiplegia which was also accompanied by aphasia. He could say only "Ah" but he could write his name. He indicated by signs what he wanted. At autopsy there were found symmetrical lesions in the second and third frontal convolutions.

This case is crucial in that the patient recovered his speech by the use of the right hemisphere only to lose it again when the new lesion affected the side which had taken up the function.

27. Bramwell (Henschen case 826). A man aged 70 years lost power of speech and ability to write and to read completely. In the course of nine days he relearned to speak well, to write to a slight extent, and to read fairly well. At autopsy the author found an area of depression and softening at the foot of the left third frontal convolution and the lower fourth of the precentral gyrus. The anterior pole of the insula was affected.

28. Dejerine-Lutelle (Henschen case 871). A man of 39 years was infected with syphilis at the age of 23. In 1900 he developed motor aphasia and transient right hemiplegia. The aphasic disturbances ameliorated rapidly. When he was examined in 1907 an aphasic study was made but absolutely nothing was found wrong. A month later autopsy was performed and the third left frontal convolution and the foot of the precentral gyrus were found softened.

29. Dufour (Henschen case 883). A man aged 38 years after an ictus was unconscious for several days. After this event he was able to comprehend spoken language but could not read. For a time he uttered only monosyllables but he later recovered even his ability to read. Autopsy showed destruction of the foot of the left third convolution with extension to the operculum.

30. Dufour (Henschen case 885). A woman of 62 years had her first ictus in 1883 with pure anarthria. She was able to write with her left hand. Twelve years later she developed right hemiplegia with contracture and motor aphasia. She began to speak again in three months and had almost completely recovered in two years. Autopsy showed softening of the foot of the third left frontal convolution and the inferior portion of the precentral gyrus, and slight involvement of the inferior parietal lobule.

31. de Font-Réaulx-Bozonet (Henschen case 905). A man aged 25 years fell with hemiplegia but without loss of consciousness three years before. He had a complete motor aphasia for six months during which time he had available one single word. When examined he had recovered except for a slight defect of articulation. At autopsy there was found softening of the posterior portion of the second and third left frontal convolutions and of the adjacent portions of the precentral and postcentral gyri.

32. Foulis (Henschen case 908). A painter aged 20 years fell from a scaffold but made a rapid recovery. At the age of 28 he had a convulsion, and as time went on he had attacks periodically until the time of his death at 64 years. He was studied shortly before his death and was found entirely free of aphasia. At autopsy the foot of the third left frontal convolution was found completely absent due to old softening. The insula was also destroyed.

33. Luys (Henschen case 1031). A young woman of 20 years suddenly became aphasic and had a right hemiplegia. While the paralysis remained permanently the aphasia disappeared in eighteen months. At autopsy there was found almost complete destruction of the third left frontal convolution.

34. Monakow (Henschen case 1116). An apothecary aged 49 years suffered a cerebral vascular accident with loss of consciousness, complete motor aphasia, and agraphia. In three days he was again able to speak. Two months later he had another insult after which he understood everything that was said but was unable to utter a word. This trouble disappeared in a few hours. Nine days later another accident left him speechless, this time for six days. He then died and at autopsy there was only one lesion, a complete softening of the third left frontal convolution, affecting also the white matter.

35. Mott (Henschen case 1123a). A woman was injured 50 years before and was mute at first. She was feebleminded but always afterward able to speak. At autopsy an area of softening was found affecting the foot of the third left frontal convolution.

36. Dejerine (Henschen case 865). A young woman of 23 years was afflicted at the age of 20 with right hemiplegia and progressive anarthria which became complete. After a year of re-education she made a complete recovery. Autopsy showed an old focus destroying the lenticular nucleus and the fibers from the third frontal convolution in their downward course.

37. Dejerine-Thomas (Henschen case 866). A man aged 45 years, after a cerebral ictus, had word deafness and inability to speak. Fifteen days later he had recovered from the word deafness but was still unable to speak. There was no word blindness. After six months he improved slightly and after five more months he was able to speak so that he could make himself understood. Autopsy showed subcortical destruction of the second and third frontal convolutions on the left. The internal capsule and the anterior one fifth of the external capsule were destroyed. On the right side the anterior portions of the first and second frontal convolutions were also affected.

38. Lange (Henschen case 986). A young man of 25 years, following a cerebral vascular accident, had a right hemiplegia and aphasia. After two months recovery began and after several more months he regained ability to speak except for some difficulty in word finding. He died a few months still later. Autopsy showed occlusion of the left middle cerebral artery with foci of softening in all left frontal convolutions and the precentral gyrus as well. The corpus striatum was softened.

39. Mills (Henschen case 1069). Nine years after a cerebral vascular accident the patient was able to enunciate single words or very short sentences but had severe paraphasia and paralexia. He wrote single words but incor-

rectly. At autopsy a large part of the sylvian area was found necrosed including the third frontal and the superior temporal convolutions.

40. Von Monakow (Henschen case 1113). A woman aged 32 years was injured and became epileptic. At the age of 47 no signs of aphasia were found. Autopsy showed a gigantic hemorrhagic cyst destroying the posterior part of the entire left frontal lobe.

41. Von Monakow (Henschen case 1114). A man aged 57 years suffered a severe cerebral vascular accident with loss of consciousness and right hemiplegia. At first he had total aphasia but this disappeared gradually. After two years he was able to designate objects correctly and he could speak to some extent spontaneously though ungrammatically. He also still had some difficulty in comprehension of spoken language. At autopsy the second and third left frontal convolutions were found completely destroyed by an old hemorrhagic cyst. Also involved were the anterior two thirds of the insula, the caudate nucleus, the precentral and postcentral gyri, the anterior two thirds of the internal capsule, and the anterior portion of the thalamus. The right hemisphere was intact.

42. Von Mayendorf (Henschen case 1136). A woman 67 years old, after a cerebral vascular accident, became mute. There was no trace of word deafness or of musical deafness. After a time there was great improvement and the patient was then able to read aloud, repeat spoken language, and even to answer questions. There was hardly any spontaneous speech. Spontaneous writing was poor but writing to dictation was almost without fault. Autopsy showed extensive softening of practically the entire third left frontal convolution, the inferior one third of the precentral gyrus, and the lower part of the postcentral gyrus. The caudate nucleus and the external capsule into the thalamus were destroyed.

43. Simon (Henschen case 1240). A weaver aged 55 years awoke one morning unable to speak though still able to understand spoken language. After four days speech returned but it was hesitant and slow. His speech became completely comprehensible without paraphasia. He died after two months. Autopsy showed a sunken area in place of the third left frontal convolution and some involvement of the second frontal. The outer border of the corpus striatum was softened.

Analysis of the preceding 43 cases.

A review of these 43 cases shows that in them the loss of emissive speech is practically the same as in cases of hemispherectomy. In the first 25 cases, cases of destruction of Broca's convolution (in a few nothing else and in many a little additional tissue), there was essentially no emissive speech pres-

ent. Since the destruction of so discrete an area has the same effect on emissive speech as does destruction of the entire area concerned with language it is clear that Broca's convolution contains the patterns of emissive speech.

In the last 19 cases there was fair to excellent recovery of emissive speech after it was entirely lost following the lesion. As a general rule the best recoveries occurred in younger patients who survived longer and consequently had more time to train the minor side. In a few instances even aged patients made a recovery. This fact shows the variability in the capacity of the minor areas of Broca to take up the function, i.e., to form engrams of how to move the vocal organs in speech.

The question whether ability to write is lost after a lesion of Broca's convolution may be answered by 9 cases in which the writing was noted in the reports. In cases 1, 7, 8, 9, and 18 ability to write was retained. In cases 6, 10, 11, and 22 it was lost. Here we have five of one kind and four of another. It is safe to conclude that some patients need the engrams of motor speech in order to write while others do not.

TEMPORAL LOBECTOMIES

There are on record 13 cases in which the major temporal lobe was either removed surgically or was damaged to such a degree that it was rendered useless. The available material is as follows:

1. Nielsen (*Textbook of Neurology,* 1941, page 525). A girl aged 3 years was injured in a fall from a swing. She was hospitalized, had right hemiplegia, and was unable to speak for a few days. She began very soon to speak again, gradually recovered from her hemiplegia and was well except for a residual homonymous hemianopia. At the age of 6 years she began to have epileptic seizures and suffered with these until her death at the age of 43 years. In the meantime, however, she developed normally as a left-handed child and learned to read and to write; no one considered her handicapped in her school work.

Autopsy showed the left temporal lobe, the angular gyrus, and the entire occipital lobe replaced with a porencephalic cyst.

This is another instance of unilateral injury or destruction of essential speech areas early in childhood without aphasia.

2. Ingham and Nielsen (1937). G. O. R., a white man of 41 years who had been in the real estate business and had been a musician by avocation, had his left temporal lobe removed for a malignant glioma. He spoke fluently and entirely correctly for a fraction of a minute at a time. He then fell into neologisms and jargon. It was quite possible to understand him because he intermixed his mistaken words with correct ones. When asked where San

Diego was he replied "It's the city down there to it. You've got to have the sodent down there to it." He was unable to recall names (nouns) and sometimes other parts of speech. He understood spoken language for a fraction of

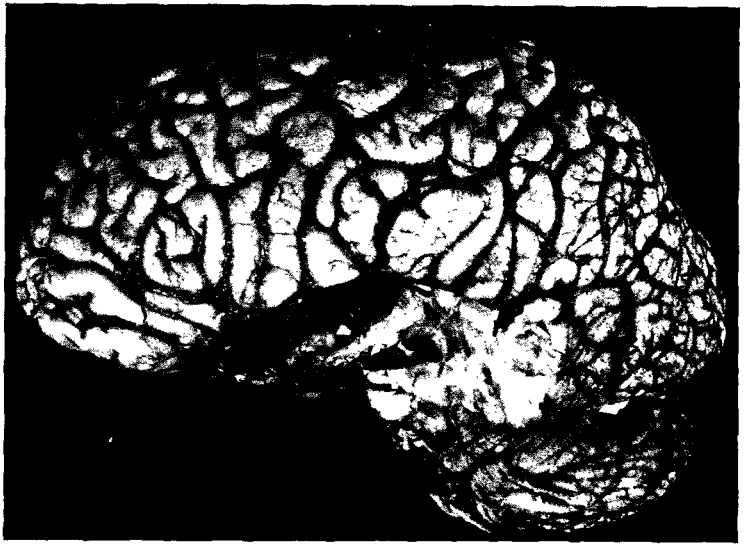

FIG. 17. Case of G. O. R. Dissection of specimen by Dr. R. B. Raney to show as nearly as possible his operation.

a minute each time he was addressed if given a rest of a minute between times. He was able to recognize written language but unable to comprehend its meaning; this was shown by having him sort out typewritten sentences in which there were only slight differences. These he matched perfectly but he failed entirely in trying to place a noun on its corresponding object. His ability to calculate was unaffected as were musical appreciation, musical performance, and reading of music. His capacity to write was badly affected as was his ability to construct words with anagram blocks. He had severe amnesic aphasia and formulation aphasia.

In this case recognition of written or printed matter, calculation, and musical functions both sensory and motor were unaffected by removal of the major temporal lobe. Formulation of language, recall of auditory verbal memories, and recognition of spoken language and writing were seriously impaired and comprehension of written language (except music) was destroyed (Figs. 17 and 18).

3. Nielsen and Raney (1939). R. R., an Italian of American birth aged 53 years, had his left temporal lobe removed for malignant glioma. The line

of incision passed through Wernicke's area but spared the portion between that area and the angular gyrus and also spared area 37 of Brodmann. As the patient was in coma before operation only his condition after surgery can be given.

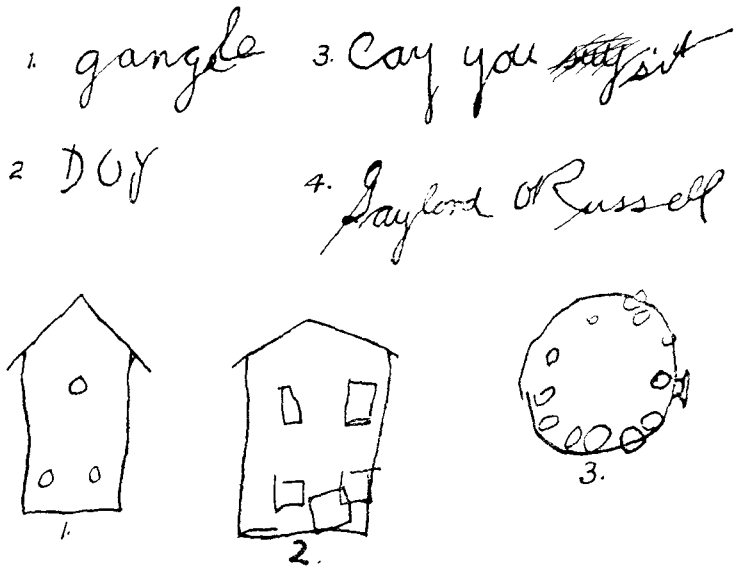

FIG. 18. Case of G. O. R. (From the *Bulletin of the Los Angeles Neurological Society* 2:7, 1937.)

Above: 1. Patient's attempt to write the words orange and bottle. The *ange* is part of orange, and the *le* is the terminal portion of bottle. We first showed him an orange and asked him to write the *name* of it. He tried to draw it. When we objected he said he could not write it. We then showed him a milk bottle and asked him to write the name of it. He said he could not, but when urged, the *gangle* resulted. 2. The result when the patient was asked to write dog. 3. We had written a request for the patient. "Can you sit up?" He could not comprehend but attempted to write it after removal of the sample. (He actually crossed out the *say* much more effectually than here shown. We modified the crossings to render the letters legible.) He never reached the word up at all. 4. The patient wrote his signature which he always did when asked to write anything without specific directions.

Below: 1. The sample. 2. The patient's copy of it. He was careless as though he considered the request puerile. 3. He drew a watch when asked to write the name of it.

Comprehension of spoken language was good for generalities for a short time. He was usually able to answer correctly about a dozen questions, after which he "broke" and suddenly comprehended nothing. The following is a verbatim copy of part of the conversation.

"What is your name?"	"Ross."
"Ross what?"	"Ratania."
"Where do you live?"	"I can't tell you where I live because I don't know where I live."
"Where do you live?"	"In my own house."
"How old are you?"	"About 25." (Actually he was 53.)
"Have you got a son?"	"Yes."
"What is his name?"	"The biggest one?"
"Yes, the biggest one."	"The biggest son is Ross."
"Just like you?"	"Yes, Ross."
"Have you a son named George?"	"George Ratania."
"What is this?" (A dollar bill.)	"A woman." (paraphasia)
"What are these?" (A pair of glasses.)	"They're all right."
"What is the name of it?"	(The answer consisted of only neologistic jargon and after that he understood nothing.)

When he could no longer understand he cocked his ears as though listening to a foreign language. (He did no better in listening to his son speak in Italian than in English.) (Fig. 19.)

In speaking spontaneously he usually produced a few short sentences correctly or with slight paraphasia. If he attempted anything long at first, or even a sentence of a few words after having talked for a minute, his speech became severely paraphasic. When asked "How long have you been in this country?" he answered: "I'm in America all my, all my life. I still live but before I die I go. When I was a young fellow I join the Army, before I join the Army, and I joined the Navy one day and made the American Army under when. But I went to Army my own record I was just make a sweat. When I left the old country I left everything here. The last time I leave my wife Catalina."

His ability to read and to write and capacity to calculate were completely lost.

It is interesting that his ability to formulate language was excellent for a short time just as were the functions of comprehension of spoken language and the capacity for emissive speech. We have no way of determining in such cases which area of Broca (right or left) is functioning.

4. Nielsen (1936). A white man of 50 years came in with acute embolic phenomena. His limited ability to comprehend spoken language is shown by the facts that he showed his tongue on command, raised his hand, and, after repeated requests, raised two fingers. After these efforts he understood nothing. At another time he showed four fingers when asked the number of

children he had (correct). When asked to write Colorado he wrote a C but could not proceed. When asked whether he had a wife he said, "no." (This is a typical performance of a minor temporal lobe.) He spoke at first with

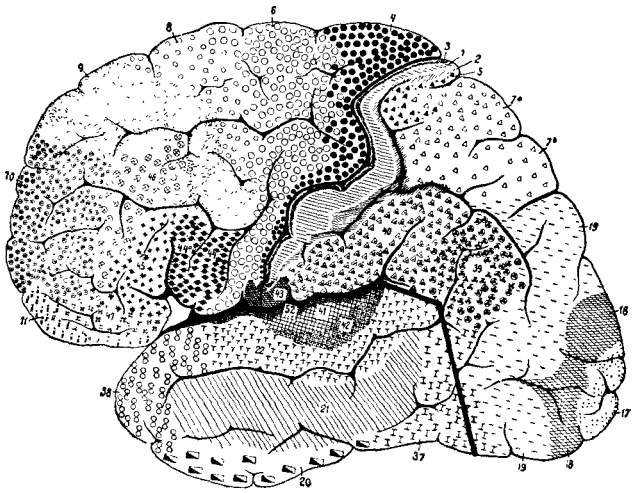

FIG. 19. Case of R. R. Diagram showing line of incision. (From the *Archives of Neurology and Psychiatry*, 42:191.)

paraphasia; later he spoke very little but what he did say was said without paraphasia. He was unable to read or to write (except his name). The period of survival was only three days.

Autopsy showed red softening of the left temporal lobe except the posterior portion (area 37 spared). There was a little damage to the angular gyrus. The transverse gyri had escaped but the temporal isthmus was close to the softening and was edematous.

5. Millitzer (1939). A Mexican woman of 62 years died four years after her left temporal lesion. She was able to understand spoken language to the extent of a few simple statements or questions. She spoke well and without paraphasia. She remained unable to read or to write though she had been able to do so before the vascular lesion occurred. She never regained any ability to calculate.

Autopsy showed complete subcortical destruction of the temporal lobe including area 37 and the angular gyrus. The temporal isthmus was severely affected (Fig. 20). (Reported in the *Bulletin of the Los Angeles Neurological Society*, 1939, page 134.)

6. Sult (1939). A white man of 63 years suffered a small hemorrhage directly into Wernicke's area on the left and died six days later of another (but

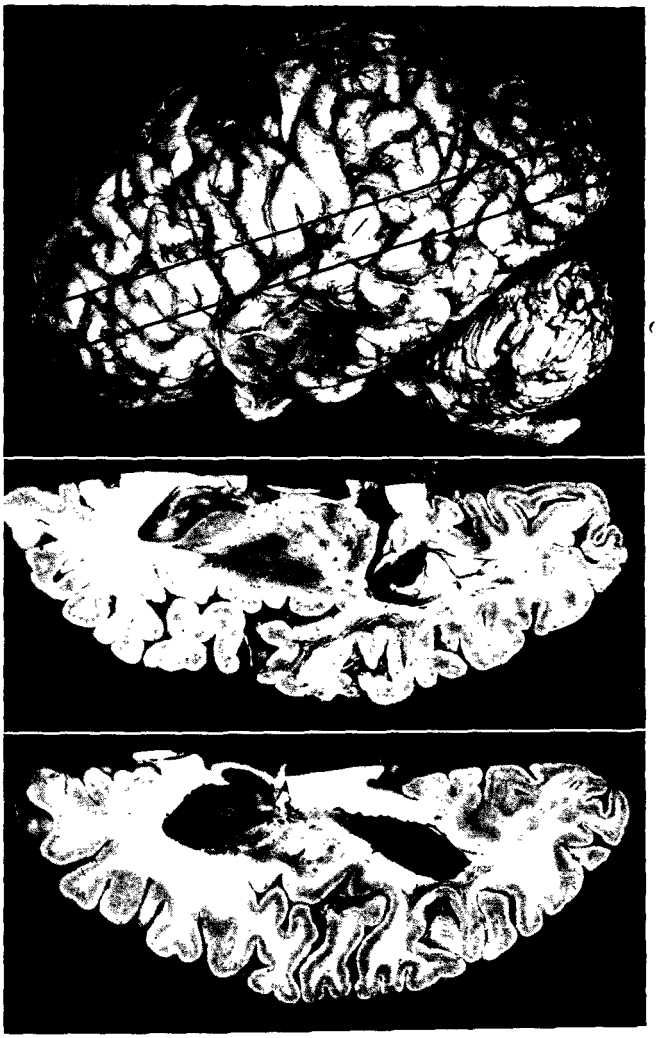

FIG. 20. Case 5 (Millitzer, 1939). The uppermost figure shows a lateral view of the brain on which are drawn lines indicating the levels of sections shown in the other two illustrations. The lower section shows subcortical destruction in the first and second temporal convolutions and an old lesion in the putamen and anterior limb of the internal capsule. The upper section shows an upward extension in the putamen and subcortical destruction in the angular gyrus. (From the *Bulletin of the Los Angeles Neurological Society*, 4:134, 1939.)

massive) cerebral hemorrhage. During the six days of life he understood only two simple spoken commands at a time. He spoke only three words spontaneously yet he was able to read as shown by his selection of words from a

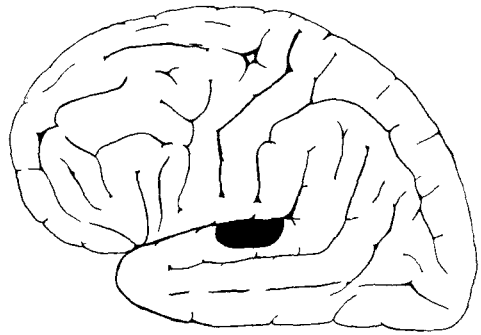

FIG. 21. Case 6 (Sult, 1939). Diagram of the lesion. (From the *Bulletin of the Los Angeles Neurological Society*, 4:134, 1939.)

series. He was asked to select those of something good to eat and never failed. He wrote a few words but could not write propositionally. At autopsy the temporal isthmus was softened (Fig. 21).

This case is of interest in that the angular gyrus remained functioning independently of a destroyed area of Wernicke. (Reported in the *Bulletin of the Los Angeles Neurological Society*, 1939, page 200.)

7. Fox and German (1935). A man of 28 years was subjected to surgical removal of his major temporal lobe at a point 3 cm. behind the junction of the fissure of Rolando with the fissure of Sylvius. It is of interest that twelve days earlier an operation had been performed in which 2 cm. less were removed and that the remaining function after each operation was essentially the same. At the final operation area 37 was not encroached upon.

The patient spoke quite well though with considerable disturbance of language formulation. He understood spoken language imperfectly and with the element of rapid fatigue manifest. Naming of objects, with a few exceptions, was impossible. Music was appreciated and enjoyed. Reading, especially aloud, was badly affected as was writing; both functions rapidly fatigued. The patient improved a great deal during the first year, chiefly in developing more endurance in his receptive language capacity.

8. Bianchi (Henschen case 377). A white man after a cerebral vascular accident survived thirteen months. He had severe paraphasia, introduced syllables and transposed letters. He was able to write but slowly and with errors. He was able to read. Autopsy showed a large area of focal softening

in the left temporal lobe. All of the white matter was destroyed. The anterior portion was normal but it was isolated from the rest of the brain. The posterior portion was normal.

9. Troisier (Henschen case 487; from Wernicke's textbook, Vol. II, page 179). A woman aged 76 years after an apoplectic insult survived six weeks. She was somewhat unclear mentally. Still she spoke at times, always with severe paraphasia except to answer "yes," "no," and "thank you." She did not obey commands or respond at all unless given unusual stimulation. At autopsy it was found that the left temporal lobe was completely cut off by a lesion in the temporal isthmus. Besides, practically the entire lobe was softened.

10. Seppilli (Henschen case 466). A woman of 56 years after a cerebral vascular accident survived one month. While she heard well she always responded with "Quello li, quello si." When she spoke spontaneously she produced severe jargon. At autopsy the entire temporal lobe was essentially destroyed. The white matter was affected so as to leave very little function remaining.

11. Kussmaul and Körner (Henschen case 418). A woman of 63 years, after a cerebral insult, although right-handed, understood spoken language well. She was also able to read printed or handwritten matter and to calculate. She spoke well though slowly and she was able to write. At autopsy an area of softening was found in the left temporal lobe 2½ cm. by 8 cm. except that the portion immediately under the fossa of Sylvius was preserved.

12. Nielsen and Raney (1939). A dentist of 52 years had his left temporal lobe removed (anterior one-half) for tumor. After surgery a hemorrhage occurred at the operative site. Following this episode the patient was unable to speak and he understood little of what was said. In the course of eighteen months he taught himself to speak very well, to comprehend spoken language unless the words came too fast (as over the radio), to read slowly, and to write, although he never acquired rapid and fluent writing. There was a little amnesic aphasia and his language had become stilted. This peculiarity was due to faulty word finding and formulation of language. He died of a neoplastic recurrence; consequently the final anatomical findings did not correspond to those at the time of the clinical study.

13. Raney and Friedman (1942). A young woman of 32 years had her major temporal lobe removed for tumor. The line of incision passed through area 37 of Brodmann and behind the zone of Wernicke. After surgery her comprehension of spoken language was limited to a few simple questions (six to ten according to her state of rest and composure). She had classic amnesic aphasia; she was unable to name objects shown her but invariably selected the correct term when she heard it. She could not talk because of in-

ability to find her words. She was unable to read except a few numerical figures. She was unable to write at all. She recognized all objects and sorted colors without error.

Analysis of the preceding 13 cases.

Of these 13 cases the first was in a child of 3 years. As would be expected there was no indication of aphasia. In case 11 the patient, although 63 years old, re-learned quickly to the point that the only remaining defect noticeable was in her writing. In case 12 for a short time there was no language function. The patient then gradually relearned fairly well in all functions except that he spelled poorly.

In the remaining 10 cases spontaneous speech was fair in 4 and poor in 6. Comprehension of spoken language was fair in only 1 and poor in 9. In 2 cases the patients were able to read well, better, in fact, than they were able to speak or to comprehend spoken language. This is surprising, especially inasmuch as one of them could not write at all and the other very little. In one of the cases the author (Sult) frankly reported it (at my instigation) because of its exceptional character.

Of the two functions, comprehension of spoken language and spontaneous speech, the former is much more severely involved in temporal lobectomy than is the latter. Or, to state the matter positively, the right cerebral hemisphere performed less well in comprehension of spoken language than in emissive speech. This is not strange since the left motor area was intact.

We shall now discuss language formulation and show that that function is severely damaged in temporal lobectomy. In no less than 8 of the 11 cases paraphasia was prominent. In three others, cases 5, 6, and 13, there was no paraphasia but the patients hardly spoke at all; their language function was so poor that they were not even capable of paraphasia. Neither could they name objects but that loss was due to the same functional defect. They, therefore, had completely lost capacity to formulate language. The inclusion of those 2 cases with the others in which paraphasia was present (in which there was disturbed formulation) leaves us with the result that all except the patient who was injured in childhood and the woman of 63 whose right side took over the function of language in its entirety, in all cases in which the formulation had to be done with the minor side there was difficulty with formulation of language.

We are now enabled to make some observations concerning the symptom of paraphasia. In the series of cases of destruction of the entire language area there were 19 cases. One is excluded because the lesion occurred in early childhood and no disturbance would be expected. Of the remaining 18 cases

paraphasia was present in only 2 or possibly 3. In the small series of 4 cases of destruction of the language area but with preservation of the angular gyrus there was no paraphasia. Thus of the 22 cases of complete or nearly complete right-sided function of language there was paraphasia in only 2 or 3 cases. On the other hand in the cases of temporal lobectomy or destruction of the temporal lobe (again excluding the case from infancy) there was paraphasia in all but 4. In these 3 cases it was absent in 1 because of complete function of the right side and in the other 3 it was absent because of failure of the right side (as well as of the left side) for speech and for comprehension of spoken language.

Now let us consider the anatomic difference between the hemispherectomy cases and the temporal lobectomy cases. So far as spoken language (emissive speech) is concerned the difference is that in the former the well trained Broca's convolutions are absent, while in the latter they are present. If we consider the similarity of the two groups of cases anatomically we find immediately that in both groups the major language formulation area is out of function and that the minor one is doing whatever formulation there is.

Adding these equations we find unequivocally that paraphasia results from the presence of an active motor speech area (Broca's region) regardless of which side it may be, but with the minor or incapable language formulation area organizing and supplying the material for Broca's area to emit. We now have the answer to the question, "Why is emissive speech badly affected in quite a number of cases of destruction of the major temporal lobe?" Language formulation must be carried out by the minor side and both Broca's regions are supplied with poorly formulated material.

SUBDIVISIONS OF THE TEMPORAL LOBE EXCLUSIVE OF AREA 37

In considering the functional activity of the temporal lobe there is a fundamental point about the anatomy which must be clearly visualized before one can evaluate clinicopathologic studies of this area. This is the fact that because of the presence of the insula anteriorly and the posterior horn of the lateral ventricle posteriorly the narrow channel of white matter between the two, called the temporal isthmus, forms the essential pathway for functional communication of the temporal lobe with the remainder of the brain.

Through the temporal isthmus go the fibers conveying impulses of hearing from the medial geinculate body (thalamus) to the transverse gyri of Heschl (transverse temporal gyri). Destruction of these fibers leaves the area of Wernicke isolated from incoming impulses. Moreover, destruction of the isthmus interrupts the posterior part of the external capsule through the posterior extremity of which the fibers of the corpus callosum connect the two

areas of Wernicke. Furthermore, since the area of Broca is functionally connected with the ipsilateral temporal lobe and angular gyrus through the fibers of the external capsule via the temporal isthmus, destruction of the temporal isthmus prevents all "priming" of the area of Broca by means of the language formulation zone. The fibers of the optic radiation also pass through the isthmus, for which reason a lesion there interrupts association of the occipital lobe with the thalamus. A destructive lesion of the isthmus, therefore, breaks functional connections between the thalamus on the one hand and the temporal and occipital lobes (except for short U fibers) on the other and thus, on the major side, severely affects the entire speech function.

This point applies to cases especially in which the temporal isthmus is involved. In cases of intact isthmus there is another crucial point which must be kept in mind, namely that the function of a given cortical area can be determined only by means of cases of bilateral essentially symmetrical lesions. This is because in cases of unilateral lesions the contralateral homologous area may take over the function and thus clinically leave no evidence of the lesion.

In the present study it is considered unnecessary to prove the function of the transverse temporal gyri. They serve the function of primary auditory perception. Destruction of one side leaves no deafness because fibers from both cochleae go to both sets of transverse temporal gyri. But destruction of both leaves permanent and complete deafness.

In considering the functions of the first and second temporal convolutions it is important to select cases in which the transverse gyri are preserved at least to the extent of leaving good hearing, because in the absence of adequate hearing recognition of spoken language cannot be discussed. In the first series of cases to be considered now only the first and second temporal convolutions are studied. Nature provides such cases much more readily than it provides those of small isolated lesions. There are 4 excellent cases available with lesions of the first two temporal convolutions and with the isthmi as well as the transverse gyri preserved.

1. Henschen case 3. This is one of the most important cases in the history of aphasia. It was studied clinically and pathologically by Henschen himself. It gave findings which make the case crucial.

From May 1902 to 1904 the condition of the patient was stationary. A woman of 54 years was able to speak on the day following a severe "stroke." She spoke fluently and without paraphasia. She had a left hemiparesis but she was mentally clear and alert; her memory was good; hearing was good and equal on the two sides. There was no tone deafness at all; she understood sounds well. On the other hand she was almost though not absolutely word deaf. She grasped letters and an occasional short syllable and word. This was

shown by her ability to repeat them. She had amnesic aphasia for only a few days after the stroke; there was no paraphasia. She read well and understood what she read. She was able to count, to calculate in her head, and to read numerals. Writing of them was not certainly stated in the report. As to music, she was able to differentiate various instruments and also pitch. She grasped various melodies and was able to follow the leader in singing.

The autopsy showed a most remarkable condition. On the left side there was softening of the posterior portions of the first and second temporal convolutions as well as the anterior portions of the transverse gyri. On the right side there was extensive destruction of the upper portions of the temporal lobe, the first convolution completely, the second almost completely, of the anterior extremity and of the dorsal portion of the third temporal convolution. all this to a depth reaching the internal capsule. There was also subcortical destruction of the third temporal convolution.

Henschen's conclusions are nearly all worthy of citation. His notes referable to the primary center of hearing will not be cited because on those matters all students are now agreed.

"The transverse gyri, which were preserved on the left, must have functioned in perception of acoustic stimuli. Since they were preserved only on the left side and hearing was good on both sides, hearing is bilaterally innervated. The preservation of the posterior section of one transverse gyrus suffices to effect a sharp capacity of hearing. The 'pure' (perceptive) word deafness was caused by the destruction of the posterior section of the first temporal gyrus (and possibly of the second). The hearing center is separate from the word-hearing center. Destruction of the 'word sound center' does not of necessity disturb the internal language of spontaneous speech.[1] The word sound center and the word sense center[2] must also be separate, and the destruction of either one of these centers produces different aphasic symptoms. There must be in the temporal lobe at least three separate centers arranged on different levels: the primary center of hearing, the word sound center, and the word sense center. The word sound center is certainly located in the first temporal convolution. The musical center does not lie in the posterior part of the first temporal convolution since these were bilaterally destroyed in this case."

"The most important conclusion to be drawn is that there are located in the temporal lobe various levels of the psychic centers of different functional capacity and with separate localizations."

Comment. All of Henschen's conclusions are justified but it is necessary to

[1] The area of Broca can utilize the language formulation area without the use of Wernicke's center.

[2] Which is identical with the language formulation area.

remember that other cases may show different symptoms from identical lesions unilaterally placed. For cases to be identical clinically they must be bilaterally identical pathologically.

2. Barrett (Henschen case 373). A man of 45 years after a cerebral vascular accident had complete word deafness. He was also partially tone deaf. In spite of being unable to comprehend any words he spoke very well though at times with paraphasia. He was able to read with good comprehension and to write well spontaneously and from copy. There were some disturbances of calculation.

At autopsy there were residuals of old softening in both temporal lobes. In the left lobe the lower lip of the first temporal convolution was destroyed and the outer one third of the dorsal surface was gone. The greater part of the second temporal convolution (dorsal surface) and the underlying fibers were affected. The transverse gyri were intact. On the right side all of the first temporal convolution and the adjacent angle of the insula showed a cortical defect and the second temporal convolution was involved.

3. Kahler and Pick (Henschen case 417). A woman of 42 years survived six months after a cerebral vascular insult. She had been mentally disturbed for a year. She did not react to sounds except to nod in their direction and she was generally considered deaf. She understood no spoken language. Her speech consisted only of mumbling.

At autopsy the convolutions of the left temporal lobe were greenish-yellow and soft. On the right side all temporal convolutions except the basilar ones were softened.

4. Seppilli (Henschen case 465). A woman of 54 years after a cerebral vascular accident was not deaf but totally word deaf. She lived six months and spoke only disconnected words. She was unable to read or to write.

At autopsy there was found softening of the first and second temporal convolutions of both sides as well as the transverse gyri on the left.

Analysis of the preceding 4 cases.

These 4 cases of bilateral destruction of the first and second temporal convolutions show immediately that, because of preservation of the transverse gyri of Heschl of one side or the other, all were able to hear. Yet none was able to recognize or to comprehend spoken language. In the 2 cases in which the patients were clear mentally (those of Henschen and of Barrett) spontaneous speech was slightly if at all disturbed. Therefore, the area of Wernicke is not essential to the priming of Broca's convolution and is unessential to good spontaneous speech. Both of the cases mentioned also showed good ability to read but neither showed ability to write.

If we subsequently show, as we expect to do, that the angular gyrus is essential to writing but that for its highest function it is usually dependent on the area of Wernicke such a concept is borne out by these cases.

These 4 cases have now demonstrated that in cases of bilateral destruction of the first and second temporal convolutions complete word deafness appears. There is in the literature an excellent case studied carefully by one of the famous neurologists, Arnold Pick, with bilateral destruction of only the second temporal convolutions. It is crucial because there was no word deafness at all. This fact shows that it is the first temporal alone which is essential to recognition of the spoken word. A very brief abstract follows.

Pick (Henschen case 458). A former soldier aged 75 years suffered two cerebral vascular accidents. After recovery he was able at all times to understand spoken language perfectly. He had at first severe paraphasia but that defect later disappeared. Reading is not mentioned but the patient had complete agraphia, being unable to write even his own name.

At autopsy a hemorrhage was found subcortically in each second temporal convolution. On both sides the posterior one-half was affected and the lesion extended partly into the white matter of the first temporal convolution.

Pick comments that from the history it was clear that the lesion on the right side was the more recent and that when it appeared a slight word deafness developed but that this defect cleared. This would show that the right side functioned at least to some extent in word hearing.

DESTRUCTION OF THE LANGUAGE AREA EXCEPT THE ANGULAR GYRUS

The results of destruction of the entire major language area having now been studied, it will be interesting and instructive to study cases which differ from the preceding in having, as the only preserved portion of the language area, the angular gyrus. In other words, we can study angular gyri which are connected only with the calcarine and general sensory areas and might theoretically still function in reading. There are 4 cases available in the literature.

1. Bernard (Henschen case 75). A woman of 49 years awoke with a right hemiplegia and found herself speechless. For two years she was able to pronounce only the words "yes" and "no." For a time she understood spoken language but for only a few seconds at a time and if it was spoken slowly. After six years she learned to speak, comprehend spoken language, and to read and write but she never regained ability to appreciate music.

At autopsy the following were found softened: the pars triangularis and the foot of the third frontal convolution, the entire insula, all of the first temporal convolution and part of the supramarginal gyrus, the head of the

caudate nucleus, the anterior one-third of the thalamus, the claustrum, and the posterior one-third of the lenticular nucleus. The sensory projection fiber system was intact.

2. Touche (Henschen case 339). A woman after a cerebral vascular accident was unable to comprehend spoken language; she repeated what was said (echolalia). Her pronunciation was good and she spoke familiar words with ease. She was unable to read and could not be made to sing.

Autopsy showed softening of the entire left third frontal convolution, the inferior portion of the precentral and postcentral convolutions, the first, second, and third temporal convolutions, and the insula. The angular gyrus was unaffected.

3. Mingazzini (Henschen case 1101). A man of 60 years, left-handed, developed left hemiplegia with aphasia. He spoke little and with paraphasia. He failed to carry out simple orders and he did not reply to questions. He failed to name objects or he gave wrong names. He read words, sometimes without error, usually not.

Autopsy showed complete destruction of the language area except the angular gyrus.

4. Mills (Henschen case 1070). A woman of 51 years after two cerebral vascular accidents had hemiplegia and aphasia. Her spontaneous speech was limited to a few syllables and she understood little of what was said. On the other hand she was able to recognize written words.

At autopsy a large sunken area was found involving the entire sylvian region except the angular gyrus.

Analysis of the preceding 4 cases.

In case 1 the patient lost all language function with the onset of the vascular accident. When she relearned she reacquired all functions. It seems certain, therefore, that the left angular gyrus was unable to carry on after it was isolated from the rest of the language area and that the right assumed the new function in company with the speech area of the right side of the brain. In case 2 the isolated angular gyrus failed to function. In case 3 words were occasionally read even without error. As the angular gyrus functioned better than the area of Wernicke, I believe that the isolated angular gyrus did continue to function to some extent. In case 4 the patient was able to recognize written words at a time when she was unable to speak or to comprehend spoken language; we must conclude that the isolated angular gyrus continued to function but that it was weakened. Paraphasia was not present in any of the four cases.

From this small series of 4 cases we see that in 2 the angular gyrus isolated

from all but the occipital and parietal lobes failed to function; in the other 2 it did function to some extent.

THE LANGUAGE FORMULATION AREA: AMNESIC APHASIA FROM PARIETAL LESIONS

As suggested earlier in this volume I have introduced the concept of a language formulation area, i.e., a more or less circumscribed area of cerebral cortex essential to the correlation of data for linguistic expression of concepts. Inasmuch as a disturbance of language formulation is manifest by paraphasia, paragraphia, or amnesic aphasia the clinicopathologic approach to the problem of delineating an area is the selection of cases of lesions in which the patient showed one or more of these defects during life. But that procedure alone is not enough because similar disturbances may be the result of purely functional inadequacies. In order to define the language formulation area it is necessary to consider the physiology of formulation of language.

Language consists in the elaboration of concepts, i.e., in the putting of concepts into symbolic form for an expression which is comprehensible to our peers. One, therefore, does not speak unless one has concepts to express and the first step in language is recall of the concepts one wishes to express. As to constituents of a concept it is true that any concept which has never been expressed in language is certain to be incomplete because the very name of a concept becomes part of it and the more one says about anything the more is the concept of the thing embellished. However, the unexpressed concept is to be recalled in its crude form before one can begin to talk about it.

As the concept is being recalled one develops, if one does not already have it, an emotional tone regarding it. This must be clear when one knows that all action, and therefore all speech, is emotionally motivated. As one begins to select the name of the concept which one wishes to express one is guided by one's emotional tone toward it. Thus, in speaking of a certain man one may say "man" but one may instead say "scoundrel," "gentleman," "cripple," "beggar," "politician," or any other name according to one's emotional state regarding him. Or, in speaking of an event, one may say "the event" but one may instead say "the outrage" or "the contribution" or "the blessing" or "the travesty." It should thus be clear that before the selection of even the *name* of a concept not only the cortex but also the diencephalon comes into play. In short, the cortex and all of its specialized functioning cannot work without participation of the thalamus or diencephalon. With this point settled we can proceed to a discussion of the cortical function without necessity for constant mention of the thalamic way station.

To take a simple illustration for the subsequent discussion we shall select an apple. The child's first perception of an apple may be assumed to be a

visual one. The engram of the visual impression is stored in area 18 of Brodmann for recognition and another engram subject to visual recall is stored in area 19. When the child first hears the name of the object the sound "apple" is recorded in the area of Wernicke. The visual and the auditory engrams are immediately associated by anatomic engrams into a unit which is an enlarged engram forming the anatomicophysiologic basis of the concept of an apple. For ever afterward the appearance of an apple recalls the sound of its name or the sound recalls the visual image. Later the child is allowed to hold an apple in its hands and the feel of the texture and the weight of the apple form parietal engrams of these sensations united with the visual and auditory engrams to form a still larger physical unit to represent the concept of apple. Now the child can identify an apple by its mere feel and when he feels it the visual and auditory images are recalled. Still later he is allowed to taste it and to smell it. When he eats it he discovers a new sensation in his stomach which also becomes with the other sensations (by means of other engrams) the embellished concept. Throughout life other associations may be formed through botanic study or through ownership of apple orchards so that the concept may never cease to grow. However, no matter to what proportions it grows or how extensive it becomes all of the associated engrams unite to form the concept of apple.

Now, if we limit the diffuseness of the discussion of the concept for the purpose of focussing our attention on the linguistic elements of it we can immediately subtract large portions of the cortex as being unessential to our subject. It is, for example, well known that both frontal lobes of the brain may be removed without destroying the concepts of objects and their relationships. But, if one removes both frontal writing centers without doing any other damage to the brain the patient no longer is able to write the name of the apple. If one removes both areas of Broca alone the patient is no longer able to pronounce the name apple. He has lost that portion of the concept. If one removes both areas of Wernicke alone the patient has lost the memory of the auditory sound of apple and his concept of apple is to that extent restricted. If one removes both angular gyri the patient no longer recognizes the written word apple and is no longer able to recall the appearance of the written word. If one removes both areas 19 of Brodmann the patient can still recognize an apple by sight but he cannot recall the appearance of an apple. If one destroys both areas 18 he can no longer recognize an apple by sight. And, to conclude, if one destroys area 37 of Brodmann the patient cannot recall the name of apple even though his entire concept of the object is otherwise intact. He will say, "I know what it is but I can't say it." And investigation will show that he cannot write the name even though he is permitted to apply every

one of his senses to the object. He can indicate how it is grown, its value in food and in economics, etc., but he has lost all language connection with the object; he knows all except the language symbol for it. He has anomia, amnesic aphasia for the apple. If one shows him the written name his face brightens up and he says, "That's it, apple." This ability is due to intact connection between the angular gyrus (for the written symbol) and Wernicke's area (auditory symbol). A moment later when shown an apple he again fails to recall its name.

Further investigation of such a case will show that not only is the patient unable to recall the name of the object but he will attempt to find the name and use the wrong one. Or, if he attempts to write it he will write the wrong one if he writes at all.

If only the major area 37 is destroyed the patient will use his minor area and will usually talk volubly but incorrectly and, of course, that is the usual clinical picture. Such a condition has been described as agrammatism—the patient's sense or knowledge of grammar is lost.

In this discussion the essential area concerned with the language formulation has been spoken of as area 37 of Brodmann. This has been done for convenience but clinical cases have shown that the area varies considerably in different individuals. In a few cases relatively small lesions in the second and in the third temporal convolutions have given rise to the syndrome of amnesia aphasia. This apparent uncongruity seems to depend on the fact that various individuals require larger cortical areas for their correlations than do others. Temporal lobectomies in which the line of incision was made anterior to area 37 have invariably caused amnesic aphasia because area 37 is thus cut off from its engram connections. Lesions of the temporal isthmus invariably cause the syndrome because the entire temporal lobe is thus isolated. Lesions of the parietal lobe have in a number of reported cases caused amnesic aphasia. These I have gathered and studied as follows:

1. Banti, 1886 (Henschen case 580). *After cerebral vascular accident the patient was able to speak and understand spoken language but had amnesia aphasia. She was unable to name objects or coins but recognized their use and value. Autopsy showed a lesion of the inferior parietal lobule and lenticular nucleus on left.*

An illiterate woman of 75 years suffered an apoplectic seizure with right hemiplegia and loss of consciousness. Her intellectual faculties returned but there was some difficulty with speech. She had a clear amnesic aphasia shown by her inability to name objects though able to indicate their use and repeat their names. She was also unable to name coins but knew their value. She could express herself otherwise.

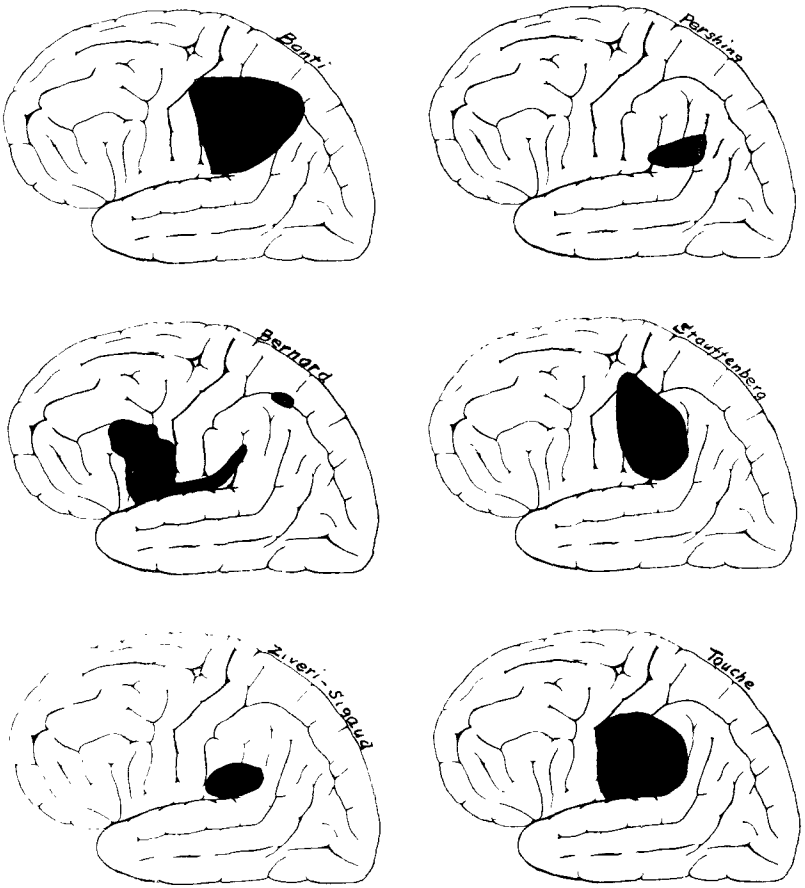

FIG. 22. Diagram of lesions in cases of amnesic aphasia from parietal lesions. These crude Block diagrams have all been drawn on similar lateral views for ease of comparison. In some instances the illustrations of the authors have been imitated, in others drawn from verbal description. In no case is the author cited to be considered responsible for these illustrations. They are intended to convey only an idea of the general site of the focus. In some cases the lesions were cortical, in others subcortical. For accurate description of the actual anatomic changes the case abstracts should be consulted. For ease of reference the name of the author has been lettered on each illustration.

Autopsy showed a gelatinous softening which occupied the middle third of the postcentral convolution and the inferior parietal lobule extending back across the supramarginal gyrus but hardly affecting the angular gyrus. The lenticular nucleus and internal capsule were softened.

2. Bernard, 1885, case 6 (Henschen case 74). *After a cerebral vascular lesion right hemiplegia and motor aphasia. Gradual recovery but with residual difficulty in speech. No auditory verbal agnosia, some degree of alexia. Agraphia. Amnesic aphasia. Autopsy showed lesion of F3 frontal and parietal opercula and slightly of the supramarginal gyrus.*

A widow of 45 years after a cerebral vascular accident had a right hemiplegia without disturbance of sensation. She understood spoken language and after gradual recovery from motor aphasia repeated well. She read the titles and names of authors of several scores of music and read several sentences in a journal but certain words escaped her. She expressed herself faultily because of difficulty in finding her words. She said, characteristically, "I know. I can't say it." She did, however, find the names of the majority of objects. She was unable to write with the left hand. (Fig. 22.)

Autopsy showed a lesion of the left hemisphere which affected the superior part of the insula, the third frontal convolution, the lower part of the parietal and frontal opercula, and a small portion of the supramarginal gyrus. There was a separate small plaque at the interparietal sulcus but no lesion in the temporal lobe.

3. Ziveri, 1917 (Henschen case 703). *Only amnesic aphasia and paraphasia demonstrated. Autopsy showed focal subcortical lesion in the inferior left parietal lobule.*

A man of 80 years after a cerebral vascular accident answered questions with paraphasia, and repetition was at first defective, later much better. He was unable to name objects shown him. He understood always simple questions. The patient was illiterate and for this reason no form of aphasia except the amnesic aphasia and paraphasia could be demonstrated to result from the lesion.

Autopsy showed a small circumscribed lesion located subcortically in the left inferior parietal lobule. Its anteroposterior extent was $2\frac{1}{2}$ cm. and it reached the ventricle without rupturing into it.

4. Sigaud, 1887 (Henschen case 686). *Word deafness with marked amnesic aphasia and paraphasia. The patient was able to read but not to revisualize words or write.*

A woman of 77 years was aphasic for two years. She was able to read but could not revisualize words and could not write except a little on dictation.

She was able to copy. There was considerable word deafness. The most marked defect was amnesic aphasia and paraphasia.

Autopsy showed a small circumscribed lesion of the left supramarginal gyrus (the size of a small nut) which affected the angular gyrus very little.

5. Pershing, 1900 (Henschen case 662). *As permanent residuals of cerebral vascular accident patient showed amnesic aphasia and jargon aphasia with agnostic alexia, and total agraphia. Autopsy showed a small circumscribed lesion in the posterior parietal region affecting probably to some extent the angular gyrus and undermining the supramarginal.*

A man of 45 years suffered a cerebral vascular accident with transitory loss of consciousness. For seven weeks he was word deaf. He had a severe amnesic aphasia and jargon aphasia. He was able to read only a few short words and had no understanding of even those. He was totally unable to write even on dictation.

Autopsy showed a small lesion immediately above the sylvian fossae 6 cm. back of its horizontal and vertical branches in the form of an irregular cone. the base being an oval area 1.8 cm. vertically, .8 cm. wide, the long axis 2.5 cm. to about the same depth at the floor of the sylvian fissure. Sections were not reported.

The autopsy findings lack study of sections and are as a whole poorly described. The lesion certainly, according to the description, undermined the supramarginal gyrus, and with the history of word deafness lasting seven weeks, one can assume that Wernicke's area was closely encroached upon. The lesion seems to have severed the temporoparietal connections.

6. Stauffenberg, 1911 (Henschen case 688). *Complete alexia and agraphia with severe paraphasia and amnesic aphasia. Autopsy showed a large lesion of the left parietal lobe affecting also the angular gyrus.*

A woman of 71 years after a cerebral vascular accident was not mind blind or demented. Comprehension of spoken language was quite well retained. She understood commands but paraphasia was severe and amnesic aphasia marked. She was unable to name objects which were shown her and was unable to recognize words. She had complete agraphia and bilateral apraxia. She spoke a great deal.

Autopsy showed a large area of softening in the left parietal region affecting almost the entire lobe and to a slight extent the angular gyrus.

7. Touche, 1899, case 6 (Henschen case 330). *Amnesic aphasia and temporary alexia. Autopsy showed softening of the left inferior parietal lobule.*

After a cerebral vascular accident a woman of 49 years had amnesic aphasia and could not recall the name of her street or (name of) her home but spoke with only slight dysarthria. She never actually lost recognition of written

words but for a time she could not read. She could not learn to write with the left hand, except that she wrote correctly letters and figures. She never had word deafness or paraphasia.

Autopsy showed softening of the inferior half of the postcentral gyrus, and the inferior parietal including the supramarginal gyrus, but the lesion did not affect the temporal lobe or the angular gyrus.

Analysis of the preceding 7 cases.

This presentation shows immediately that amnesic aphasia may be due to a lesion of the parietal lobe. Closer scrutiny shows that the site within the parietal lobe which is common to all the cases is the supramarginal gyrus. Further, the impingement upon the posterior portion of the superior temporal convolution is immediate in every case. In harmony with this there was disturbance of writing in every case in which this element could be studied (there were two illiterates). There was disturbance of reading in all cases except the one of Sigaud. However, anatomic data are not sufficient to determine the finer points. The lesion in the supramarginal gyrus potent to cause amnesic aphasia impinges on the temporal isthmus and thus interferes with association of the entire temporal lobe.

Lesions of the angular gyrus have also been studied. Anatomic consideration of such cases shows clearly that the angular gyrus can hardly be removed surgically, and deep lesions of it cannot occur, without interfering with the temporal isthmus. The explanation of the occurrence of amnesic aphasia in some cases of angular gyrus lesions is therefore the same as for parietal lesions.

Now concerning cases of isolated lesions in area 37 we find that such instances are difficult to find in vascular cases. But the type of case in which the symptom of amnesic aphasia occurs par excellence is otitic abscess of the temporal lobe. Such abscesses typically extend upward directly into area 37 from the middle ear. The large series of cases gathered by Henschen, by Blau, by Sonntag (1928), by Piquet (1931), and many personally observed leave no doubt on this score.

THE TEMPORAL ISTHMUS AND ITS CLINICAL SYNDROMES

The temporal isthmus is a structure which, while well known to neurologists, is accorded little attention. It is seen most characteristically in a horizontal section of the cerebrum and appears as a narrow channel of tissue between the posterior horn and the extreme posterior part of the inferior horn of the lateral ventricle and the posterior border of the insula. It measures from 10 to 15 mm. across and is in height nearly equal to that of the thalamus. Its length is ill defined but for practical purposes may be considered as 1 cm. It is

bounded posteriorly and inferiorly by the ventricle; laterally it merges with the white matter of the temporal lobe; mesially it is bounded by the posterior limb of the internal capsule and the thalamus. Its anterior and posterior sur-

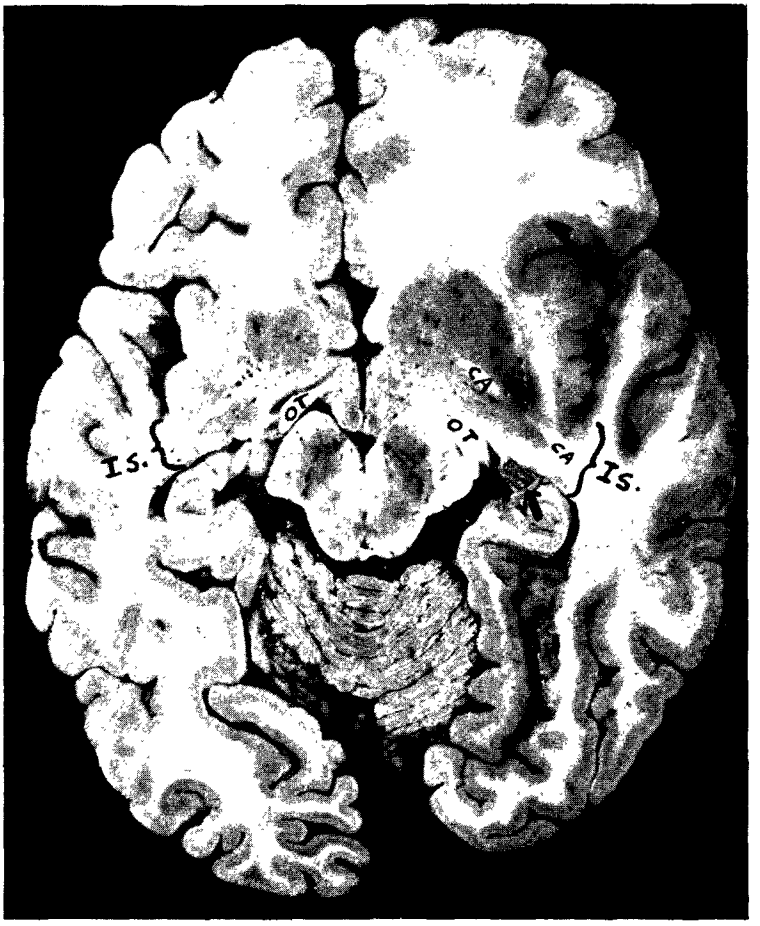

FIG. 23. Horizontal section of brain close to the inferior edge of the temporal isthmus (*IS*). *OT* optic tract, *CA* commissura anterior. *Arrow* indicates lateral geniculate body on the right side.

faces slope from above downward and forward (Figs. 23, 24, and 25). Its clinical importance rests on the occurrence of a syndrome of its lesion for each side, one for the major, one for the minor side of the brain.

The artery of supply of the isthmus is the anterior choroidal, but dissecting hemorrhages beginning in the external capsule may extend backward into the

isthmus. In the cases due to neoplasm one can be certain that the lesion is unapproachable for surgical intervention.

The isthmus is essentially a cross road of fibers; the only nuclear masses

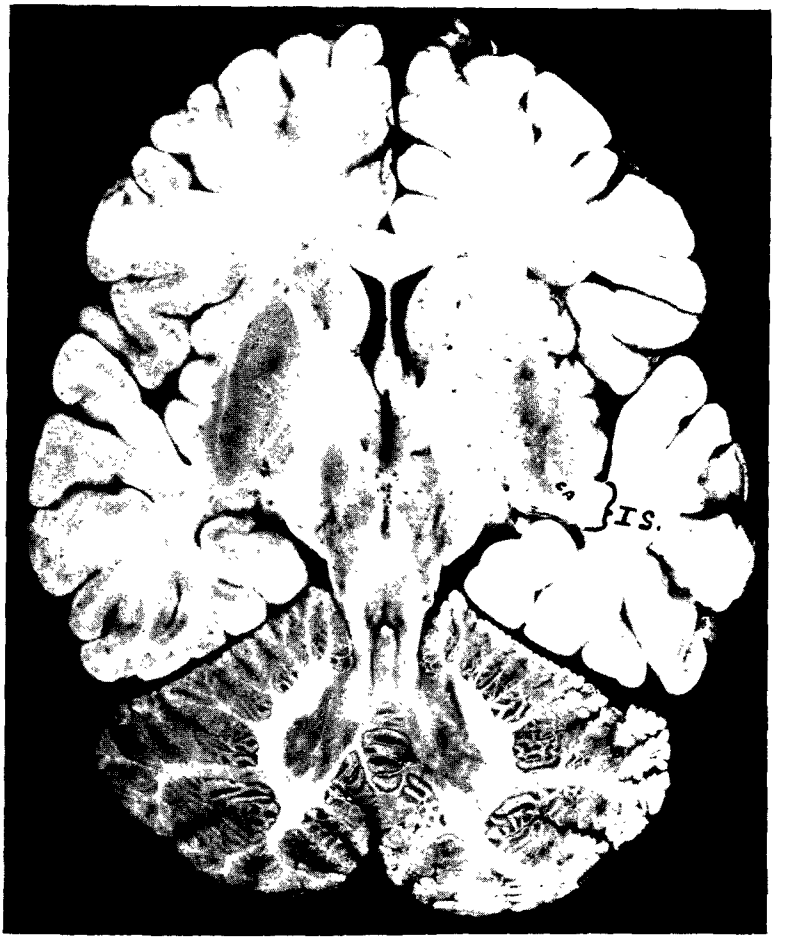

FIG. 24. Oblique section of brain through both superior cerebellar peduncles and both red nuclei. *C.I* commissura anterior, *IS* temporal isthmus.

lying directly in it being the tail of the caudate nucleus, the posterior extremities of the claustrum and putamen, and part of the amygdaloid nucleus. A lesion of these structures does not give recognizable symptoms.

The most important fiber tracts passing through the temporal isthmus are as follows:

1. The optic radiation as it makes its way from the lateral geniculate body

to the borders of the calcarine fissure. The lower portion sweeps forward into the temporal lobe before it goes backward (Flechsig's, Meyer's, or Cushing's loop).

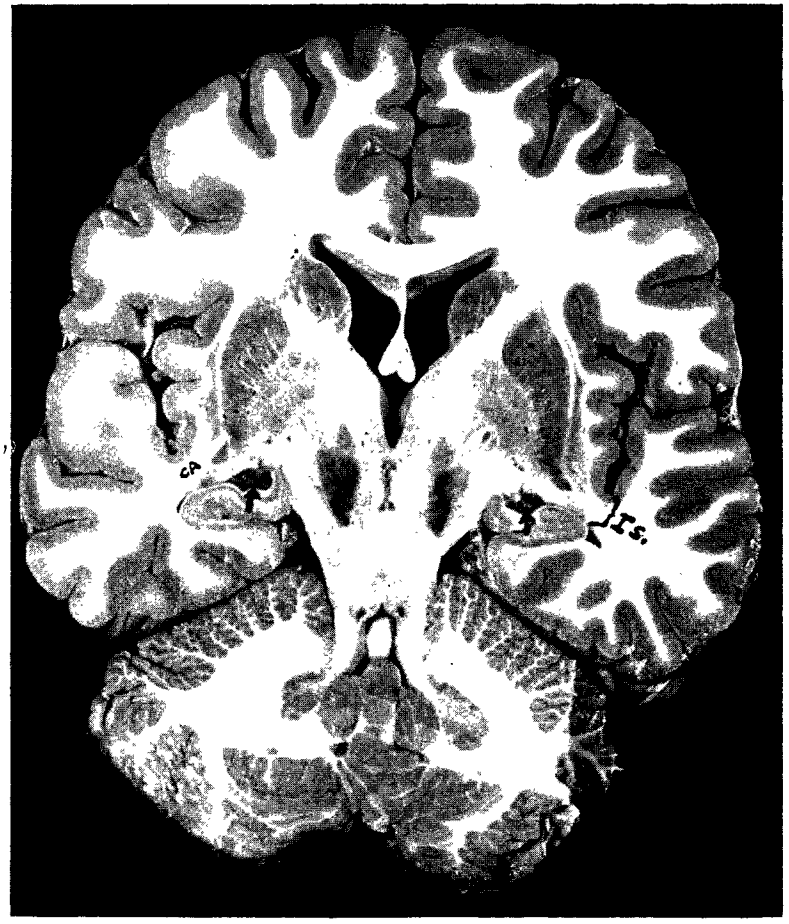

FIG. 25. Oblique section of brain through both lateral geniculate bodies (*arrows*) and both dentate nuclei. *CA* commissura anterior, *IS* temporal isthmus.

2. The auditory fibers from the medial geniculate body to the transverse temporal gyri.

3. Fibers of the anterior commissure to the second and third temporal gyri and to the amygdaloid nucleus. Those from the temporal gyri connect (in all probability) homeotopic cortical areas. Those from the amygdaloid nucleus go at least in part to the lateral mammillary nucleus.

4. The fibers of the posterior thalamic peduncle uniting (according to Rose) the first and second occipital convolutions, the cuneus, the lingual, fusiform, angular, and supramarginal gyri with the pulvinar and the bordering region of the thalamus.

5. Fibers of the corpus callosum uniting homeotopic posterior areas of the first temporal convolution.

6. Fibers from the parietal and occipital lobes to the cerebral peduncle, in the interests of reflex control of the extraocular nuclei.

7. Parts of the fasciculus arcuatus, fasciculus uncinatus, and the inferior longitudinal fasciculus.

8. The posterior extremity of the external capsule and part of the tapetum.

A lesion of the optic radiation, of course, causes homonymous hemianopia. But a lesion, even complete, of the auditory fibers of one side causes no perception deafness in either ear. Lesions of the amygdaloid-mammillary connections may possibly give rise to outbursts of rage but this cannot be stated dogmatically as yet.

On the other hand a lesion of the thalamoparietal peduncle of the minor (usually right) side causes in the great majority of instances delusion of the body scheme. This symptom taken with hemianopia establishes the site of the lesion if only one lesion is present.

On the major side the symptomatology is most striking. As a lesion of the temporal isthmus severs practically all long association connections of the temporal lobe with the remainder of the hemisphere such a lesion causes practically complete aphasia, the global aphasia of the older writers.

The clinical syndrome of a lesion of the major temporal isthmus.

The syndrome recognizable clinically from a lesion of the major side varies somewhat with the pathologic nature of the lesion, whether vascular or neoplastic. If the lesion is hemorrhagic or embolic, the patient is suddenly affected with severe cerebral symptoms. If the anterior choroidal artery is thrombosed instead of being ruptured the results are less devastating but nevertheless severe. A benign neoplasm brings about the symptoms so slowly that the patient recognizes only a gradual diminution of the function of language. Later, pressure symptoms are added.

Hemianopia is a constant symptom. In an acute case this is at times difficult to demonstrate due to the patient's inability to speak; one must rely on one's own acumen in detecting lack of reaction to visual stimuli coming from the opposite side. The factor of inattention is always a problem because a lesion so close to the thalamus, in fact separating the thalamus from the temporal and occipital lobes, reduces the patient's ability to focus his attention. If the

examiner happens to stand on the hemianopic side the loss of attention is apparently even greater, as the patient cannot see him.

Why a patient with an acute lesion of the temporal isthmus suffers from a severe aphasia is probably dependent not only on the disorganization of the language function but also on the factor of inattention. One must keep clearly in mind that the performance in the sphere of language of which any patient is capable after a cerebral lesion depends on the remaining language mechanism, not on the portion destroyed. In the case of an isthmus lesion, there is no actual destruction of any of the cortex concerned with language. The suggestion therefore arises that the patient might be able to carry on better than if cortical areas were destroyed, e.g., the area of Wernicke, the area of Broca, or the angular gyrus. The explanation of the greater disturbance in a lesion of the isthmus is not difficult to find.

In the first place the cortical zone of primary auditory perception (in the transverse temporal gyri) is out of function because it is separated from the medial geniculate body. The area of Wernicke (immediately lateral to the transverse gyri) in the superior temporal convolution is, therefore, to be stimulated, if at all, from the opposite area of Wernicke. But the fibers of the corpus callosum are interrupted by the lesion in the temporal isthmus and if we consider that the impulses might cross via the anterior commissure we are reminded that that structure is also interrupted by the same lesion. All recognition of spoken language must, therefore, be mediated by the minor area of Wernicke.

The major area of Broca is not affected by the isthmus lesion. Why cannot it, then, function? Why does the patient not speak? This question is answered by a consideration of how Broca's area functions. In it are located engrams of how to co-ordinate the muscles of speech to produce words. But such co-ordination cannot function unless language is formulated. In other words, Broca's area must be supplied with the material of language, and the formulation of concepts into language goes on in the posterior temporal region, a large area including the posterior portion of the first, second, and third temporal convolutions as well as the area 37 of Brodmann. This entire area of the major side is unable to communicate with Broca's area because of the isthmus lesion. The major area of Broca is, therefore, functionless unless supplied with internal language formulated on the minor side.

The next question is, why cannot the patient write? This leads to a consideration of the writing mechanism. The writing mechanism involves the intactness of an area for language formulation, intactness of the angular gyrus for revisualization of the written words, and communication between the angular gyrus and Broca's convolution or the frontal writing center.

The frontal writing center is organized like Broca's convolution. It cannot write, any more than Broca's convolution can speak, without being supplied with formulated language.

The connections between the language formulation area and the angular gyrus are not broken by the isthmus lesion, but the connections between the angular gyrus and Broca's convolution, and between it and the frontal writing center are broken because the external capsule is interrupted in its posterior portion. Besides, writing is the most highly evolved of the language functions and is, therefore, easily interrupted.

Why cannot the patient read? Reading involves not only recognition of words, but also their interpretation. The angular gyrus of the major side would have to receive its impulses from the minor occipital lobe because of the hemianopia, usually quite possible. It can, therefore, probably recognize written and printed words, a function of which it is usually capable without association with the area of Wernicke. But the angular gyrus is rarely capable of interpreting the significance of written or printed matter without association with the area of Wernicke because auditory memories are essential to interpretation of many words. For example, the written statement, "They read the manuscript," is not clear unless one knows the sound of the word "read." The angular gyrus is, therefore, out of function for reading, and the patient must depend on the minor angular gyrus (a poorly trained structure).

When one then recalls that the left thalamus is dissociated from the temporal and occipital lobes by the lesion, and that, therefore, attention is poor, one can readily see that the entire function of language is severely affected.

To all this difficulty, in cases of hemorrhage, the patient usually has blood in the cerebrospinal fluid because the isthmus is so small that a hemorrhage into it frequently breaks through into the lateral ventricle. Rigidity of the neck, headache, Kernig's sign, and rise of temperature are usually added to the picture.

The clinical syndrome of a lesion of the minor temporal isthmus.

A lesion of the minor temporal isthmus is usually more easily recognized than is one of the major side because the patient co-operates much better in the examination. Beside the left homonymous hemianopia anosognosia or delusion of the body scheme is usually seen.

Anosognosia of Babinski is the syndrome of lack of recognition by the patient himself, of his hemiplegia. The symptom is usually seen in lesions of the temporal isthmus, involving, or situated very close to, the thalamus. The exact mechanism is not understood, and we are unable to explain why some

patients have it and others do not from an identical lesion. Of course, the lesion must extend forward far enough to produce the hemiplegia also, for which reason it does not occur in lesions confined to the temporal isthmus. There is only one exception to this, reported by Olsen, in which the lesion causative of the hemiplegia was a cortical one. There were two lesions in the case.

Delusion of the body scheme is not necessarily associated with hemiplegia. The isthmus lesion apparently causes the syndrome by interrupting the portion of the posterior thalamic peduncle concerned with proprioception referable to the opposite side of the body, usually only the arm.

Unless the patient volunteers the information that the arm (usually the left) is no longer present or that it feels as though it were absent, one does not discover the delusion or illusion except by inquiring for it. The examiner asks the patient to put his right hand on his left. He cannot locate it. The examiner then closes the patient's eyes and holds the patient's left hand up within the line of vision of the sound field and asks the patient what "this object" is. He replies that it is the doctor's hand. A colored patient even believes that the colored hand belongs to the white examining doctor. The illusion or delusion is usually recovered from in a few days, but it has been known to remain for as long as five years.

If only one lesion is present, the syndrome of left homonymous hemianopia with delusion of the body scheme is pathognomonic of a temporal isthmus location. The delusion alone can result from a cortical lesion of the supramarginal gyrus.

THE QUADRILATERAL SPACE OF MARIE

In 1906 Pierre Marie offered the startling concept that there was no such thing as Broca's aphasia, but that the condition described as such was Wernicke's aphasia plus anarthria. He drew a simple scheme on the horizontal section of a left cerebral hemisphere by placing two parallel lines perpendicular to the sagittal plane and intersecting the anterior and posterior extremities of the insula. His dogma was that lesions anterior to the two lines did not provoke dysarthria or aphasia; lesions in the quadrilateral space caused anarthria; lesions behind the two lines caused aphasia. The area in which lesions were supposed to produce anarthria has come to be known as the quadrilateral space of Marie.

The important anatomic structures in the quadrilateral space, that is to say, the structures which might be concerned with motor control of the bulbar nerves or with language, are the basal ganglia, the internal capsule, the external capsule, and the insula. It is too well known to necessitate discussion that the

anterior limb of the internal capsule contains projection fibers from the precentral gyrus to the pons and medulla for control of tongue and larynx. These tracts, however, are not concerned with speech except insofar as they are mechanical conveyors of impulses to the effectors. A lesion of them affects the innervation of the muscles of speech and swallowing and on the major side will cause dysarthria or anarthria until the minor side takes over the lost function. During this period of dys- or an-arthria the patient knows clearly what he wishes to say; he is merely unable to effect motion of the speech organs until he can send the impulses down from the minor area of Broca and the precentral gyrus or send them across the corpus callosum from the major side. This, then, outlines the function of the internal capsule in speech and the pathogenesis of anarthria when it occurs from a supranuclear lesion.

The thalamus is concerned with attention and with co-ordination of intercortical impulses. Destruction of the thalamus disrupts cerebral function in general and consequently the function of language, but language does not suffer out of proportion to other cerebral functions.

The function of the corpus striatum in language is not so easily disposed of. Henschen had great difficulty in deciding its function and vacillated back and forth to the end of his study. Probably his illness accounts for the defect. In general he ascribed some function to the lenticular nucleus in articulation and considered a lesion in it capable of producing dysarthria. Yet, when he studied the case of Bonfigli which follows, he wrote, "Almost unique case of bilateral (complete?) destruction of the lenticular nucleus without symptoms." When he studied the case of Constantini (2, following) he wrote, "Here were bilateral foci in the putamen without dysarthria (see also Constantini's cases II and III where nothing is mentioned about dysarthria and in spite of this foci were present in the putamen, caudate nucleus, etc.)" In discussing the case of Liepmann (3, following) he commented, "Seems to show the lack of significance of the lenticular nucleus in speech. The excessively brief report does not afford an answer to the question, What is meant by the expression 'good speech'? There was no dysarthria." Abstracts of those three and report of one of our own cases follow.

1. Bonfigli (Henschen case 781). A man aged 50 years was under observation four years during which time he did not show any signs whatever of disturbance of speech, paralysis, or sensibility—in short nothing to indicate any focal cerebral lesion. Autopsy showed symmetrical old foci of softening which had destroyed practically the entire lenticular nuclei. He died of a cerebral hemorrhage.

The original account says, "Focolai simmetrici di antica data, i quali avevano distrutto quasi totalmente ambedue i nuclei lenticolari . . ." Henschen in

commenting on the case took the precaution to mention that the totality of destruction was not certain. Yet he considered it a crucial case showing that the lenticular nuclei were not concerned with language.

2. Constantini (Henschen case 859). A man aged 68 years showed disturbance of memory and limited "intelligence," but there was no trace of dysarthria or of aphasia. Autopsy showed on the right side a small focus of softening in the ventral margin of the putamen. On the left there was a loss of substance in the same location as on the right, but to a much higher level and extending to the external capsule. There were foci of softening in both thalami.

3. Liepmann (Henschen case 1018). The patient after a cerebral vascular accident suffered for a few days with mutism. Then, according to members of the patient's family, there was some unclearness of speech and difficulty with word finding. Four years later a second cerebral vascular accident occurred with transient difficulty with finding of words after which the patient was under observation fifteen months to death. During that time there was never any disturbance of speech. Autopsy showed bilateral rather large lesions in the lenticular nuclei themselves. According to the illustrations the external capsules were intact.

4. Nielsen and Friedman (1942). *A man with two attacks of left hemiparesis and one of right hemiparesis showed no aphasia at any time. Death a week after study due to thrombosis of the right middle cerebral artery. Autopsy showed thrombotic softening of both putamina and right angular gyrus.* (Los Angeles County Hospital, No. 736–134.)

W.M.D., a white man of 67 years, was admitted Oct. 7, 1940 semicomatose and without history. His blood pressure was found to be 210 systolic and 130 diastolic; the pulse rate was 84 per minute and the temperature 98 F. Except for the usual signs of hypertensive heart disease there was nothing in the general physical examination worthy of note. Neurologic examination showed the cranial nerves to be normal except that the pupils were small and without reaction to light. In the upper limbs the deep reflexes were increased on the left, but the right knee jerk was exaggerated, and the achilles tendon reflexes were equal. Babinski signs were present bilaterally.

On the following day the patient's wife gave the history that two years before his admission he had had a "stroke" affecting the left limbs, one year before a similar attack affecting the right ones, and two weeks before an attack of left hemiplegia again. In all of the previous attacks there had been no disturbance of speech and the patient had recovered well each time from his paralysis.

Examination on the second day in the hospital showed the patient conscious

and clear mentally. He had some dysarthria but no aphasia. He understood commands and expressed himself clearly. He was even able to read and write. He had clear spinal fluid under a pressure of 210 mm. of water and it was normal in every way. On the seventh day he died of thrombosis of the right middle artery.

Autopsy showed, in addition to the findings of hypertensive heart disease, some extremely significant cerebral softening. Both putamina were softened, the right lesion being considerably older than the left one. There was also evidence of old thrombosis in the right middle cerebral artery, especially the angular gyrus branch and the terminal portion of the posterior temporal branch. A few small lesions in the right thalamus are hardly worthy of note. The last thrombosis of the right middle cerebral artery was too recent to have caused softening. (See Fig. 26.)

The appearance of the lesions would indicate that the one in the left putamen and the ones in the right temporal and occipital regions were old and were probably caused by the episode of a year before. The lesion in the left putamen, on the other hand, was not more than two weeks old and to it, through edema of the internal capsule, must be ascribed the right hemiparesis which occurred at that time. It is to be especially noted that the fibers of the external capsule were preserved. That is, the structure through which pass the fibers of association between the angular gyrus and the frontal speech region (Henschen, 1920–22).

In these cases there can be no question of vicarious function of one side for the other because both sides were affected in the same way. We can, therefore, concluded that the lenticular nuclei are not concerned with speech.

With regard to the cortex of the insula and to the external capsule, clinical cases with autopsy findings are not available for separation of the function of the two structures. Certain cases suggest that lesions of the insula without involvement of the external capsule do not cause aphasia. In studying cases of destruction of the external capsule it is necessary to have cases of relatively long standing because in the acute ones there is complicating pressure or edema of the internal capsule with resulting hemiparesis. In the literature are found 6 such cases to which we can add two of our own. The case abstracts follow.

5. Raymond (Henschen case 1191). A woman aged 68 years, suffering with cancer, developed mental derangement and visual hallucinations, then paraphasia illustrated by her calling a fork a plate. She was able to repeat words but a minute later unable to say them again. There was no paralysis. At autopsy there was found a yellowish area on the convolutions of the insula 3 by 4 cm. on the surface and this was 3 mm. deep. It did not affect the corpus striatum.

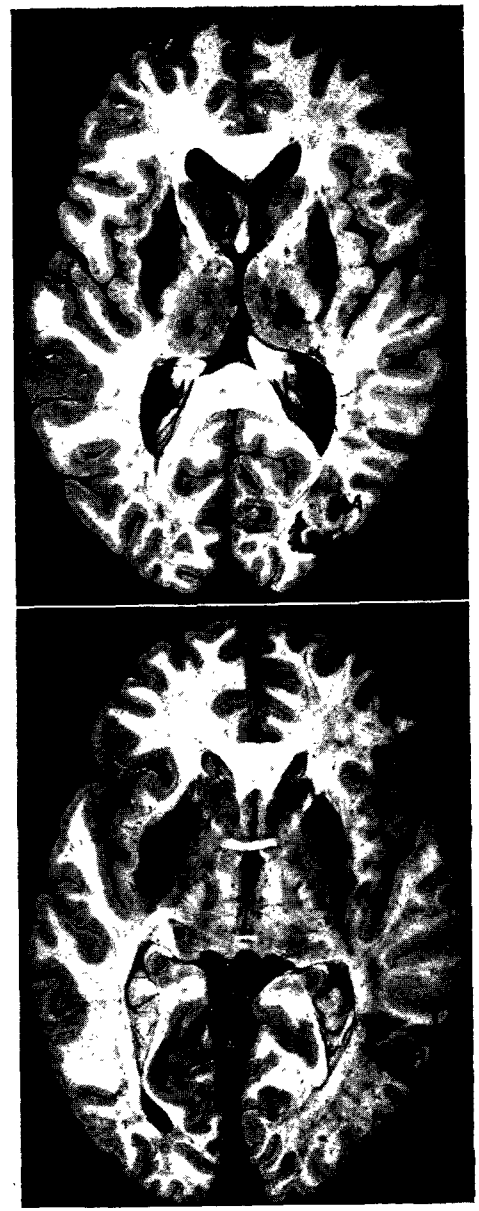

FIG. 26. Case of W. M. D. The upper figure shows softening in both putamina (slight on the right side), a little group of lesions in the right thalamus, and a lesion in the right occipital cortex. The lower figure shows more complete involvement of the putamina and one in the right temporo-occipital cortex.

6. Sollier (Henschen case 1248). A man aged 72 years had a right hemiplegia for sixteen years at the onset of which he had paraphasia. Twelve years later he suffered a second stroke followed by almost complete aphasia. At autopsy there was found an old hemorrhagic focus which had destroyed the external capsule and a part of the claustrum. A symmetrical lesion was found on the other side of the brain.

This case shows that the paraphasia which developed from the first stroke was mediated by the right side. Hence the first lesion put the major side out of function as far as co-ordination between Wernicke's area and Broca's convolution was concerned.

7. Souques (Henschen case 1251). A man aged 60 years had had a hemiplegia and motor aphasia for four years. He carried out spoken commands, but he responded to all commands with the same few syllables. Repetition was slightly less disturbed. Spontaneously he was able to write only his name. Writing was poorly executed with the left hand; copying was slow and poor. He did not read to himself as well as he comprehended spoken language. Autopsy showed no lesion of the cortex. There was an old hemorrhage between the insula and the lenticular nucleus on the left side. This had destroyed the claustrum and the external capsule and had affected the posterior portion of the lenticular nucleus. The gray matter of the insula was intact as was the third frontal convolution. On the right side there was a recent hemorrhagic focus in the external capsule destroying also the lenticular nucleus and the posterior limb of the internal capsule.

8. Touche (Henschen case 1279). A man of 68 years with a right hemiplegia understood well all that was said to him and carried out orders. Spontaneous speech was completely lost. He responded with the one word "yes" and with grunts. As for reading he recognized his name and "Paris." He died after five years. At autopsy the only lesion was a hemorrhagic cavity which had destroyed the left external capsule.

9. Touche (Henschen case 1280). A man aged 44 years was hemiplegic for eight months. There was no trace of word deafness but there was paraphasia. He was able to read. His speech was characterized by dysarthria, hesitancy, and incomprehensible words. He was able to write spontaneously only his own name, but he could copy. At autopsy the cortex was found normal but there was a linear lesion in the external capsule.

10. Touche (Henschen case 1281). A man aged 70 years had a right hemiplegia but not anesthesia. There was no alexia and calculation was preserved. There was dysarthria on spontaneous and on repetitive speech. He had agraphia though he could write his name. He wrote only one word on dictation but copied perfectly. Autopsy showed only one lesion which was in the

external capsule. It had destroyed the posterior portion, but did not extend back beyond the lenticular nucleus.

11. *Clinical picture of senile dementia: Paraphasia with slight retained ability to write. Autopsy showed old slitlike destruction of left external capsule.* (Los Angeles County Hospital, No. 196–989.)

John B., aged 88 years, was admitted to the psychopathic ward Dec. 27, 1939 with a diagnosis of senile dementia. He was discharged in a day, but was readmitted nineteen days later to the neurologic ward because of inability to void. He died in a few hours. The blood creatinine content was later reported as 9.1 mg. per 100 cc.

From the family it was learned that the patient had suffered a cerebral vascular accident months before. He fell and was unable to rise for a few hours. After a few weeks he was no longer paralyzed, but his right limbs were not well controlled. He talked peculiarly, was unable to find his words, and used the wrong ones to express himself, yet he was able to "write a little." He had been right-handed.

Autopsy showed a marked arteriosclerosis, but only one focal lesion which was an old slitlike destruction of the left external capsule. (Case No. 196–989.)

12. *Bilateral cerebral vascular accidents at different times. Paraphasia. Hemorrhagic cavity left external capsule.* (Los Angeles County Hospital, No. 239–025.)

Wm. Can., a white man aged 52 years, known to be suffering with hypertension, was admitted Sept. 14, 1932 following a "stroke." He was too stuporous to give any history but from the family it was learned that two years before he had had some sort of a cerebral vascular accident affecting the left limbs. Following his recovery his left upper limb had been somewhat "stiff" and he was unable to return to work.

After a few months he had a somewhat similar episode, but on the other side, as his right limbs were affected and not rigid but flaccid. He also had difficulty in finding the right words to express himself; he was unable to write any more, and the family thought he was confused. There was no tremor of the limbs.

A third episode occurred five weeks prior to admission and the patient was of necessity confined to bed. His condition had become worse two days before, and this turn of events resulted in his admission to the hospital in coma.

Physical examination showed him to be stuporous and when aroused he was disoriented. He was totally unable to carry on conversation but when excited he muttered noisily. Blood pressure was 160 systolic and 110 diastolic and the pulse was rapid. The left pupil was larger than the right, both sluggish

to light. The deep reflexes were all quite active, more so in the upper limbs. There was no actual paralysis but both lower limbs were weak and a bilateral Babinski response was obtained.

All tests for syphilis were negative, but the spinal fluid pressure was 220 mm. of water; globulin was increased, but there were only 2 cells per cmm.

The patient became comatose; his temperature rose to 105 F; the left pupil became fixed in its reaction to light, and he died on the 5th day.

Examination of the brain showed in addition to the generalized arteriosclerosis an old hemorrhagic cavity in the left external capsule and a recent hemorrhage in the right parietal region.

Analysis of the preceding 12 cases.

These cases show that within the quadrilateral space of Marie the external capsule has much to do with language by virtue of its conveying of impulses from the language formulation area forward to Broca's convolution. Destruction of it causes either paraphasia and agraphia or motor aphasia and agraphia. The paragraphia is due either to faulty formulation in the posterior language formulation area or to faulty execution by Broca's area. In either case the implication is obvious that the co-ordinated function of the major language zone is disturbed because of faulty communication between its centers. (The external capsule is only a fiber tract.)

After a destructive lesion of the external capsule the patient will either have to use the speech mechanism of the minor hemisphere as a unit or he will perform one of the two functions (formulation or emission) on the minor side and the other by means of the major side. If the minor area of Broca is utilized, motor aphasia results until time is available for training. If the minor area of formulation is used and the impulses are conveyed across the corpus callosum to the major Broca's convolution paraphasia results. A lesion of the anterior limb of the major internal capsule will produce dysarthria or anarthria by virtue of interrupting the projection fiber tracts from the precentral gyrus to the pons and medulla. Such a disturbance has no relation to aphasia.

INTERHEMISPHERIC SHIFTING OF FUNCTIONS

Except for the few results of electrical stimulation of speech centers by Foerster the science of aphasia rests on clinicopathologic material. The defects in such material are partly due to incomplete studies of the clinical manifestations, partly to the poor general condition of patients with cerebral vascular lesions, and partly to incomplete study of autopsy specimens. But, the greatest

difficulty encountered by students of the subject is the fact that nature does not often produce discrete lesions so placed as to make the case a good experiment.

Henschen overcame much of the chief difficulty by accumulating all of the material in the world's literature on the subject of aphasia. A review of the cases gathered by him shows that, occasionally, nature does produce an experiment almost ideal, a case which establishes broad generalizations. Necessary surgical operations in the last few decades, more particularly in the last decade, have also given us cases which prove crucial points. One of the best is the removal of the entire pallium of the major side. This operation showed that the minor side can, to some degree, perform the function of language.

The material presented in this section shows that the emissive function may be shifted, after a lesion of the major side, to the minor side without other functions following suit, that the major temporal lobe is not necessarily ipsilateral to the major occipital lobe (when one occipital lobe is the major), and that the function of auditory language may be shifted independently of the emissive function. Other points are also clarified. To the autopsy material have been added a few clinical cases which also are decisive concerning certain general principles of aphasia.

1. *Right hemiplegia with motor aphasia. Recovery in time from both except for residual dysarthria. Nine years later the patient developed left hemiplegia and again motor aphasia absolutely complete. She retained the ability to read, write, and to calculate all within limits and with errors. Autopsy showed old subcortical hemorrhagic cyst mesial to Broca's convolution on the left and recent softening in the same area on the right, as well as in the right lenticular nucleus and insula. There were a few scattered distant lesions.* (Los Angeles County Hospital, No. 237–408.)

Mrs. Mary M. W., a white woman aged 66 years, was admitted June 23, 1937, because of a left hemiplegia. Dr. Boris Arnov, her family physician, had been called when the patient's landlady found her that morning unable to get out of bed and unable to speak. By framing questions which could be answered by gesticulations of "yes" or "no" the family physician was able to gather that her paralysis had come on during the night without loss of consciousness.

Dr. Arnov gave the additional history that the patient had suffered an attack of right hemiplegia with aphasia nine years before, from which she gradually recovered. Her daughter gave the information that she was able to talk "a few weeks" after her "stroke" and was able to "write," though "illegibly," two and a half months after the episode. Her hemiplegia had almost completely disappeared and the only ultimate remnant of her aphasia was a moderate dysarthria in the form of thickness and slurring of speech.

The patient lived alone although she had a married daughter and also a granddaughter living in the same city.

She had been under the care of her doctor for seven years for diabetes and had gone to his office regularly for examination of the urine and blood. She had always been able, since her recovery from the early stages of the aphasia, to say anything she chose. She had been able to read and to write and deport herself so that she was not considered limited in speech or in general mentality. On the morning of June 23, however, Dr. Arnov had found her unable to say a single word although she seemed to comprehend all that was said to her. It was strange to him that it was a left hemiplegia from which she was suffering this time though her speech was affected as it had been at the instance of the first (right) hemiplegia.

It was also learned that the first attack of hemiplegia was considered the result of an automobile accident. Except for that accident there had been no other trauma and no serious illnesses, but the patient had suffered with bladder trouble for some years which had necessitated her voiding every hour during the day and several times during the night. There was, however, neither burning nor pain associated with this.

Physical examination on admission to the hospital showed an obese and senile woman with a left hemiplegia. Slight movement was possible in the lower limb but the upper was completely flaccid. The blood pressure was found to be 180 systolic and 96 diastolic and the palpable arteries were sclerosed. The heart was enlarged to the left but murmurs were not present. There were surgical mid-line scars on the abdomen though no history of operations was obtained from anyone. All deep reflexes were absent on the left side, but active though not spastic on the right. One could not be certain of hypesthesia. The Babinski sign was marked on the left and weak on the right. The patient seemed much discouraged at her inability to express herself and had to be urged to co-operate.

Laboratory work confirmed the diagnosis of diabetes. There were sugar and acetone in the urine and the blood sugar determination gave 290 mg. per 100 cc. of blood.

On the 25th the patient attempted to write her requests when she could not talk. She had no trouble in using the pencil with her right hand (natural for her) but was acutely ill and could not sit up well. One could make out, "I want a drink of water and something to eat." (See fig. 28.) A study of her aphasia was undertaken with the result that she was found to have a complete subcortical motor aphasia, i.e., she was unable to say a single word. She could not repeat on request and she never swore or uttered any emotional expression such as a patient with Broca's aphasia usually does.

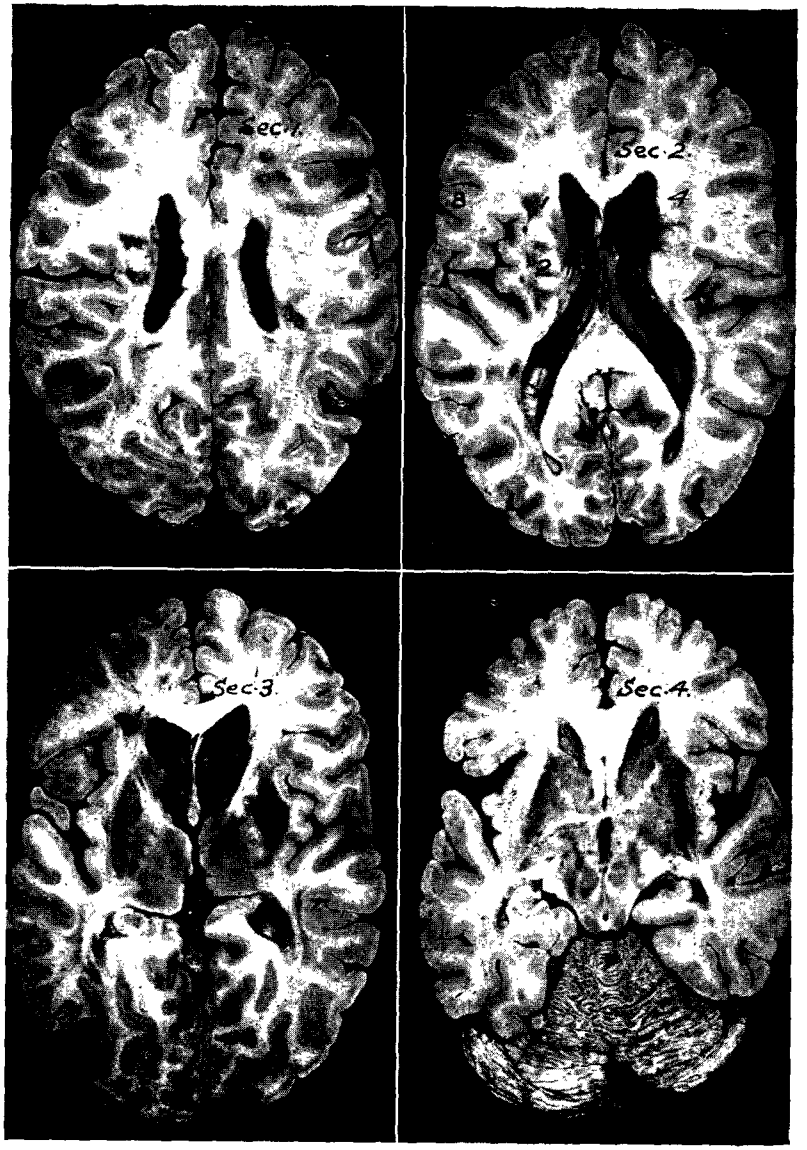

FIG. 27. Case of Mary M. W. The upper left section was made 1 cm. above the base line (which passes through the center of Broca's convolution and Wernicke's area on each side). It shows a gross area of softening (*arrow*), the extension of which in section 2 creates a lesion subcortical to Broca's convolution on the right side. This was the recent lesion. Section 2 (upper right) shows an area of destruction (marked *1*) subcortical to Broca's convolution on the left (*B*). The same lesion is marked *1* in section 3. Section 3 (taken 1 cm. below the base line) also shows red softening of the right lenticular nucleus (which, however, cannot produce aphasia). Section 4 is taken 2 cm. below the base line

She was able to read so far as recognition of symbols and words was concerned but could not comprehend complicated expressions. She was similarly able to write but always made some errors. Calculation was good within

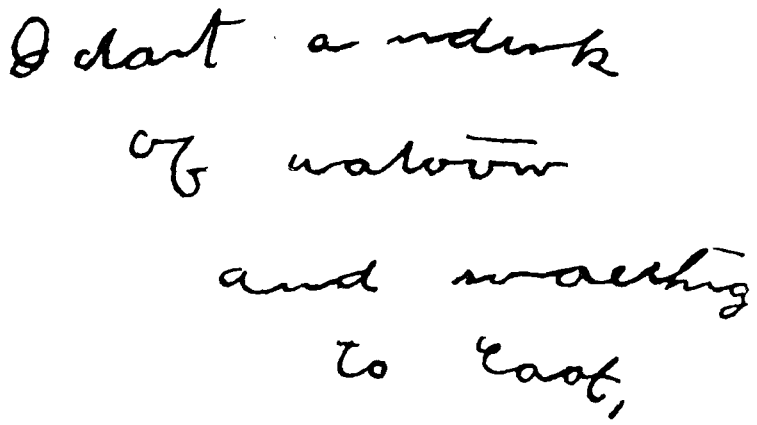

FIG. 28. Case of Mary M. W. Attempt of the patient to write. "I want a drink of water and something to eat."

limits of simple sums and multiplication. In striking contrast to this her comprehension of spoken language was excellent. There was no hesitation whatever; she carried out spoken requests with alacrity. Even long and somewhat complicated sentences were grasped without hesitation. It was concluded that her defects of language, except the motor elements, were all quantitative defects, i.e., semantic defects. On the other hand her motor defects were apractic. She had difficulty in protruding her tongue and in pursing her lips as well as complete inability to speak.

A diagnosis was made of bilaterial subcortical lesions in the insula or Broca's areas and in the basal ganglia.

On the fourth day in the hospital the patient developed signs of congestion in the chest which gradually became a frank bronchopneumonia, of which the patient died June 29, 1937.

At autopsy the brain was preserved and studied after two weeks of hardening in formaldehyde solution. The brain was sectioned horizontally parallel to a plane passing through the center of Broca's convolutions and Wernicke's center. The first section illustrated (Fig. 27) was made 1 cm. above that plane. It shows four small areas of softening, one acute and three of long standing. On the right side are seen one in the subcortical prefrontal region and one just lateral to the lateral ventricle in the parietal region. On the left are seen two lesions, one old one at the base of the central sulcus just lateral

to the ventricle, the other a small recent hemorrhage in the cortex of the supramarginal gyrus. These are all above the areas concerned with speech and have no bearing on the present problems.

Section 2 passes through Broca's convolution and Wernicke's center of each side. In order to insure this location the sections were made from each side separately to meet in the midline. On the right side is seen softening of the basal ganglia only. On the left side one sees softening of the basal ganglia (marked 2) and also an independant lesion (marked 1) just at the border of the insula and the frontal operculum. This is the most important lesion as it is subcortical to Broca's convolution (B). Softening can be discerned grossly at 4, subcortical to Broca's convolution of the right side. In the next lower section we shall see the direction and extent of this.

Section 3 was made 1 cm. lower than the second. The lesion to be followed (marked 1) is seen to be located at the border of the insula and the frontal operculum just as in the previous section but measurement shows it to be 1.5 cm. farther forward. It thus runs obliquely downward and forward or upward and backward. There is no other lesion in this section on the left side of the brain. The tiny cysts on the lateral border of the putamen are very old and of no importance. On the right side, however, one sees as a continuation of the softened area and of the section above, acute and complete softening in the lenticular nucleus. An area of softening extends forward from it into the anterior limb of the internal capsule subcortical to Broca's convolution.

Section 4 shows on the left side very old and small cysts on the lateral border of the putamen and on the right side a continuation of the softening of the lenticular nucleus. The level of this section is easily determined by the anterior commissure and the red nuclei.

Comment. This patient was right-handed. When she had her first (right) hemiplegia it was caused by a hemorrhage anterior to the basal ganglia on the left side of the brain. At the time this was an acute lesion with surrounding edema, and when the patient regained her speech one would naturally have thought that she had merely recovered from the effects and was again talking by means of the left cerebral motor speech area. She had a residual dysarthria which would have been corroborative of this interpretation. However, when she suffered another hemiplegia and this on the opposite side and again developed aphasia which left her speechless, and the autopsy showed acute subcortical softening of the right Broca's area and of the basal ganglia, there can be no doubt that her speech for the nine years was carried on by the right motor speech area. The case is therefore crucial as proving in this case the capacity of the minor motor speech area to take over the function when the corresponding area on the left side was destroyed.

Kuttner, in reporting a similar case, cited without reference a case of Norsellis which showed that in certain instances the minor side is totally incapable of assuming a certain function. The patient suffered a gunshot

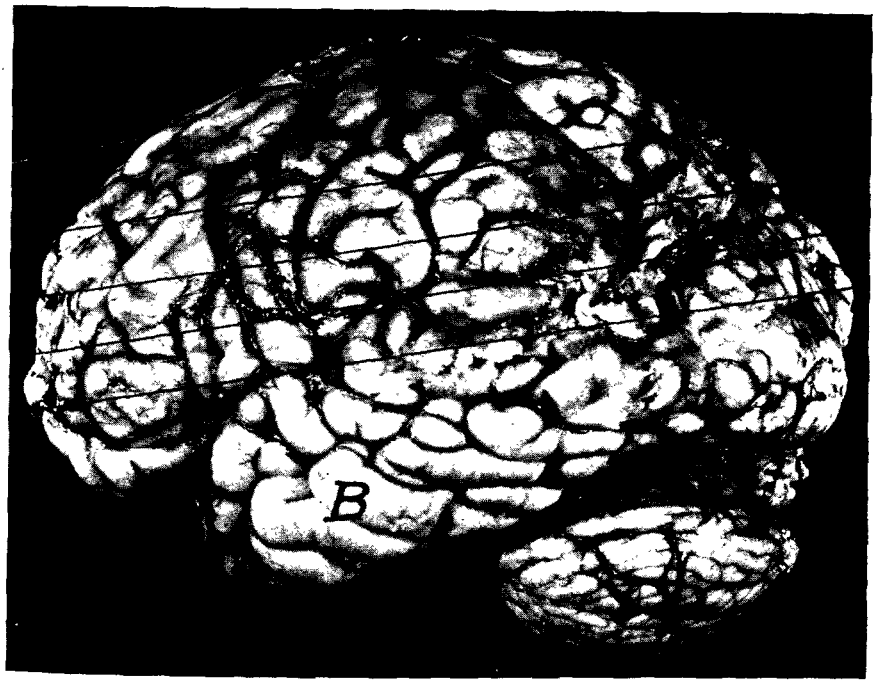

FIG. 29. Case of Lulu L. Lateral view of brain showing lines of sections which appear in Fig. 30.

wound of the brain corresponding to the motor speech area. He was for a time unable to speak, but in two years he recovered almost completely. After five years a plastic operation of the skull was undertaken and through infection an abscess developed at the old site. Then aphasia again set in. This proves that the right side did not take over the function but that the left side recovered only to lose the function again when a new lesion occurred. Kuttner concluded that in some cases the right side takes over the function of speech very well, while in others it does not take it over at all.

2. *Cerebral thrombosis in April with gradual recovery from paralysis and aphasia. In August a fresh cerebral thrombosis but of motor area which left the patient speechless. Autopsy showed an old thrombosis of posterior temporal and angular gyrus arteries and recent thrombosis of rolandic artery, all on the left side.* (Figs. 29 and 30.) (Los Angeles County Hospital, No. 728–373.)

Lulu L., a white woman aged 55 years, was admitted on Aug. 8, 1940, following a cerebral vascular episode which had left her speechless though able to comprehend spoken language. She was also weak on the right side. Rela-

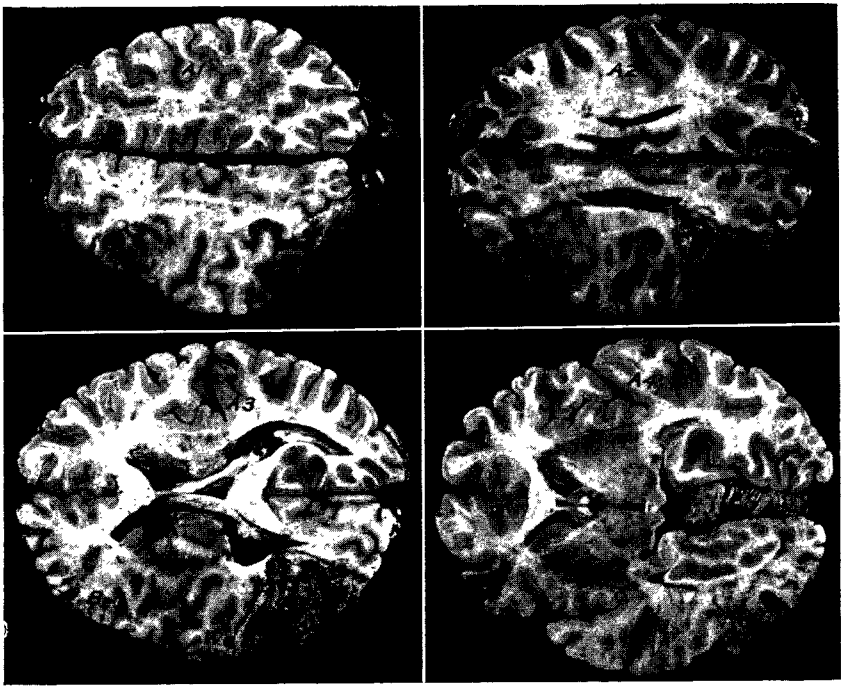

FIG. 30. Sections from case of Lulu L. The upper left section shows the two darkened thrombotic areas (rolandic and angular gyrus areas). The upper right section shows the lesion in the angular gyrus extending inward to reach the lateral ventricle. In the lower left section Broca's convolution *Br* and Wernicke's area *We* are marked. Both are clearly out of function. The lesions are still visible in the lower right section.

tives gave the additional history that the patient had had a previous "stroke" in April but from which she had recovered, both in motor power and in comprehension of spoken language. The physician who had cared for her also stated that she comprehended spoken language and spoke well a few months after her first illness.

General physical examination showed a hypertension, the blood pressure being 190 systolic and 120 diastolic. The heart was enlarged to the left and rales were heard at the bases of the lungs. She was unable to swallow or to control her sphincters.

Neurologic examination showed small but equal pupils which reacted promptly to light and on convergence. There was a right homonymous

hemianopia. The right lower facial muscles were weak, the right limbs spastic but not entirely paralyzed. The uvula and palate both deviated to the right on protrusion. The deep reflexes were all increased and almost symmetrical. All of the common pathologic reflexes were present on the right side only. There was also a distinct hypesthesia on the right side.

She improved for the first two days and a study of her aphasia was made. She was totally unable to write or to utter a word. However, she understood spoken language and this enabled the examiner to determine that she could recognize mathematical figures as well as all general objects and colors and that she had no apraxia. Yet she was totally unable to read letters or words. She was able to demonstrate fingers on request and was never confused regarding laterality. There was no evidence of syphilis physically or serologically and the spinal fluid was also otherwise normal.

On the fourth day in the hospital the patient developed pneumonia and died. Examination after two weeks of fixation showed an extremely interesting situation. As shown in the photographs there was an old softening involving the entire angular gyrus and the superior first temporal convolution of the left side. The angular gyrus lesion extended in to the lateral ventricle. In addition there was an acute lesion only a few days old resulting from thrombosis of the rolandic artery.

In Fig. 30 (1, 2, 3, and 4) are shown the sections indicated by the lines on Fig. 29. The third section from the top passes through Broca's convolution (*Br*) and Wernicke's area (*We*) shown in Fig. 30. It is seen that both areas are destroyed by softening. The posterior portion of the line passes through the angular gyrus as does the section immediately above.

Comment. The destruction of the temporal lobe and the angular gyrus was of long standing; that of Broca's convolution was recent. Since the stroke in April, therefore, the patient must have used her minor temporal lobe for comprehension of spoken language. That was why she did not lose that function when the new stroke occurred. On the other hand she must have continued to use her left Broca's convolution for speech; otherwise she would not have become mute with the advent of the fresh thrombosis (of the rolandic artery). Here, then, is an instance of recovery from auditory aphasia by use of the minor side but with continued use of the original Broca's area.

The alexia (visual verbal agnosia) needs no elucidation in view of the complete destruction of the angular gyrus. It is of interest that there was no finger aphasia or apraxia since the lesion was located where, in certain cases, it would have caused such a deficit. It is probable that if the patient had lived a few more months she would have regained her ability to speak.

This case shows that the minor temporal lobe may assume the function of

the major without the minor motor speech area also being required to function for its major.

3. This case has been reported before (1940) and will therefore be mentioned only briefly. The patient suffered visual verbal agnosia due to a lesion in the left angular gyrus and later auditory verbal agnosia from a lesion of the left superior temporal convolution. He later developed visual agnosia for inanimate objects associated with a left homonymous hemianopia. Autopsy showed vascular lesions in the left temporal and angular gyri and in the area of distribution of the right posterior cerebral artery, calcarine branch. In the same article another case was presented with visual agnosia from a lesion of the right posterior cerebral artery, but, as there was no lesion in the left hemisphere one cannot postulate that the patient was actually left-brained though she was right-handed. In the case cited (case 3) however, there cannot be any doubt as lesions were found in the left temporal lobe and left angular gyrus. He was therefore left-brained so far as the temporal lobe was concerned but right-brained in the occipital lobe. There are 2 similar cases reported in the literature.

Two cases without autopsy examination seem sufficiently clear to warrant brief citation.

4. Mrs. Emeline Th., aged 76 years and suffering with hypertension, was admitted because of left hemiplegia. She had the delusion that her left upper limb did not belong to her. There was no immediate evidence of aphasia.

On taking the history it was learned that she had had a right hemiplegia five years before with complete aphasia but that she had recovered her speech function in eight months. With this information her case was studied more in detail and it was found that she was totally unable to read.

Physical examination showed a complete left homonymous hemianopia and loss of knowledge of position of the left upper limb. She was unable to find the left arm with the right hand and sense of touch was greatly diminished in it.

As it is well known to us that delusion of the body scheme means a lesion of the thalamosupramarginal fibers, such a finding in association with hemianopia and loss of sense of position of the limb places the lesion accurately. With this information we can state without doubt that as a result of the first vascular episode five years before and the resultant aphasia which left the patient even unable to write her name, to speak, or to comprehend spoken language, her language area moved to the minor side. If it had not moved she would not have developed alexia with this new episode so clearly localized in the white substance between the right thalamus and the parietal cortex.

5. This case was reported by me (1937) and will be abstracted briefly here.

A man of 68 years who was left-handed from birth was compelled by his teachers to use the right hand for writing. After the age of 8 years he never attempted to use the left hand for that purpose.

At the age of 68 years he suffered a left hemiplegia with aphasia. (He must still have been right-brained.) He recovered from this episode completely. Seven months later he suffered an attack of right hemiplegia but without apparent aphasia (as would be expected if the right hemisphere had resumed its language function). However, examination showed that he had a complete right-handed agraphia unknown to himself. He offered to write but found himself unable to form any letters. The artificially produced writing center on the left side of the brain (produced by the compulsory use of the right hand) was out of function; there was enough strength in the right hand for using instruments such as a fork. When he was then asked to write with the left hand he found to his astonishment that he could write with it very well, though he had not tried for sixty years. These facts show that a writing center was formed on the left side of the brain and that it went out of function sixty years later with a vascular lesion of that side. In spite of the formation of this writing center the speech area in general did not move over as shown by the fact that he developed aphasia with his left hemiplegia.

Conclusions.

From the cases presented and cited here the following conclusions can safely be drawn:

1. The putamina have no function in language (this statement does not apply to the external capsules).

2. The minor cerebral hemisphere assumes the function of the major in language with great facility in some instances, with difficulty in others, and not at all in some persons.

3. The language function if partly destroyed does not usually transfer to the minor side in toto; the visual, auditory, or motor functions may be transferred separately.

4. An artificial writing mechanism may be formed on the minor side by training; when this happens the entire speech mechanism does not of necessity move to the minor side.

5. The major temporal lobe and the major occipital lobe may not be ipsilaterally located.

6. It is unsafe to lateralize a lesion on the basis of an aphasic manifestation alone.

ADDITIONAL CASES

1. *Motor aphasia, agraphia. Autopsy.*

Edward Ingols, aged 68 years, had a mastectomy for carcinoma of the left breast Aug. 29, 1934. He remained well until December 4 of the same year, when he entered his home complaining of "numbness" of the right arm and hand and of poor function of the right side of the face (monoplegia brachiofacialis). He was also unable to talk for about ten minutes, although during that time he was able to understand what was said. Within half an hour his face and hand recovered their strength.

He was well the rest of the day and was still feeling as usual the next day when he reported to the commission on malignancy at the Los Angeles County Hospital for inspection. This was at 2:00 P.M. After returning home he went to the bathroom for a few minutes, but when he came out to say something he found again that he could not talk. He was dyspneic and this fact was observed by the family which called a physician. After receiving a stimulant he felt stronger but was still unable to talk, although he understood what was said as he obeyed commands.

Upon admission to the hospital he was found to have a mild right hemiplegia and weakness of the lower right face. He had auricular fibrillation or ventricular extrasystoles. There were no sensory changes. To all questions he replied, "I don't know" or "Yes." When questions requiring other answers were put to him, he tried to say what he thought but was able to utter only these words. He was unable to name objects shown to him at the time. Later when he was again plied with questions, he succeeded in naming several objects presented, but he never spoke a sentence.

There was not any apraxia present, as he used pencil, scissors, and other objects correctly, and he used his hands as directed.

There was obviously no visual agnosia, as he recognized objects about him. We could not determine whether he could read. He did not carry out written commands.

Writing was possible to about the same extent as speech. He succeeded in writing his name with small error when he was first examined. Later, at the time he was able to name occasional objects presented to him, he was able to write several words, but not sentences. His agraphia was therefore incomplete. The patient went rapidly downward and died of multiple emboli to other parts of the body.

At autopsy (Fig. 31) the brain showed on the surface pathologic changes at only one area—the foot of the third left frontal convolution. This was

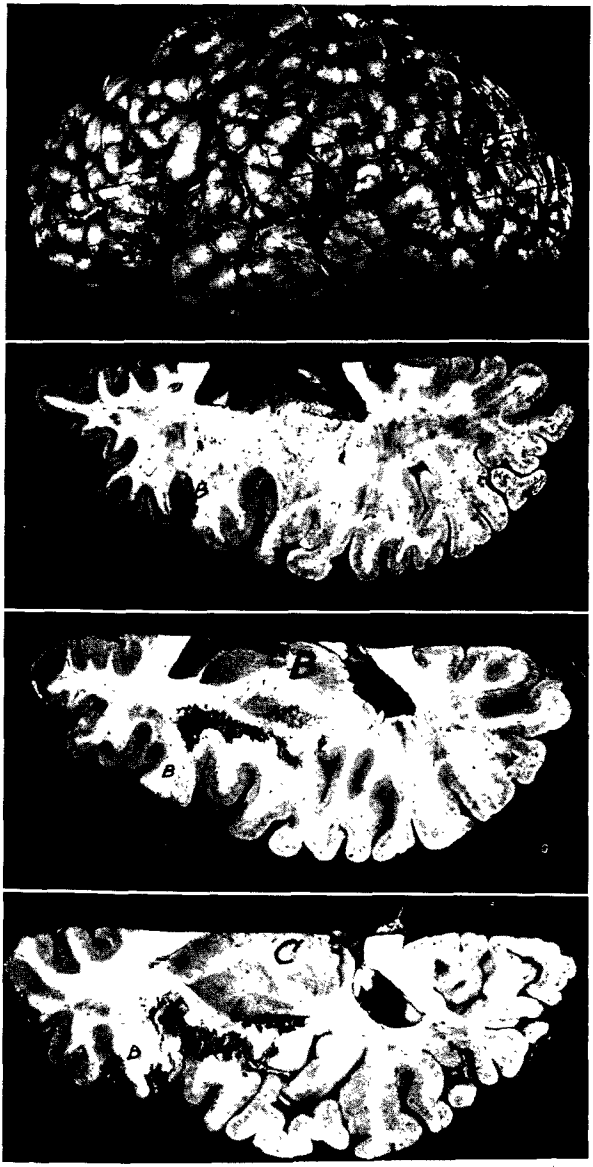

FIG. 31. Case of Edward Ingols. The uppermost illustration is a lateral view showing the lines of section. The serial views below are from photographs looking upon the three sections shown in the lateral view. In the uppermost illustration softening is seen in the anterior edge of Broca's convolution (marked *B*). There is also softening in the caudate nucleus. In the second section the cortex of Broca's convolution has fallen off and there is subcortical softening in the insula and the putamen. In the lowest section the softening is more extensive along the insula. Broca's convolution is entirely disintegrated.

entirely softened except for a small portion (the upper one fourth) of Broca's convolution. There were no changes in the right half of the brain.

The left hemisphere was sectioned horizontally on the lines shown in the illustration. Photographs are reproduced looking down upon the planes made by the upper three sections. The upper section shows softening along the anterior margin of Broca's convolution and in the upper portion of the caudate nucleus. The second shows the outer cortex of the pars triangularis and of Broca's convolution missing, because it was too frail to remain. There is also softening in the white matter below this convolution and along the upper portion of the external capsule throughout its length as well as the upper portion of the putamen. The third section shows complete disintegration of the pars triangularis and Broca's convolution. There is also a narrow skirting of degeneration in the capsula extrema separating the cortex of the island of Reil. Besides this there is softening in the posterior half of the putamen. Aside from these lesions there were no areas of softening. Sections were taken as shown (Fig. 31) for microscopic examination.

2. *Motor aphasia without instrumental amusia.*

David L. Hal. came to the hospital with a monoplegia brachiofacialis of the right side and gave the following history. He was a professional violinist and was playing in an orchestra when he was taken with a feeling of fullness in his tongue as though it were too thick for his mouth. A moment later the member of the orchestra next to him spoke, and he found himself unable to answer. He continued to play, however, for "five or ten minutes" without being able to say a word. The condition gradually increased in severity until he became so weak in his arm that he could not play.

When seen two days later he was able to talk with a little dysarthria, but the arm was still weak. He was able to write tremulously with either hand.

3. *Surgical removal of the anterior half of the left temporal lobe for cystic astrocytoma. Secondary hemorrhage into the posterior portion, with resultant hemianopia and aphasia. Recovery from the latter to a large extent. Residual alexia, agraphia and disturbance in calculation. Slowing in comprehension of spoken language. Stilted spontaneous speech. Retention of personality.*

J. E. M., a dentist aged 52, underwent operation on Jan. 3, 1936 for cystic astrocytoma of the left temporal lobe. The growing portion of the tumor was attached mesially, and Dr. R. B. Raney considered it dangerous to attempt complete removal. After recovery from the anesthesia the patient talked fairly well and was in good condition except for weakness of the right arm and right side of the face for thirty-six hours. On the afternoon of the second day he became stuporous, and his temperature rose to 109 F. After this episode he

was completely hemianopic, which he had not been before, and was unable to talk at all. By slow degrees he relearned to speak.

A record of the various stages is long and not particularly instructive. The patient taught himself to read, looking up words in the dictionary and practicing writing because he could no longer spell correctly. He worked so diligently at this re-education that he showed signs of overwork. The examination recorded here was made after eighteen months.

Comprehension of spoken language. The patient failed to grasp spoken language if it came to him a little rapidly. He listened as though trying to comprehend a foreign language; he cocked his ear when listening at all times, yet often had to ask to have conversation repeated. If speech came too fast the words were all jumbled to him.

"Tell me about the jerking of your limbs." "It's almost every day (we mustn't talk too loud; my wife . . .). The tongue doesn't move as often as it did." (The patient meant that the jacksonian jerking of the tongue had diminished.)

"What does move?" "Why, nothing in the mouth. It's cutting down on itself."

"Are you taking the tablets (phenobarbital)?" "Why, yes, right up to date."

"How bad are the jerkings?" "Not nearly so bad as they were. They are cutting down."

"How long do they last?" "Oh, not enough to put me out, and they used to do that. They don't do that, so I think I'm ahead."

"Put your left index finger into your right ear." He hesitated a moment, smiled, and did so.

"Hold up the index and little fingers of your left hand and the middle finger of your right." He asked to have the request repeated, then did correctly as requested.

Spontaneous speech. His speech was peculiarly stilted. This is shown by the following conversation.

"What do you think of the administration?" "National or city?"

"National." "It's doing things of unusual . . . Roosevelt undoubtedly saved this country when he closed the banks. He undoubtedly is going into the expensive condition too deeply. To get into the billion dollars, something that is never happened to the world. Therefore, the good that he does is not sufficient to wipe out the bad that he does; I don't know whether that meets with your approval or not; I'm a Republican as far as that's concerned. Not that I want a Republican elected every time."

"What do you think of the strikes?" "Well, I think that there is a greater number every time there is a success in the nation. Success in the nation. No. I'll take that back. Every time there is a greater income. . . . You know I think faster than I can talk, and I may not talk like I think." (Consciousness of his paraphasia.)

"Why don't you?" "Because the vocabulary isn't there, and I find my forgetful-

ness comes upon myself before I can change. I don't want to say that either. I can't answer you."

"You are doing very well." "Well, I'm thinking just the right way according to I read, but I don't read as rapidly as I used to. But people are too impatient to let me talk slow. My wife and my family, my daughter wouldn't do it on a bet."

"Is it harder to read than to understand spoken language?" "Oh, yes . . . Bob and his mother don't get along."

"Why not?" "I don't know. It's real and not imagined, and it's possibly that thyroid of hers. Anyway, she ought to have more patience with him and with me."

"What irritates you most, quarreling?" "No, there isn't anything that irritates me, because when my wife enters with an argument on my son, I just walk out on it. So that's the reason I want to go away. I was there six weeks, and I only got back Saturday (from a sanatorium). And I wouldn't have quit there if it hadn't been for the heat." (It had really become warm.)

"Do you do any writing?" "Oh, yes, I practice on it constantly. But I can't write fast as I used to. Nor spell. Oh my, whew! I'm learning all over again. See, I have the dictionary over there, and wherever I go I take it with me."

"Do you do any physical work?" "No."

"Is your hemianopia improving?" "No. They are just as bad as they ever were."

"Do you have any sensory loss anywhere?" "No, no, but I still have those manipulations here." (He indicated his right leg.)

"Do you mean jerkings?" "Yes, but I don't tell her" (his wife).

Calculation. Considering the fact that the patient had been especially capable in mathematics, one must conclude from his performances in the following test that this function had been considerably affected by the operation. He was unable to solve the problem $\frac{21 \times 43 \times 20}{4 \times 22}$. He failed to observe that cancellation was possible, and even when this was suggested he did not know how to proceed. He finally multiplied as follows:

Then divided as follows:

$$\begin{array}{r} 43 \\ 21 \\ \hline 43 \\ 83 \\ \hline 873 \\ 20 \\ \hline 17460 \end{array} \qquad \begin{array}{r|l} 88 & 17460 \;/\; 219 \\ & 88 \\ \hline & 866 \\ & 6 \\ & 8 \\ & 2 \quad 12 \end{array}$$

and then he surrendered. He correctly multiplied 3 by 4, but when asked about 7×9 he hesitated and said: "That's too far. Let's see. I can figure it

out if you'll wait a minute—63, is that right?" When asked how he figured it out, he explained that he multiplied 7 by 3 three times. He correctly calculated 8 × 11, but with 9 × 9 he hesitated for some time, repeated 9 × 9 and said: "I can't even guess it. I give up." When asked whether he had been proficient at mathematics he replied: "I'll say; I'll come back at it."

Before his illness the patient had been a very good chess player. After the surgical procedure he was handicapped by hemianopia, but apart from that element was a poor player. When warned in a friendly way that an important piece was being attacked, he saw the danger and prepared to defend, but forgot this and made some other move. In other words, he thought slowly, and his retentive memory was impaired.

Change of personality. The patient's wife was questioned about any change in his personality. She thought he was more irritable than he should be, and more than he had been before the operation. The patient himself stated that his wife found constant fault with the son and that the two did not get along at all. This constant conflict irritated him, and he tried to get away from the house. (Our own observation was to the effect that the wife was really irritating, because of hyperthyroidism. She was somewhat overactive and tended to dominate the home. She was an almost constant talker and had a somewhat rasping voice. It is probable that the patient's criticism was correct. If this is so, there was no discoverable change in his personality.) He was deliberate and extremely thorough and persistent.

In this case re-education had a most salutary effect. The defects in language formulation and in writing are evident.

(Case reported by Nielsen and Raney in the *Archives of Neurology and Psychiatry* 42:189.)

4. *Left temporal lobectomy. After operation marked acoustic verbal agnosia, alexia, and agraphia with the syndrome of exceedingly rapid fatigue of all functions of language. Retention of personality.*

R. R., an Italian building contractor aged 53, under the care of Dr. C. B. Courville, had a condition that was diagnosed as tumor of the left temporal lobe. Because of changes in personality, one could not approach the patient with the problem of treatment, and the family delayed until the patient was in coma. Dr. R. B. Raney at this point removed the left temporal lobe for an infiltrating astrocytoma. The line of incision, as mapped by Dr. Courville, who watched the operation, is shown in Fig. 19. After operation, the following study was made.

Comprehension of spoken language. Comprehension of spoken language was good for generalities for a short time. The patient was usually able to answer correctly about a dozen questions, after which he "broke" and sud-

denly comprehended nothing. The following conversation will demonstrate this.

"What is your name?"	"Ross."
"Ross what?" (hesitation)	"Ratania."
"Where do you live?"	"I can't tell you where I live because I don't know where I live."
"Where do you live?"	"In my own house."
"How old are you?"	"About 25." (Actually he was 53.)
"Have you got a son?"	"Yes."
"What is his name?"	"The biggest one?"
"Yes, the biggest one."	"The biggest son is Ross."
"Just like you?"	"Yes, Ross."
"Have you a son named George?"	"George Ratania."
"What is this? (a dollar bill)?"	"A woman." (Paraphasia.)
"What are these (a pair of glasses)?"	"They're all right."
"What is the name of it?"	(The answer consisted of complete neologistic jargon, and after this he understood nothing.)

On another day, after a nap, when the patient was feeling well, the following took place:

"Stick out your tongue."	(He did so.)
"Close your eyes."	(He did so.)
"Put your finger on your nose."	(He put it into his mouth.)
"Put your finger on your ear."	(He stared blankly.)
"Raise your hand."	(He stuck out his tongue.)

On still another day the following conversation took place:

"What is your name?"	"Ross Ratania."
"Where do you live?"	"Los Angeles."
"What street do you live on?"	"You find out the way you live yourself. You ask my wife, that's all." (He then threw up his hands in expression of helplessness.)

After this he understood nothing. He cocked his ears and listened as though listening to a foreign language. He did no better in listening to his son speak in Italian than in English. (He was born in America.)

Spontaneous speech. In talking spontaneously he usually spoke a few short sentences nearly correctly or with slight paraphasia. If he attempted anything long at first, or even a sentence of a few words after having talked for a

minute, his speech became severely paraphasic. When asked: "How long have you been in this country?" he answered, "I'm in America all my life, all my life. I still live, but before I die I go. When I was a young fellow I join the army, before I join the army, and I joined the navy one day and made the American army under when. But I went to army my own record I was just make a sweat. When I left the old country I left everything here. The last time I leave my wife Catalina."

Reading. When shown A he said, "I can read it," but he did not. When shown S he read it correctly. When shown 5 he read it correctly, but when shown 8 he read "5–10." Then, when shown 7 and 4 he read both correctly. When shown "Signs of the Times," he read, "Ninyet nugut tus tus."

After a long rest he was shown C, but could not read it. When shown the word cat he read "Catherine." For "no breakfast" he read "new broker," and for "house" he read "careless."

To ascertain that the lack of ability to read was not merely paraphasia, i.e., inability to say what he meant, my co-workers and I prepared a series of words, such as "key," "safety pin," "red," "yellow," "green," "blue," "half dollar," "quarter," "pen," and "dime," and the corresponding colors and objects were provided for the patient to place on the appropriate words. He was shown several of them first. After a long time we gave up the attempt to teach him. He was totally unable to read (say) the words or to determine what they meant. He finally said: "No, no, my eyes. You know your business. I know my nation. I'm in no position to answer my nation." He had complete agnostic alexia.

Writing. The patient had complete agraphia. He was unable to use a pencil at all, though he had had a fairly good education. (He had had agraphia before the operation.)

Calculation. It was difficult to determine whether he was able to calculate. He gave the correct answer to $2 + 3$, but to similar problems he gave wrong answers. Of course, he had severe paraphasia, which prevented him from giving the correct answers orally, and he could not write them. Neither could he read.

No other form of agnosia was present. The patient was fairly well oriented, considering the circumstances. His change of personality had essentially disappeared as a result of the operation. He could not play cards, but had never been able to play.

(Case reported by Nielsen and Raney in the *Archives of Neurology and Psychiatry* 42:189.)

5. *Severe semantic defect, some visual verbal agnosia, a little visual figure agnosia, agraphia, acoustic verbal agnosia.*

Samuel K. Da., a white man of 50 years, came to the hospital Oct. 23, 1934, acutely ill, showing a marked jargon aphasia, and talking a great deal. He had no paralysis. He was too acutely sick at the time to justify a study of aphasia; so he was allowed to wait until the next day. The jargon aphasia had disappeared.

Introductory. "Good morning. How are you feeling?" The patient shook his head. "What is your name?" He shook his head.

Visual agnosia. Shown the words bread, house, yard, horse, and meat and asked to point out those that are of something good to eat, he said, "Not good to eat, not a thing, Oh my." Shown various letters he occasionally read one correctly. When shown the figure 4 and asked to show what it was by the number of fingers, he showed four. For 3 he showed three fingers. When shown 3, 6, 8, 4, 9, 2, he called the 3 an 8 but could not read any more. He seemed to have a degree of paraphasia.

Acoustic verbal agnosia.

"Let me see your tongue." Showed it.

"Raise your hand." Raised it.

"Are you sick?" "Not a thing at all."

"What church do you belong to?" No answer.

"Catholic? Methodist? Episcopal? Salvation Army?" Patient showed disgust and said, "I'm done for."

The rapid fatigue is characteristic of minor temporal lobe performance.

Apraxia. "Put your finger to your nose." Patient put it into his mouth. "Have you a nose?" Patient showed his teeth. "Show me two fingers." Patient showed his teeth. "Show me two fingers." Showed one finger. "Show me two fingers." Finally showed two fingers. When he was asked to sit up in bed, he did so with some difficulty. This difficulty seemed to be apractic. It was also observed that he tried to write but put the pencil into his hand wrongly, using the eraser to write.

In view of the fact that he had a partial acoustic verbal agnosia, it was deemed wise to test him with all movements *shown*. With agnosia thus eliminated from the tests he did everything well except movements concerning the mouth area.

Motor aphasia. Patient said very little. Out of a clear sky he said, "I have no . . ." Above it is recorded that he said, "Not a thing at all" and "I'm done for." Patient is asked to tell us how he became ill. He said, "I certainly went" and then stopped.

"Have you a wife?" "No."

"Have you children?" "Yes."

"How many?" Shows four fingers.

"Can you say 'Sacramento?'" "Yep."
"Do you want to get well?" "Yep."
"Say 'I have no.'" "Well."
"Can you say, 'I have no?'" "Yeh."
Again he showed evidence of fatigue.

This is clearly not the response of one with Broca's aphasia. Neither is it sufficiently diminished for subcortical motor aphasia. We concluded that speech is reduced because of severe interference with his internal language, i.e., he had the previous evening a severe jargon aphasia during which time he talked volubly but incorrectly. Now he was still poorer and did not frame his ideas into sentences at all. There was a severe defect of language formulation. He showed a marked embarrassment at his inability to talk.

Writing. Spontaneously he wrote, "SKKSSSSK." This was evidently written because his name was S. K. Davis and he was used to writing it that way. Here he perseverated. When asked to copy the word sin he wrote "SiS." When asked to write Colorado, he wrote only a C and a scrawl.

From this we concluded that he had agraphia and inability to copy. This was in harmony with the partial visual verbal agnosia.

It was impossible to determine whether he could recognize colors. He failed to select certain colors from a series.

The final conclusion was that the patient had a lesion affecting the superior left temporal convolution and to some extent the angular gyrus.

The patient died the next day from embolism of the tibial artery, the abdominal aorta at the bifurcation, and in the brain also of the left middle temporal artery and the terminal branch of the artery of the angular gyrus. This last was so located that it affected the posterior margin of the angular gyrus. This lesion was very small. An area of softening in the anterior portion of the angular gyrus was the cause of the alexia. There was no lesion of Broca's area. (See the illustrations, Figs. 32 and 33.)

6. This case is not reported in detail because the manifestations of deprivation were relatively minimal. The patient was operated on by Dr. R. B. Raney for an astrocytoma of the right temporal lobe and the lobe removed. Prior to the surgical procedure the aphasic manifestations were slight. For four weeks the patient, who was a German-born sailor and able to speak, in addition to his native language, Danish, Norwegian, Swedish, Dutch and English, was unable to spell Deutschland, America, California, or other simple words. He also was unable to calculate or to count correctly in Danish or Swedish, although he could still count in English, German, and Dutch. It was clear that his concepts had suffered considerably, and we are compelled to assume that the right temporal lobe functioned to some extent in language before the

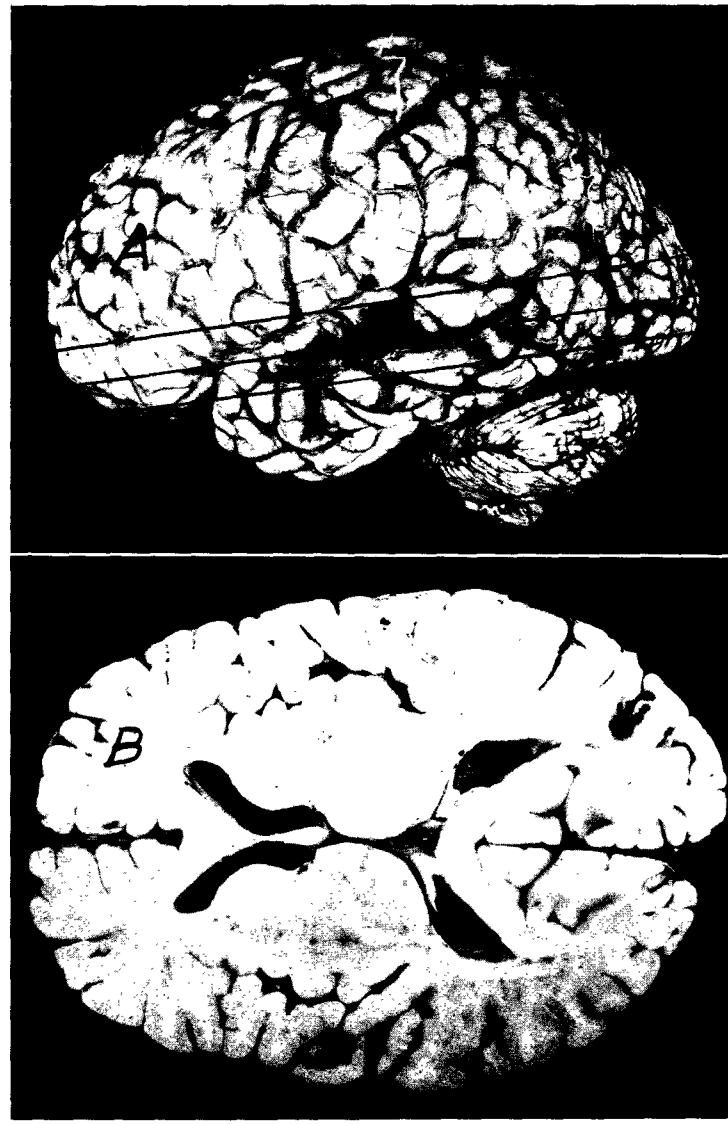

FIG. 32. Case of Samuel K. Da. Lateral view and first section of the brain. The upper figure shows the lines of sections. There is visible in the superior temporal convolution an embolic hemorrhage through which the second line passes. The photograph of the section was taken looking up. In the posterior part of the angular gyrus there is a small (dark) lesion.

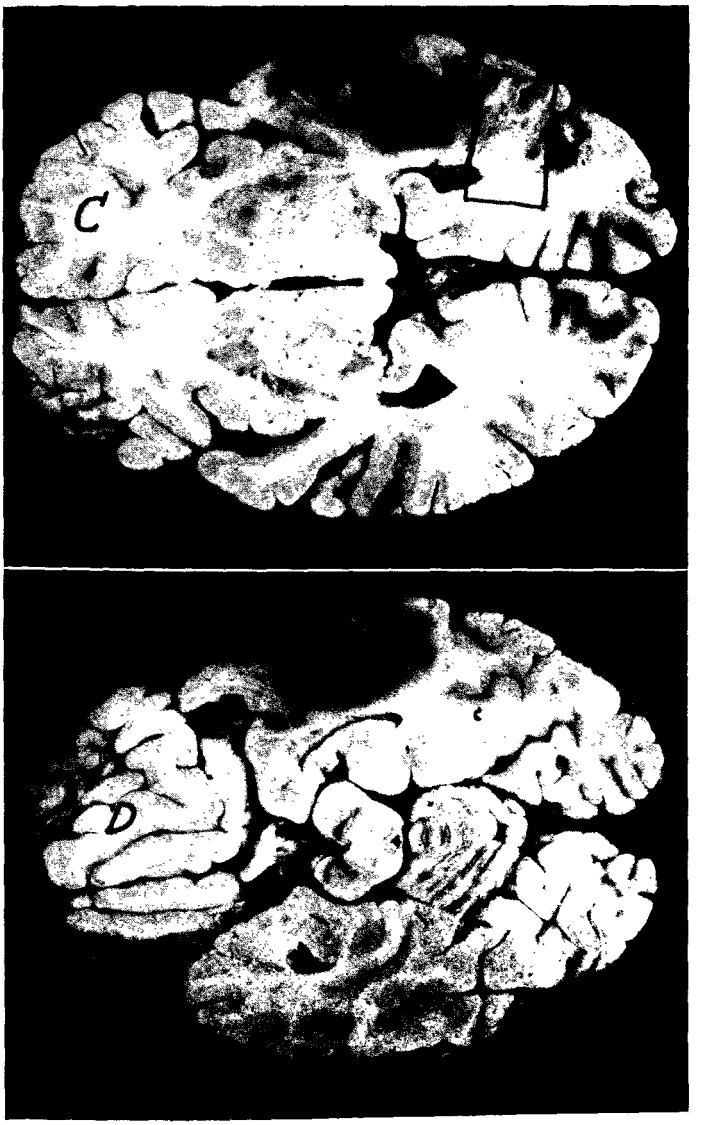

FIG. 33. Case of Samuel K. Da. Second and third sections of the brain shown in Fig. 32. In *C* the embolic hemorrhage is seen in the first temporal convolution (upper half, as the picture was taken looking up). The oblong area was examined microscopically and lesions found throughout it. In *D* it is seen that the first and second temporal convolutions were entirely out of function.

operation. However, the left lobe took over all the function after about five weeks, as shown by the fact that all these defects had disappeared at the end of that time. This case is possibly exceptional; one must avoid generalizations on the basis of it.

(Case reported by Nielsen and Raney in the *Archives of Neurology and Psychiatry* 42:189.)

7. *Epileptic seizures for fifteen years. Three episodes of cerebral vascular accident; after the second great difficulty in comprehending spoken language. The patient spoke in short sentences, with paraphasia. Marked general defect of memory. Ability to read. Death after three years from thrombosis of the basilar artery. Autopsy: (1) old softening of the left lenticular nucleus; (2) destruction of the left angular gyrus and entire temporal lobe, except the tip; (3) recent softening of the right margin of the cerebellum, and (4) acute occlusion of the basilar artery without softening. The clinical picture represents the results of destruction of the left temporal lobe.*

Mrs. A. W., a white woman aged 57, was admitted in coma on July 14, 1937. She was irritable and responded to painful stimuli with a cry, but did not speak. The body temperature was 100.2 F., the pulse rate 92, and the respiratory rate 28 per minute. Her eyes did not fix, but roamed in all directions. The pupils reacted to light, though not quickly. The vessels of the fundi were severely sclerosed. The heart was essentially normal; the blood pressure was 220 systolic and 110 diastolic. There was a Babinski sign bilaterally. The extremities were spastic and resisted motion, the left upper limb less. The right arm was in hemiplegic contracture, and there was ankle clonus on the left. The deep reflexes were symmetrical and increased; the abdominal reflexes were absent. Hoffmann's sign was present on the right. The patient died two days later, without having regained consciousness.

A history of the patient's earlier life was obtained from a son and a daughter, after autopsy had revealed the unusual condition to be described. Before marriage the patient had finished two years of high school and had then worked in a department store as a saleswoman. She had also done some artistic painting as an avocation, had made artificial flowers and had been a "great letter writer," i.e., she had been active in keeping up correspondence with friends. In all this diversified work she had always been right-handed. In recent years she had been a Christian Scientist and had therefore ignored illness. She lived during part of the time with a sister of the same faith. (This must be taken into account in interpreting the statement that she had been in good health all her life until 1933, when she had illness which could not be ignored.)

She had been subject to epileptiform convulsions for fifteen years but her

family considered her otherwise well until 1933. Change of eyeglasses was regularly attended to. In July 1933, during a period in which she lived alone, she was found one morning (by a sister who chanced to call) lying unconscious on the kitchen floor. She recovered half an hour after she was found and resumed her work the same day.

She was then considered well again until August 1934. On a certain Sunday she had spent the entire day attending church service. On arriving home that evening, at about 10 o'clock, she was unable to unlock the door with the key which she carried. She called on the landlord, who lived in an apartment above, and obtained help. To the landlord she seemed very weak, and he was uneasy about her. On the following morning, when she did not appear after a few hours, his wife investigated and found the patient semiconscious, still in bed. She did not breathe stertorously "nor foam at the mouth, but was unable to talk or give an account of herself." She did not respond when spoken to and "did not seem to be entirely aware of her surroundings."

After this she was unable to take care of herself, and was therefore taken to live with her daughter. She gradually regained power to speak, but did not speak clearly unless she was excited or angry. At such times she suddenly spoke without any dysarthria. Furthermore, her sentences were always short, and she often used the wrong word to express herself. For example, she said: "Turn out the fire," when she meant: "Turn out the light." However, after she had again learned to talk the daughter had no difficulty in comprehending what the patient meant.

Her comprehension of spoken language was more defective. She could not follow the trend of spoken language unless one spoke slowly and in short sentences. She attended church services regularly until her last illness, in 1937, but it was difficult to know how much she comprehended of the spoken language. She avoided becoming involved in conversation with friends at church because she felt inadequate to take part and confined her speech to such casual conversation as the time of day.

On the other hand, she was able to read. She read the newspapers and her lessons in a church manual, and took her church *Quarterly* regularly and read it. When fitted with glasses shortly before her last illness, she read every word of the small print. She also read novels, of which she kept half a dozen about the house. These she read and reread innumerable times. She never remembered anything she heard or read for more than a day, often not for more than an hour; she thus was entertained time and again by the same novels and considered them new each time. In the interpretation of this "reading" one might question how complete the process was. Certain it is that she gathered information from printed matter, so we shall consider that

she read, but she did not retain the information except for a very brief period.

Her general memory defect was to the family the outstanding difficulty. She was not able after the episode of August 1934 to take care of the house,

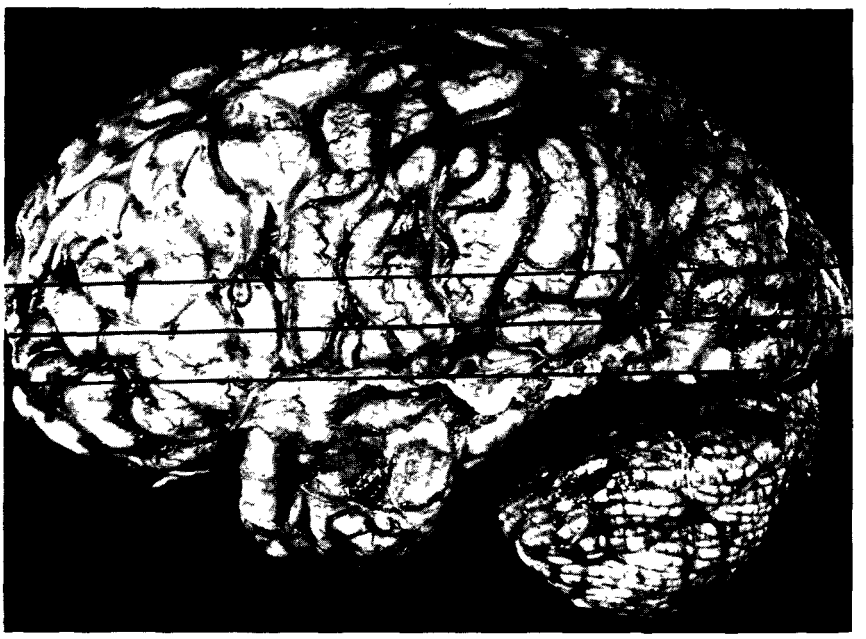

FIG. 34. Case of Mrs. A. W. Lateral view of the brain. The gross destruction of the temporal lobe with extension into the angular gyrus is clearly visible. The lines indicate the sections from which the next three illustrations are obtained. (Figures 34, 35, 36, and 37 from the *Archives of Neurology and Psychiatry*, 42:189.)

order food, or plan meals. This was mostly because of the general loss of memory. When she went to the market she could not remember what to get if there were more than two things. She could not write because of paralysis of the right arm, which never disappeared, although the leg recovered enough for her to walk to church and do work about the house. She therefore never wrote notes for herself, chiefly, she said, because she could not write well enough with the left hand. Concerning her ability to calculate it was difficult to obtain definite information. Her family could not depend on her calculating the change correctly, but always gave her small amounts; they depended on the merchant to give her the correct change. They thought she would not have been able to remember how much money she had had.

Concerning comprehension of music, it is known only that she listened to the radio and enjoyed the "music."

Her personality was distinctly changed after the episode of August 1934. She was more irritable, impatient, and excitable. The family considered this to be due to her greater difficulty in comprehending and speaking. She

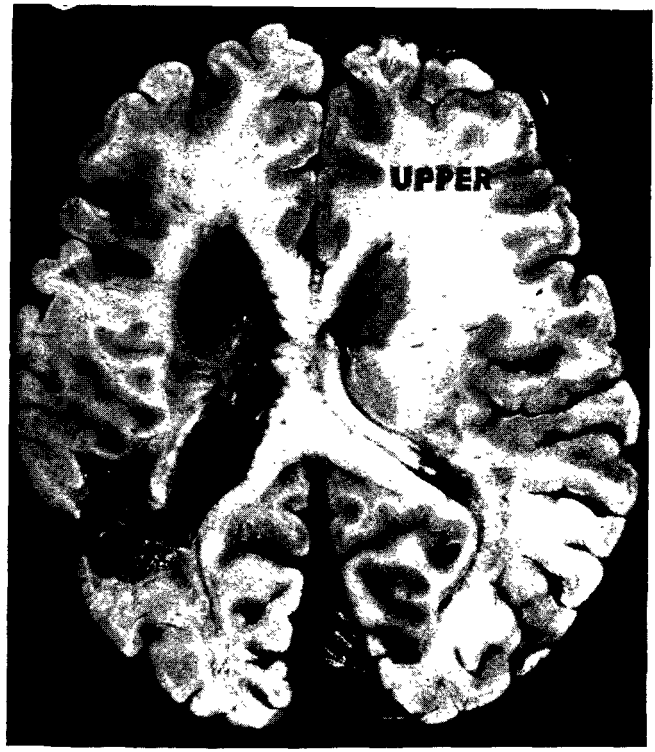

FIG. 35. First section from the brain of Mrs. A. W. It shows complete destruction of the angular gyrus with a hemorrhagic cyst extending into the lateral ventricle.

always made mistakes and always gathered information incompletely and imperfectly, and therefore felt excluded from much of what went on about her. Evidence of visual defect which might be interpreted as hemianopia could not be obtained.

This was her condition up to a point just prior to her last illness. On the morning that she was taken to the hospital her daughter thought she did not look as well as formerly and advised her to remain in bed. When she went to work she said "good-bye" to the patient, who replied: "I'll be all right. I'll get up after a while." During the morning she was found unconscious; she did not regain consciousness.

Autopsy. The left temporal lobe was practically nonexistent, except for

the tip. The left angular gyrus was replaced by a hemorrhagic cyst, causing the left lateral ventricle to communicate with the surface. The ventricle was dilated (Fig. 34 to 37).

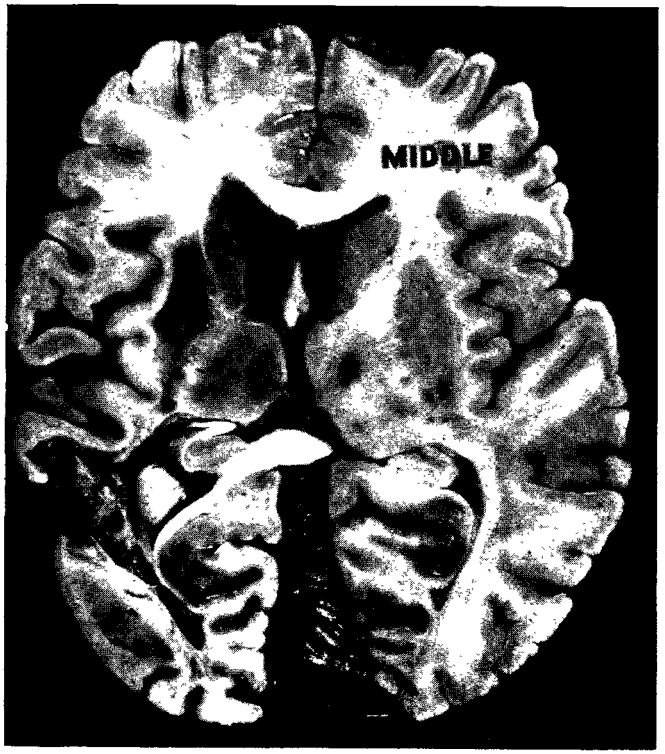

FIG. 36. Second section from the case of Mrs. A. W. The continuation of the angular gyrus lesion is evident. There is also a lesion in the left putamen.

The fact that the patient never learned to write with the left hand is ample proof, if any is needed, that she was right-handed. As writing is the most complex function of language, it is not strange that this was the function which was not learned. It is remarkable that she learned as much as she did. The most surprising fact was her ability to read with absence of the angular gyrus. Henschen has observed such cases, but they are rare. We must postulate that the right angular gyrus was able to take over the function of recognition of letters and words in this case.

(Case reported by Nielsen and Raney in the *Archives of Neurology and Psychiatry* 42:189.)

8. *Jargon aphasia, agraphia, apraxia of the right hand of the ideokinetic type, amnesic aphasia.*

William H. Bohrer, aged 70 years, had had a "stroke" a week before, and upon examination had apraxia of the right hand and some weakness.

Introductory. "Good morning, what is your name?" "My name? I know.

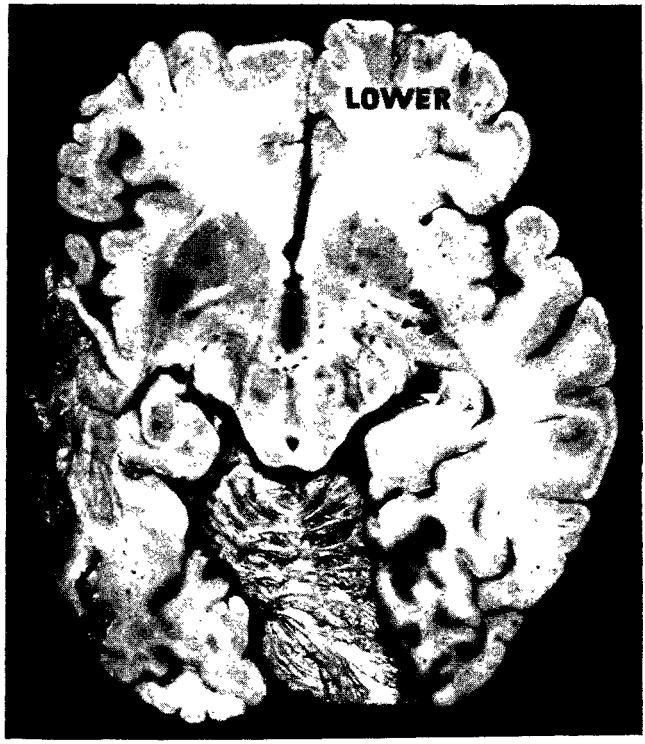

FIG. 37. The lowest section in the case of Mrs. A. W. Destruction of the temporal lobe is evident.

You, Bohrer." "What is your wife's name?" "My name is, Oh, Now it is one of the two weaknesses. I know perfectly well, know her name and everything but don't know where she lived. I see you and see everything in the world but can't tell everybody else where the city is."

It was clear that we were dealing with a case of marked paraphasia.

Visual agnosia. When he was shown written matter he immediately began to read aloud. He was urged not to read aloud but to keep still and merely do what the written matter said. He then carried out some written requests but failed in about one third of the trials. When shown the name "Bob Fitzsimmons," he held up his fists. When shown the name of the president, he tried to talk but failed to say anything. He was then asked, "Is he the

vice president?" He said, "No. He is the main chief, the big cheese." The patient read the papers.

It was concluded he had no visual verbal agnosia.

Acoustic verbal agnosia. We had already seen evidence that he understood most of what was said. We asked, "*What is your wife's name?*" again. This time he said, "I can tell all her name, can tell her first name, last name, but can't spell her last name." We asked, "What is her first name?," to which he answered, "Her first name is, let's see. I am somewhat excited right now. Her name is not. For God's sake, I can't right now. I can tell her for a moment." He held up his hand (left) on request and stuck out his tongue.

It was concluded he had no acoustic verbal agnosia but much amnesic aphasia.

Apraxia. The patient had clearly a localized apraxia of the right hand of the ideokinetic type. Whenever he tried to do anything with it he failed to grasp the thing he intended to take. Sometimes the hand flung out so wildly that the movements resembled those of chorea. He was totally unable to write with it. With the left hand he was able to write and to draw but much more poorly than does the average person.

It was concluded that he had ideokinetic apraxia of the right hand.

Aphasia. The patient clearly did not have motor aphasia. Nor did he have transcortical sensory aphasia. Amnesic aphasia has already been determined. He recited the alphabet perfectly on command. When we asked him, "What sort of work did you do?," we got a volume of paraphasic jargon. He said. "I tried to Camp Superior. That isn't correct, but I am a district agent. That is her name. Don't tell him. I know what it means but I can't tell exactly what it is, but I have been a thousand years at it." (We failed to test singing.)

Amnesic aphasia. We already knew he had amnesic aphasia. We could, however, show that he recognized all objects by the fact that he selected from a mass of objects anything named. This also corroborated our impression that he had no acoustic verbal agnosia.

Semantic defects. When the case was first studied, the patient was easily confused by complicated statements. After four weeks he told us that he was clearly oriented, had in mind much more clearly what we spoke of, could recall stations to which he formerly dispatched trains, etc. We were therefore certain of the presence of semantic defects at first.

Writing. The patient was able to copy in print a few letters with the left hand. This was the sum total of his ability. Never at any time was he able to write letters with either hand. He was not even able to copy with the left hand.

We concluded that he had complete agraphia.

(Calculation was not tested.)

9. *Amnesic aphasia, paraphasia, paragraphia, paralexia, semantic defects. Occlusion of lateral sinus.*

Elaine Bar was a young girl of 18 years who had been suffering with otitis media on the left in the course of which she developed slight chills. Because of this sign and a rise of temperature, a diagnosis of thrombosis of the lateral sinus was made, and the left jugular vein was ligated. After ligation of the vein the otologist proceeded to operate on the sinus but found that it was not thrombosed. The patient then developed aphasic symptoms. The eyegrounds were normal. There were increased deep reflexes and a positive Babinski sign on the right.

Introductory. "Good morning. How are you?" The patient said, "All right, my after was here head all right." This was clearly paraphasic speech, and one was to expect symptoms referable to the left temporal lobe. However, it was decided to try for visual agnosia.

Visual agnosia. Shown a watch and asked what it was, the patient said, "Onion, no pencil, no, no." Asked, "Is it a clock?" she replied, "Yeh, no." We suggested a watch, to which she said, "Yeh, that's what it is, a wash, wash, washing machine." Then she was distinctly distressed and said, "I know what it is. Honest. I know." Next she was shown a white coat and asked what it was. She said, "I know what it is, dress, dress, no, white . . ." Then she said, "I know what you know but you don't know." When we handed her a pencil she recognized it and wrote with it. She was unable to name any streets in Glendale where she then lived or in the last city in which she lived for many years. She could not name the states bounding California, although she was a brilliant student in school.

She was asked to read the sentence, "Amazing facts about salt." She said, "Amazing perhaps about salt." When asked to read it again, she read, "Amazing perhaps about perhaps."

We concluded that the patient had no visual agnosia but, on the other hand, marked amnesic aphasia.

Acoustic agnosia. She comprehended imperfectly what was said to her. Questions had to be repeated several times, although her attention was good and she was embarrassed at not understanding easily.

This indicated that the major area of Wernicke was out of function and that the minor side was in use.

Apraxia. She was unable to protrude her tongue, show her teeth, or grasp an object on command. However, she took a pencil and wrote when asked. She drank and fed herself without difficulty.

The inability to protrude the tongue, etc., would ordinarily be taken as

evidence of apraxia, but we have established that she did not grasp clearly what was said. The fact that she was seen to use objects as she chose and had no difficulty in performing purposeful acts when she understood what was wanted showed that the defect was one of comprehension and not one of apraxia.

Her general orientation was shown by the following:

"What state is directly north of California?" "I don't know."
"What state is south of Washington?" "Washington is not a state."
"Is it a city?" "It's not a city."
"Is it a nation, ocean, kingdom?" "No."
"Is it a state?" "No."
"What is Spokane in?" "In Washington."
"Is Washington a state?" "No."
"What state is south of Washington?" "Idaho. No, west. No, east. I used to live in Idaho."
"What state do you pass through in going right straight south from Washington to California?" Patient is unable to recall.
"Who is president of the United States now?" "Where at what Idaho?" (Complete fatigue of temporal lobe.)

Writing. She wrote her name correctly. She could not think of anything else to write. She was asked to write Alabama. She did correctly.

"Now write 'Michigan.'"	"Kineel."
"Try again."	"K. . . . .
"Now write 'New York.'"	"New York."
'Write 'Tennessee.'"	"Tenecesse."
"Write 'Pennsylvania.'"	"Pennecesslya."

This showed performance of the minor side.

The patient calculated quite well with small arithmetical processes.

In the course of four days all the symptoms cleared up, and the patient made an uneventful recovery. The syndrome was ascribed to vascular stasis produced by the sudden stoppage of circulation on the left side, particularly in the temporal region and in the area for formulation of language.

10. *Auditory verbal agnosia. Visual agnosia for inanimate objects while recognition and revisualization of animate objects was retained. Autopsy showed lesions of the left temporal lobe and the right occipital lobe.*

C. H. C., an unusually well developed and muscular self-made man of 85 years who had during most of his life despised scientific medicine, was well until Dec. 21, 1937, when he awoke at 3 in the morning unable to move his lower limbs. During the previous evening he had taken his usual walk of

several miles about his estate for the exercise which he believed was the basis of his marvelous constitution.

He was seen by the writer on the fifth day because of delayed improvement. He was at that time able to sustain his weight on his lower limbs and barely able to walk a few steps. Physical examination showed a man 187 cm. tall, weighing about 90 kg. though he was lean. There was a well developed arteriosclerosis; small rings of calcium salt deposits were felt in the radial and brachial arteries. Fundus examination showed sclerosis of the retinal arteries. Blood pressure was 190 systolic and 95 diastolic. The heart was slightly enlarged to the left. Aside from these facts and motor weakness of the lower limbs without sensory loss nothing worthy of note was found. Babinski signs were present bilaterally and the knee jerks were increased while the achilles tendon reflexes were absent. It was impossible to determine whether the apparent thrombosis had occurred in the spinal cord or in the vertex of the cerebrum bilaterally although the former was considered more likely.

The patient refused intravenous fluids to facilitate resorption of the clot; and, though he consented to take potassium iodide by mouth he changed his mind after four days and refused further medication. After a week's observation he could no longer be induced to remain in the sanitarium, but during the night before his contemplated departure another vascular accident occurred and the patient awoke with a severe paraphasia. This turn of events induced him to remain and accept intravenous fluids for about a week, and he remained two additional weeks awaiting air-conditioning apparatus which was being installed in his home. He then went to his estate where it was found difficult to induce him to remain in bed.

Just as this was becoming impossible he suffered an attack of anginal pain and dyspnea. An electrocardiogram taken then showed evidence of coronary thrombosis. He consented to remain in bed, but insisted on so much exercise in bed that he suffered two more attacks of coronary pain and his cardiovascular system became so weak that the act of standing caused a systolic blood pressure drop of 70 mm. of mercury. This evidence threw the balance for him and he remained quiet.

One morning in March 1938 the patient had a "dizzy spell" after which he said he could not understand what he saw printed. Examination disclosed visual verbal agnosia and agraphia except for retained ability to write his name. He could see print and could spell the words by reading a letter at a time but they meant nothing to him. He accepted this philosophically, saying he did not "need to read anyway."

In April it was suddenly discovered that the patient had an upper right quadrantanopsia. There was no recognizable episode of headache or dizziness.

The course was in general downhill. The next episode of dizziness left him with astereognosis in the right hand. To this point the diagnosis was thrombosis of a small artery in the temporal lobe close to the insula, of the angular gyrus branch, and of the left parietal branch, all in the distribution of the left middle cerebral artery because his right hand had become weak. In May 1938 he had a more severe attack which left him with a complete left hemiplegia. He was stuporous for a time but as soon as he was able to co-operate he recognized his left arm and also the paralysis (no anosognosia or delusion of body scheme). A capsular thrombosis was diagnosed. (This was shown at autopsy to have been in the right uncinate region.) From this time to his death the patient was subject to outbursts of violent rage. In June another attack left him with an added left hemianopia so that only the lower right quadrant remained in which he could see.

Accurate evaluation of the situation could not be made for several weeks because of his critical condition. When it was possible to make accurate determinations it was found that he no longer recognized objects or persons by vision alone, though he was keen in recognition of persons by their voices. Through the summer he gradually improved and the right visual field defect disappeared, in spite of repeated brief syncopal attacks in which the nurses thought he would surely die. As he improved, strength gradually returned in the left limbs until he could again use the left hand to hold food.

In September he had another episode which left him with inability to comprehend spoken language except for brief terse statements or questions and even these for only a fraction of a minute. He behaved regarding comprehension of spoken language exactly as do patients who have had their major temporal lobes removed surgically. This condition never changed radically though it improved somewhat.

In November the course was steadily downward and his death was expected during that entire month and part of December. When the middle of January 1939 was reached he began to eat of his own accord which he had not done for two months and he greatly improved. A study late in January showed the following: The patient understood simple spoken phrases and short sentences but tired rapidly (as do minor temporal lobes). Vision had returned in the upper right quadrant. He recognized nothing by vision alone if he was tired at the time; if he was tested when fully rested he recognized persons (his attorney, Mr. Wood; an old friend, Dr. Averill; his adopted son, and often his doctor) but never any inanimate object. It was very striking that he recognized a set of artificial teeth, fingers, arms, etc., but not objects other than parts of a body. He could count fingers but with some difficulty because of the left homonymous hemianopia. He was able to revisualize persons but not inani-

mate objects. He could not revisualize his estate or even a large reservoir which he had built himself and of which he had been inordinately proud for years. He had often invited inspection of it by engineers who invariably praised it (justly) but even this work he could not recall. Neither could he recall any part of his estate, the air conditioning recently installed, the rooms, the city in which he lived, the mountains or any other scenery. When a derby hat was held up before him he was unable to recognize it, even when he was allowed to hold it (he asked to feel its weight). He similarly failed to recognize a desk telephone by sight or by touch or both. (Amnesic aphasia was eliminated as the cause of the confusion by asking him the use of the objects in question. He had no suggestions.) He did not recognize food until he tasted it; he always demanded to have the food named before accepting it in his mouth and he recognized it by name (one word at a time). Automobiles seen through the window were strange to him and he asked what "those things" were. A bottle of milk and his drinking glass were unknown objects. He failed to recognize an airplane by sight or by sound.

Yet with all this visual disability relative to inanimate objects he recognized and revisualized all living things. He knew his most intimate friend (Mr. Wood), his doctor, and the six nurses in attendance, even recognizing them by their names as well as by sight alone. When he could not recall owning his estate he nevertheless knew his housekeeper and if her name was mentioned he immediately revisualized her person. Discussion of former friends by names brought vivid recollections and he would ask about relatives and their children, whether they were married, etc., whether former business partners were still alive and what they were doing.

When it became so evident that he was drawing a sharp line between animate and inanimate objects, both for recognition and revisualization, a flower was presented and he was asked what it was. He immediately named it (a daffodil) correctly. He knew other flowers also, and not only identified them, but named them. He was thus categorizing flowers as animate objects. Still he could not name colors though he apparently recognized them.

As his right quadrantic visual defect had disappeared it was thought that perhaps the left angular gyrus lesion had diminished. It was found on testing that he could read single words and could write single words again (able to revisualize them). His ability to read words was present while he could not recognize any inanimate object. This condition persisted for more than six months. By September 1939 he was again failing.

From this point onward his general condition became so poor that further study was impossible. He developed pneumonia and died Nov. 15, 1939.

Autopsy showed a severe generalized arteriosclerosis, which was most

marked in the coronary arteries and aorta, and bronchopneumonia. The cerebrum showed as a result of the arterial sclerosis a thrombosis of the sylvian artery on the left and moderate softening of the left temporal and parietal

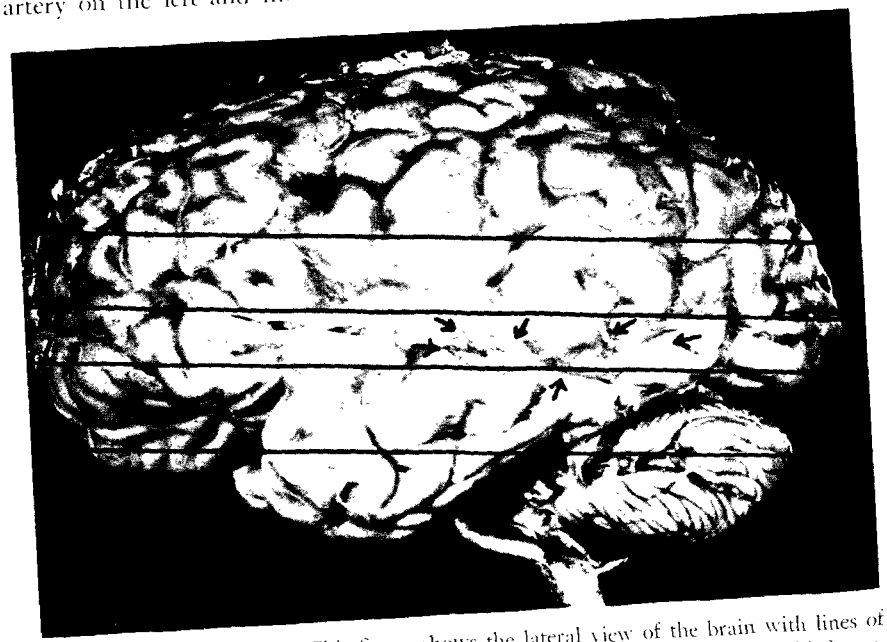

FIG. 38. Case of C. H. C. This figure shows the lateral view of the brain with lines of section. A band of softening is seen outlined by arrows in the second and third temporal convolutions 4 cm. long by 1½ cm. in vertical height extending from the middle of the temporal lobe back to the temporo-occipital notch. The apparent lesion in the cortex of the angular-supramarginal gyri is an artefact. A small lesion is seen in the lateral aspect of the cerebellum. (From the *Bulletin of the Los Angeles Neurological Society*, 5:135.)

lobes but this was at the most a few days old and had no bearing on the clinical picture which had been studied.

The lateral aspect showed on the surface a band of old softening with yellowish discoloration (in the fresh specimen) in the second and third temporal convolutions. This extended forward from the temporo-occipital notch for a distance of 4 cm. and it was 1½ cm. in vertical height. On the inferior surface of the right occipital lobe was a sunken and scarred area 1 cm. in diameter. The hippocampus on the right showed a small old cystic area of vascular origin.

The brain (Figs. 38 and 39) was sectioned horizontally on lines parallel to the usual base line which is made to pass through the center of Broca's convolution and Wernicke's area in order to have the most important speech areas

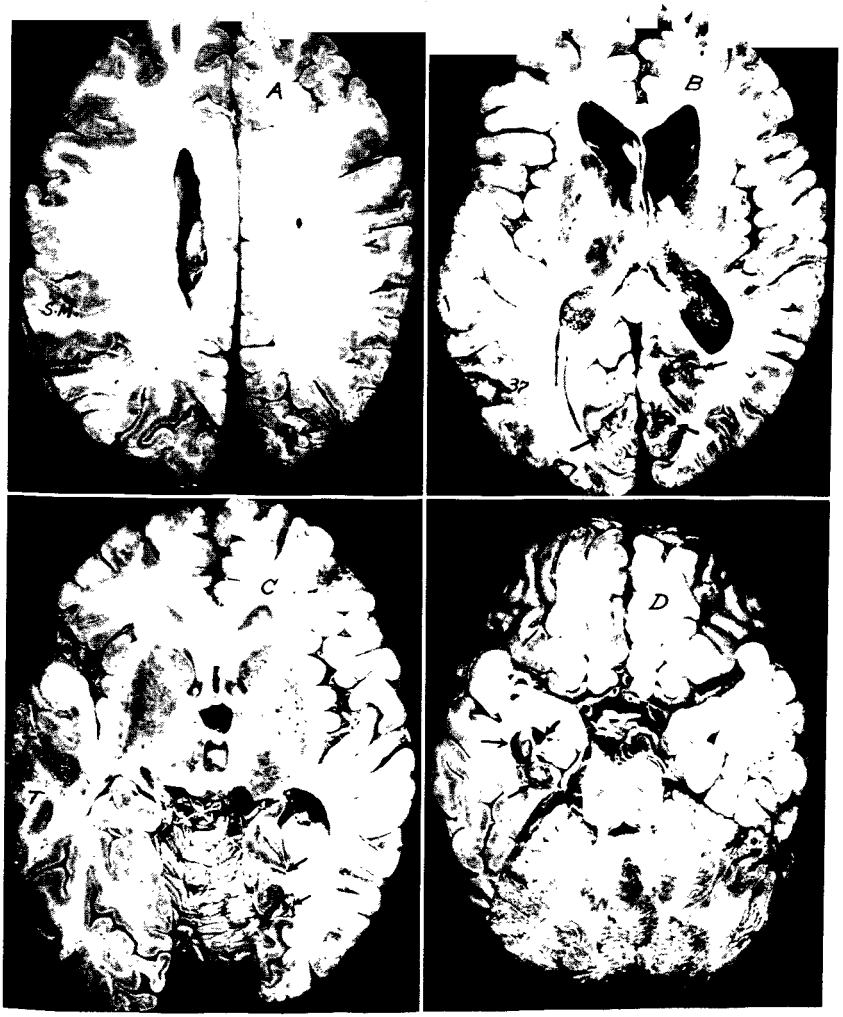

FIG. 39. Case of C. H. C. These four sections are of the brain in Fig. 38. In *A* is seen subcortical softening in the left supramarginal and angular gyri. In the right occipital lobe is seen a lesion indicated by the *arrow* (areas 18 and 19 of Brodmann), subcortically placed in the cuneus so as to destroy completely the function of that structure. In *B* are seen three lesions: The first is cortical in the left area 37 of Brodmann which is the posterior extremity of the softening in the left temporal lobe. The second is a very small lesion in the left occipital lobe (*arrow*) cortical in area 17 (in the lingual gyrus) and hence affecting the visual projection fiber system, not association tracts. The third (*arrow*) almost completely destroys the lingual gyrus on the right side, anteriorly destroying areas 18 and 19 also (association tracts). In *C* are seen three lesions. On the left side the first destroys the temporal lobe (*TT*) to within 1 cm. of the lateral ventricle (the anterior end escaping). The second destroys the lingual gyrus on the right anteriorly to the posterior horn of the lateral ventricle which is dilated in consequence. This affects association areas (areas 18 and 19 of Brodmann). The third is a small and insignificant lesion in the right prefrontal cortex. In *D* the photograph is taken looking up and the cystic lesion (*arrows*) in the right hippocampus is on the left side of the figure. The slight cortical lesion of the left cerebellar lobule (*arrow*) is visible on the right side of the figure. (From the *Bulletin of the Los Angeles Neurological Society*, 5:135.)

on one plane. This passed through the posterior part of the softened band seen on lateral view. It disclosed a small diamond-shaped lesion mesial to the posterior horn of the lateral ventricle below the calcarine fissure. Its position in this location precluded its interference with associative functions. On the other hand the right occipital lobe was seen to have its entire lingual gyrus destroyed, the occipital pole escaping. There was also a softened area corresponding to the posterior extremity of the lesion seen on lateral view.

The next section was made 1½ cm. above the base line and showed cortical and subcortical destruction of the angular and supramarginal gyri. There was no lesion of the left occipital lobe, but complete subcortical destruction of the right occipital (areas 18 and 19 of Brodmann).

The third section was made 8 mm. below the base line and showed an intact lingual gyrus on the left but complete destruction of the lingual gyrus on the right extending to the posterior horn of the lateral ventricle. It also showed destruction of the left temporal lobe to a depth of 2½ cm. An incidental lesion was found in the right prefrontal area, small and of no clinical consequence.

The fourth section was taken 2 cm. farther down (2.8 cm. below the base line). This showed a cystic destruction of the right hippocampus but an intact left temporal lobe in its inferior portion.

Comment. Clinical correlation with the pathologic lesions seems entirely possible. The attack of alexia from which the patient did not recover was due to the subcortical lesion in the angular gyrus. The next episode was manifest by upper right quadrantanopsia and this was due to the one small lesion in the left lingual gyrus. Recovery from the symptom is understood in consideration of the small size of the lesion which caused edema at the time but left only a very small permanent defect which I did not discover because only finger tests of the fields could be carried out. The auditory verbal agnosia is clearly explained by the severe left temporal lesion. The permanence of this symptom shows that the right temporal lobe could not assume the function of the left at the age of 85. The two lesions in the right occipital lobe came at about the same time, i.e., the destruction of the visual cortex itself and the lesion in the association area shown in Fig. 39, *A* (areas 18 and 19 of Brodmann). This explains why with the left homonymous hemianopia the patient also developed visual agnosia for objects. The left hemiplegia from which he recovered was due to the lesion in the right uncus with extension of the edema and circulatory effects of the right internal capsule. This was a severe lesion at the time.

Care must be taken not to ascribe any occipital associative functional loss to the lesions of the visual cortex itself. It is well known that even with complete bilateral destruction of area 17 of Brodmann (area striata) visual memories

are not interfered with. But destruction of the area parastriata or peristriata, whether cortical or subcortical, causes loss of the associative functions. Careful scrutiny of the sections shown reveals that in the left occipital lobe the associative areas were intact while on the right they were destroyed. The lesions indicated by the arrows in Fig. 39, *A* and *C*, are in the associative areas. The right occipital lobe was therefore the major one for nonlanguage associative functions.

Case reported by Nielsen in the *Bulletin of the Los Angeles Neurological Society* 3:135.)

11. *During drainage of a lung abscess sudden asphyxia from welling of pus into nose and throat. Visual agnosia for animate objects with retained recognition of colors. Death. Autopsy: cortical destruction of the left occipital lobe; two small parietal lesions.* (Los Angeles County Hospital, No. 781–866.)

Flora D., a legal stenographer aged 46 years, was admitted Oct. 17, 1941, following an illness of a month with chills, fever, nausea, vomiting, and cough. She had been treated privately for influenza and was referred to the hospital when the sputum became purulent. She had had pneumonia twice, at the ages of 7 and 9. Other history was irrelevant.

Physical examination revealed a seriously ill patient with findings not pertaining to the present study but diagnostic of pulmonary abscess of the left upper lobe. On November 26 an operation was performed for drainage of the abscess. Suddenly a large quantity of pus welled up into the patient's nose and mouth and she became cyanotic. Immediate treatment was given; the table was lowered into the Trendelenburg position; a tracheal catheter was inserted, and breathing was re-established. However, in spite of oxygen therapy the patient remained cyanotic for the remainder of the day. Neurologic consultation was requested.

On the second postoperative day the patient did not voluntarily look to the left, but the eyeballs did turn reflexly. Visual acuity was impaired and there seemed to be a right homonymous hemianopia. The fundi were negative and the pupils were equal. They reacted well to light and on convergence, though convergence was obtained with difficulty. For several days there was a definite left hemiplegia which was denied by the patient. When she wished to move the left arm she picked it up with the right hand. She failed to recognize by touch any object placed in the left hand though she could do so in the right. On several occasions she denied ownership of the left hand. The deep reflexes were all increased on the left side.

On the 1st of December the patient lay in bed quietly looking directly at the ceiling. She did not appear to observe her surroundings. When two fingers were held up before her on the right side and she was asked how

many fingers there were she said, "two." However, when the number was increased she continued to say, "two." And when the hand was withdrawn she still insisted that she saw it. When two hands were held up before her she saw only one.

She correctly identified a penknife, a watch dangling from a chain, a pen, and a pencil, though she failed to recognize a key and a coin. She could read a few printed words. Because of perseveration tests had to be made in stages.

Reflection over the observations led to the suspicion that the patient failed to recognize our hands because they were parts of the body. We then studied the case for autotopagnosia and found that she not only failed to recognize hands, fingers, and laterality by vision alone but failed to recognize any living thing. When her own artificial teeth were shown to her she failed to identify those also. She could not recognize that any person's face was a face though she said that it was pink. When a doll was shown to her she did not know what it was but described the color.

Revisualization as well as recognition of inanimate objects was good. She described the local city hall, streets, and directions. She also revisualized the colors of a canary bird, the ocean, etc. We neglected to test for revisualization of animate objects until it was too late. She died after two weeks.

The brain was examined after preservation for two weeks in formaldehyde solution. The vessels were not sclerosed. There were only two external markings worthy of note. The left occipital lobe showed a cortical discoloration over the entire convex surface except the pole. The inner medial surface was not affected. In addition there were two small lesions just anterior to the parieto-occipital sulcus close to the medial plane. These are shown in Fig. 40. The lesions were almost identical with those commonly seen after carbon monoxide asphyxia. Sections failed to show any other lesion.

Comment. This is the second case known to the writer in which the only clinical finding was visual agnosia for animate objects with retained recognition of inanimate ones. In the first there was a focal lesion partly cortical and partly subcortical in the left occipital lobe. It affected only the association areas, not the projection fiber tracts. The case of Flora D. is similar, except that the lesion affected diffusely the convex cortex of the left occipital lobe. Inasmuch as areas 18 and 19 of Brodmann were cortically destroyed on the convex surface and as the patient failed to recognize animate objects while still able to recognize inanimate ones, it is tempting to postulate that the inanimate objects were recognized by means of the right occipital lobe. In a case previously reported (by the author) the patient showed the reverse of this clinical picture through destruction of the right occipital lobe, i.e., he failed to recognize inanimate objects, but he recognized animate ones. The three cases agree on one

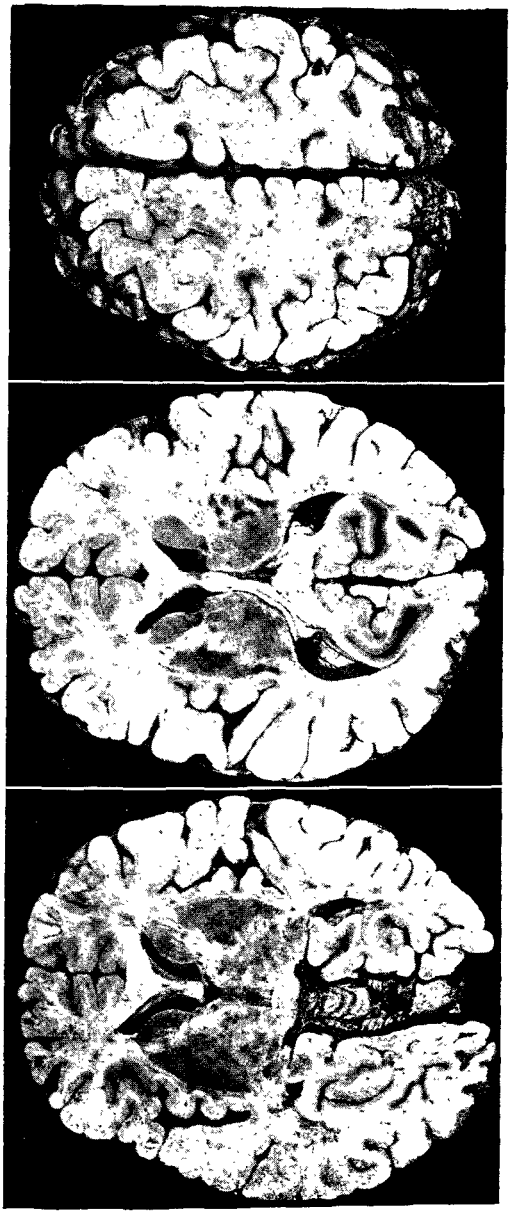

FIG. 40. Brain of Flora D. (visual agnosia for animate objects). The uppermost figure shows a focal area of softening in each parietal lobe at the right border of the section as seen in the illustration. The other two sections show cortical destruction of the left occipital lobe (lower right-hand portion of the figures). (From the *Bulletin of the Los Angeles Neurological Society*, 7:102.)

point, that one occipital lobe may serve in recognition of animate objects while the other serves for inanimate ones. Yet in spite of this correlation we do not feel justified in drawing general conclusions because there are many cases on record in which, in spite of destruction of one occipital lobe, there were no symptoms of deficit whatever; either lobe was able to function for all associations.

It is clear that in cases of visual agnosia for animate objects one cannot speak of Gerstmann's syndrome, of anosognosia, or of delusion of absence of the minor limbs because these are all subdivisions of the general condition.

(Case of Nielsen and Sanborn, 1942.)

12. *Severe arteriosclerosis and hypertension. Acute episode of left hemiplegia in a left-handed but "right-handed writing" man with aphasia. Recovery in three weeks from hemiplegia and aphasia. Seven months later patient awoke in morning with right hemiplegia but no aphasia. Examination showed complete agraphia for letters and words with the right hand only. The patient was able to write well with the left hand. No other elements of aphasia.*

William R. M., a white man of 68 years, had been employed as a banker during the greater part of his life and was known to have severe arteriosclerosis and hypertension before the episodes which marked his illness to be described. In February 1937, according to the history given by himself and corroborated by his wife, he suffered an episode of left hemiplegia with complete motor and sensory aphasia. He was unconscious for several hours and afterwards his legs were too weak to carry him. He made a complete recovery from aphasia in twenty-four hours and from the hemiplegia in three weeks. In answer to questioning he stated that he had been left-handed at birth but that his teacher in school had made him learn to write with the right hand. He had remained left-handed for all other skilled acts but since the age of 8 years had never written with the left hand.

After that episode, he became as well as usual and gradually increased his ability to walk until he was able to tramp 2 miles daily. He lived on Catalina Island and attended baseball games quite often. On Sept. 4, 1937, he attended a baseball game as usual but left in the middle of the game because he became extremely "nervous." When he reached home, according to his wife, he was "hysterical" and continued so for several hours. He gradually calmed down but was not quite himself even on the following day.

On the morning of September 6 he awoke with a right hemiplegia and was taken to the Glendale Sanitarium where he was seen by the writer on the 11th. Examination showed a marked sclerosis not only in the peripheral arteries but also in those of the fundi of the eyes. Blood pressure which had

been 248 systolic and 130 diastolic four days before had come down under treatment to 194 and 120 respectively. There was evidence of myocarditis and the pulse rate was 120 per minute. The lungs were negative as were the abdominal viscera.

Neurologic examination showed the pupils to be equal and reactive to light and on convergence. There was no evidence of increased intracranial pressure. Fields of vision were normal. There was a right hemiplegia affecting the lower limb most, face next, and arm the least. The patient was able to hold a pencil in the right hand with sufficient force to draw and attempt to write. The hand grip measured by dynamometer was 95 on the right and 140 on the left. On the other hand there was a distinct hemihypesthesia on the entire left side of the body. There were bilateral Babinski and Chaddock signs but neither Gordon nor Oppenheim signs were present.

Examination from the standpoint of aphasia revealed the remarkable fact that the patient, unknown to himself, had a complete agraphia for letters and words affecting the right hand only. There was no other element of aphasia. When asked whether he could write he replied, "Certainly," and took the pencil. To his astonishment he was unable to produce a single letter. He made a few straight lines and curves. He was then persuaded against his protest to write with the left hand and found to his surprise that he could do that very well. He wrote New York, and California, without the least hesitation. When this had been done he was nevertheless unable to copy with the right hand what he had just written with the left one. He was then tested for ability to write figures with the right hand. He was able to write 1, 2, 6, 3, 5, 0 on command. Strangely, when he at first attempted to write 6 he tried to write six. He succeeded in writing so that one watching the process could detect what the letters were supposed to be. After all this effort he also succeeded in writing N and Y of New York, but each letter came only after a severe struggle.

In contrast to all this difficulty with writing he was able to read *without* the least trouble. He also was able to cut with scissors with the left hand very skillfully. He was able to draw human faces and geometrical figures with either hand though better with the left one. Calculation, especially in view of the fact that the patient had been a banker, was much disturbed. He insisted that $3 + 4$ were 14 and then later "corrected" the answer to 12. After a little practice, however, 7×8 were 56, 2×6 were 12, and 3×4 were 12. There was no apraxia. Lighting a match, feeding himself, cutting pictures and drawing and other similar acts were unhesitatingly performed.

When he was seen again three days later the hemiplegia had greatly diminished under intravenous fluids and lowering of the head of the bed. However, there was no improvement in his ability to write. He was examined every few

days and on the twelfth day he again was able to write. The lower limb remained severely paralyzed.

Comment. This most striking case throws considerable light on the cerebral physiology of language in this instance. This patient's language area was on the right side of the brain from birth. When he suffered a left hemiplegia, the left hemisphere did not take over the function of language; it was the right which again took up the work after recovery. If this were not so he could not have escaped with a mere agraphia from a later lesion on the left side. Neither would he have had aphasia with the left hemiplegia. We can conclude, then, that in spite of his being compelled to write with the right hand and developing a writing center on the left side of the brain the remaining language function did not develop on the left side—even with sixty years of training.

As to his cerebral mechanism for writing we are compelled to postulate at least two separate writing centers, one on each side of the brain. During his normal adult life the one on the left side of the brain must have had available to it the entire knowledge of language stored on the right side of the brain, because a writing center can be constituted only of the engrams for the movements necessary for writing and not of all knowledge of language. The writing center on the left side must have had functional contact with the language area on the right side via the corpus callosum.

Now, when a lesion on the left side produced hemiplegia and agraphia, the writing center on that side must have become separated from its connections with the general area for knowledge of language on the right side by a lesion of the corpus callosum. The patient then could not write with the right hand but could still write with the left. This concept is further supported by the fact that he was able to write figures. The left-sided writing center was thus separated from one area without being separated from the other.

(Case reported by Nielsen in the *Bulletin of the Los Angeles Neurological Society*, 1:73.)

13. *Apoplectiform seizure followed immediately by disturbance of estimation of distance, simultanagnosia, lower right quadrantanopsia and upper right quadrantachromatopsia, disorientation in space. Second seizure with total blindness and loss also of sense of time. Third seizure with right hemiparesis, unconsciousness, then death. Autopsy.*

S. E. H., a white man aged 66 years, was admitted to the hospital March 3, 1936 complaining of dizziness and headache of three weeks' duration. The history was to the effect that for about a year his memory had not been so good as formerly, but this defect had not interfered with his work, which consisted in collecting clothing to be dry-cleaned. It had also been his duty to return the garments to their owners and to keep his accounts. He had inci-

dentally dabbled in real estate; and, while his education had not been extensive, he had been apt at mathematics and at the time of onset of his illness had been able to do calculation very well.

He was well and at work in the middle of February 1936. One day while standing in a bank making deposits or drawing money (he could not recall exactly what he was doing), he felt a sudden sensation in his head which was like that of a "fuse blowing out" immediately upon which he was unable to see for a few moments. The complete blackness soon cleared, and he was again able to see about as well as before. His friends seem to have noticed something wrong with him, however, as they offered to help him home.

His memory for occurrences from that date was extremely bad. No matter how closely we questioned him, he was unable to give any account of his doings, although he remembered scenes and events not involving action. Yet, he had at all times been able to read. This he was sure of, because he had read addresses and directions. He had also read newspapers during his spare time. He had another difficulty, however, namely inability to keep track of the slips upon which were written his instructions. This was because he could not file them and keep them in order.

The major difficulty was his inability to find his way about. The old familiar streets even about his home had become strange to him, and he had become lost on many occasions not only at his work but on his way home in the evening. He could not remember how to get to addresses of homes from which he had taken clothing even though the addresses were written for him on the slips. After three weeks of painful attempts to work, he came to the hospital for treatment.

Aside from arteriosclerosis and hypertension (blood pressure 170/100) there was nothing of note in the general physical examination.

Neurologic examination gave two positive findings: A lower right quadrantanopsia with loss of color recognition in the remaining right quadrant, i.e., upper right quadrantachromatopsia. There were no other findings; not even a Babinski sign was elicitable, and the deep reflexes were symmetrical. Abdominal reflexes were normal. No weakness of the extremities could be found.

Inquiries were then made relative to orientation in space. It was found that the patient had no idea of direction even in his own neighborhood. He was unable to tell how to get from his home to the ocean, although it was only a matter of going along the street for half a mile. He did not know how to get to the locally prominent public buildings from his home. When questioned about the characteristics of his own home it was found that he could not revisualize the structure. He did not know whether the front door was in

the center or on one side. Neither did he know whether the front door gave entrance to a vestibule or to the living room directly. He could not tell whether there were steps leading to the porch. He could not tell whether the house was built of stucco or was covered with boards.

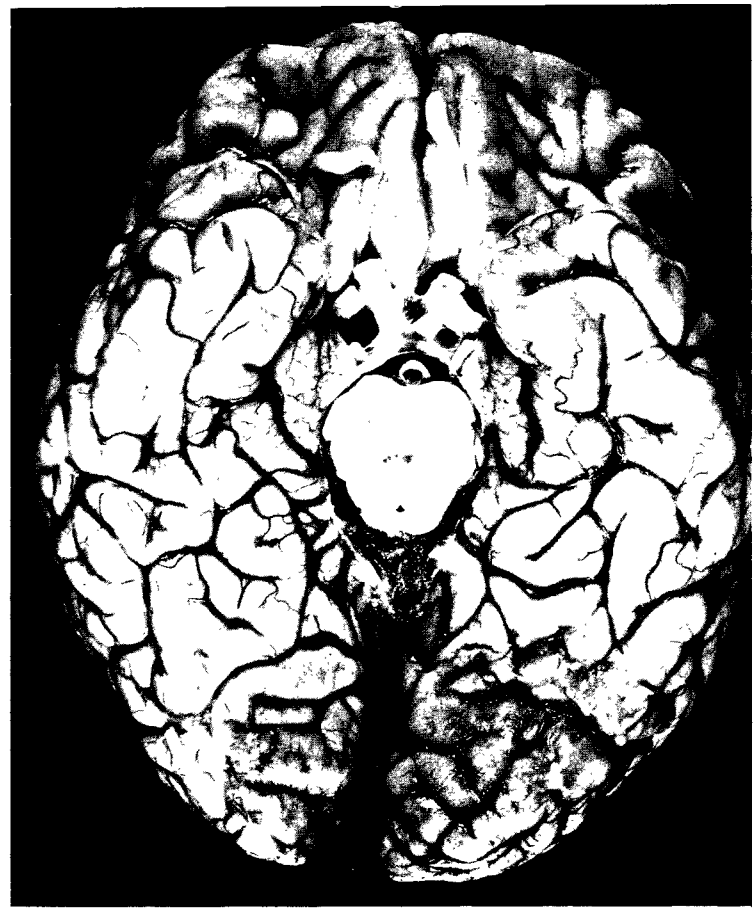

FIG. 41. Case of S. E. H. This shows the base of the brain and the areas of softening in the occipital lobes. On the right side (left of the photograph) the area is small, on the left side, large.

Although he had been well acquainted in the city of Los Angeles also (he lived in a suburb), he could no longer tell how to get from the City Hall to the Hall of Justice three city blocks away. Other prominent places were equally unfamiliar to him. He thus had disorientation in space.

Investigation for simultanagnosia showed that while he was able to read

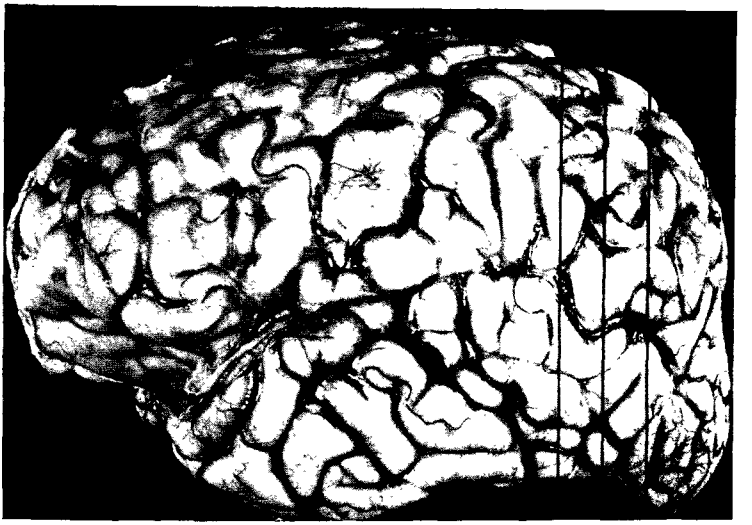

FIG. 42. Case of S. E. H. Lateral view of the brain with lines of sections.

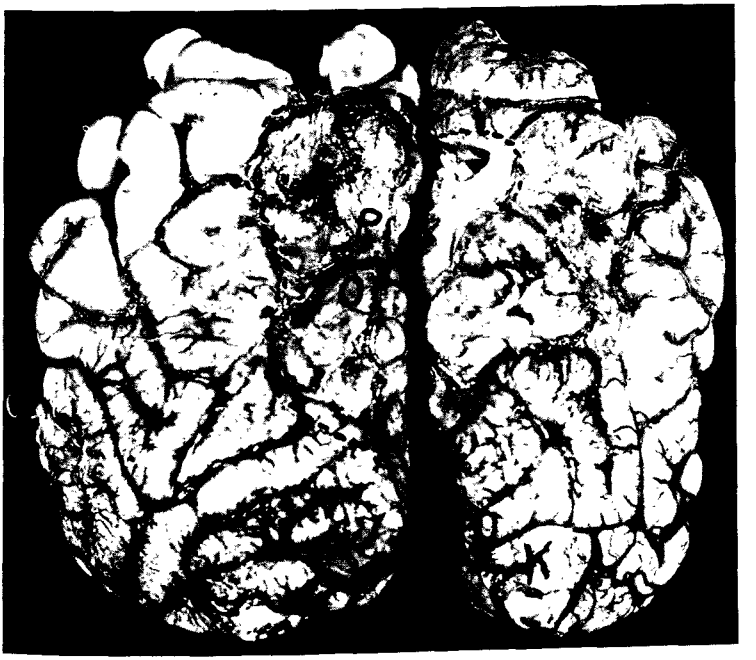

FIG. 43. Case of S. E. H. Photograph of the occipital lobes seen directly from behind. *K* on each side indicates the calcarine sulcus. *P* is just above and *O* is just below the left parieto-occipital sulcus. The area of softening is outlined with a dotted black line. The softening visible from this angle is entirely above the calcarine sulcus. The occipital poles are spared. This section was just 2 cm. thick.

even fine print and was able to identify elements of a picture, he was unable to determine the process going on as portrayed by the illustration. A picture of a hockey game he described as a man bending forward, wearing a red

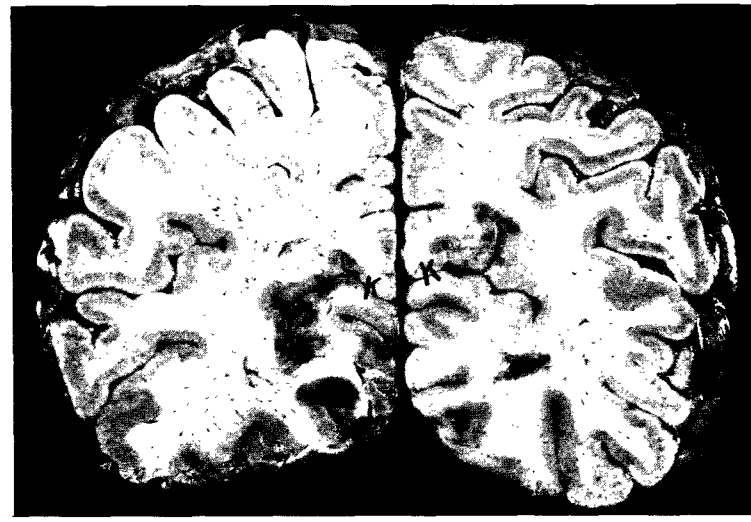

FIG. 44. Case of S. E. H. This section 2 cm. from the occipital pole is 1 cm. thick. *K* is again placed on the calcarine sulcus of each side. *Left side:* Fresh softening is visible along the left calcarine area more in the lingual gyrus (below) than in the cuneus (above). At the inferomesial angle there is also fresh softening while in the remainder, especially along the inferior surface, the softening is old. This last-mentioned softening involves the surface of the fusiform gyrus and the third occipital convolution. *Right side:* The softening is all recent and involves the calcarine area both above and below.

shirt and having something black on the ends of his arms, but he could not tell that the black objects were gloves or that the man was skating or playing hockey. There were other figures in the same picture which he similarly described. When looking at a picture of a circus elephant bearing a mahout on his head and picking up a load with his trunk, it took him a great deal of time to determine what it was that the mahout was sitting on. At first he said it was a mountain. After a great deal of careful inspection, he determined that the large object was an elephant, but he was never able to determine that the elephant was in the act of picking up a log. When shown a large picture of Babe Ruth (a well-known former baseball player) in the act of batting a ball, he studied it in great detail and even stated that he recognized the face, but he could not tell whose face it was, nor could he tell what the player was doing. It was difficult for us to keep his attention on the picture, as he insisted on reading the large and even fine print at the sides of the

picture, because he stated that this was so much easier, and he could easily tell what it said. When shown a picture of a horseman who had fallen with his horse and in which the horse and rider were both struggling to regain their

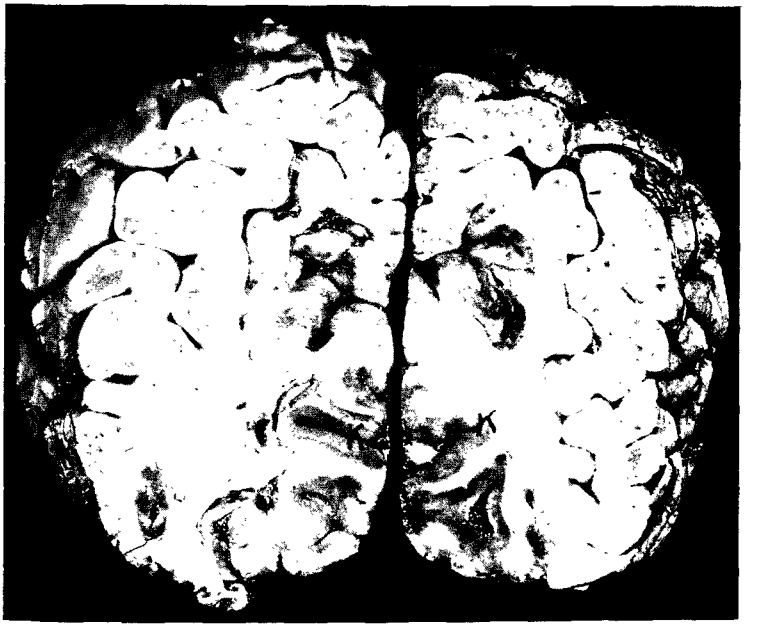

FIG. 45. Case of S. E. H. Section 2 cm. from the occipital pole. *K* again indicates the calcarine sulcus. *Left side:* The entire mesial portion is softened. Above the calcarine fissure the softening is older. Still higher there is softening about the parieto-occipital sulcus. This is a continuation of the areas marked *P* and *O* in the posterior view. The softening of the inferior surface extends to a depth of 1 cm. *Right side:* There is a wedge-shaped area of softening below the calcarine fissure which appears fresh. The only other area of softening is along the borders of the parieto-occipital sulcus above. This is a continuation of the area seen from behind on the first section. This section is also 1 cm. thick.

feet, he pointed out the horse's head, his tail, his feet, and the rider's head and body but did not have the slightest idea of what was going on. He finally suggested that perhaps the man was riding on the horse, but he added that this was only a guess.

It was determined that the patient was unable to revisualize actions previously experienced or observed. He could not recall what he was doing in the bank when his "fuse blew out," nor could he give an account of any of his other actions during the period of his illness.

The patient was able to obey written commands, but when we prevented him from reading the command aloud, it was more difficult for him to com-

prehend. He wrote very well and built words with anagram blocks. His attention in general was excellent. When instructed to build the word Alhambra, he was given a jumble of letters which required considerable hunting.

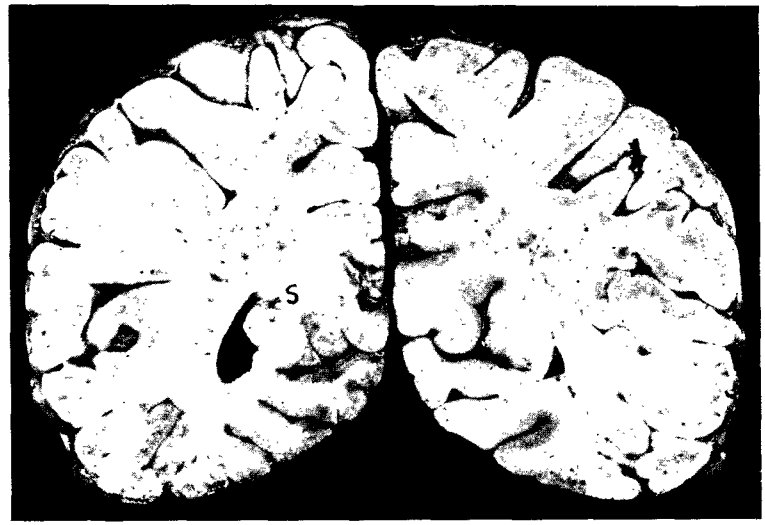

FIG. 46. Case of S. E. H. This section is 4 cm. from the occipital pole. It shows only one small area of softening (at *S*) which is continuous with the splenium of the corpus callosum. It is mesial to the posterior horn of the ventricle.

In spite of a good deal of time lost in hunting for letters, he never lost track of the fundamental purpose, and whenever we asked him what it was he was building, he could answer the question correctly.

He was clearly oriented for time. He knew exactly how long he had been trying to work with his handicap and how long he had been in the hospital.

During the examination it became evident on several occasions that he was not a good judge of distance. When he wished to point to anything on the newspaper he was reading, he would put his finger past it, usually below, and would continue to indicate with his finger as though it were on the paper. When his attention was called to this defect he was able to correct the error. but again made the same one spontaneously.

Laboratory work gave entirely negative results. Serologic tests for syphilis on both blood and spinal fluid were negative.

On the ninth day in the hospital (March 12) the patient had an attack in the morning with blindness, dyspnea, disorientation, anxiety, and a little pain in the chest. Dr. Clarence Olsen thought this to be an attack of coronary oc-

clusion and ordered an electrocardiogram. This showed sinus arrhythmia, evidence of myocardial damage, and coronary disease.

After this episode the patient was given a rest and then studied again for previous findings. He was found to have added symptoms, blindness and loss of sense of time. He no longer knew how long he had been in the hospital, nor how long he had worked after his first attack in the bank. He was, moreover, totally blind. His sight never returned. Still he was able to calculate, and he was still able to revisualize colors. He knew that a canary was yellow, the grass green, and the ocean "water color." He had loss of the sense of position in the right hand. Still there was no semblance of speech disturbance and no weakness of the limbs. It was thought that both posterior cerebral arteries had become occluded.

Two days later, in the early morning, the patient attempted to get out of bed without calling for help. He was unable to walk and sank to the floor. He was unconscious and remained so to his death six hours later.

The autopsy showed arteriosclerosis of the coronary arteries and of the body in general. There were no other essential lesions.

Examination of the brain (Figs. 41 to 46) showed areas of old softening of the borders of the parieto-occipital sulcus of each side and equally old superficial softening of the inferior surface of the left occipital lobe. There was in addition to these lesions fresh softening of the lingual gyrus of the left side and of a portion of both the lingual and lower portion of the cuneus of the right side. There was thrombosis of the basilar artery which was evidently so recent that softening had not taken place in its distribution. In the cerebellum there were two old areas of softening, one large one in the distribution of the right posterior inferior and superior cerebellar arteries, the other on the left in the distribution of the superior cerebellar artery. These vessels were thrombosed.

Comment. The thrombosis of the basilar artery was only a few hours old and was obviously the lesion which caused the patient's death.

The other lesions were of two distinct ages as seen in the illustrations. The recent ones, a few days old, appeared as fresh hemorrhages about the calcarine area on the right and below the calcarine fissure on the left. These were the cause of the blindness. The focal cause of the disorientation in time cannot be stated definitely.

The disorientation in space and the inability to revisualize former images can safely be ascribed to the superficial softening on the inferior surface of the left occipital lobe. This is in harmony with the work of other authors. This lesion was weeks' old. The lesions about the parieto-occipital sulci were of the

same age and seem definitely to be the cause of the simultanagnosia and perhaps the loss of the sense of distance.

(Case reported by Nielsen and Olsen in the *Bulletin of the Los Angeles Neurological Society,* June, 1936.)

14. *Delusion in the sphere of body scheme. Left hemiplegia and hypesthesia. Bilateral Babinski sign. Autopsy showed softening of the right thalamus.* (Los Angeles County Hospital, No. 38–220.)

J. M., an Austrian pipe-layer, 42 years old, was brought to the hospital on March 1, 1937, because he had fallen unconscious while at work that morning. Members of his family stated that he had suffered a syncopal attack once in 1935, and that for three weeks prior to admission he had complained of coldness and numbness of the feet, but otherwise had seemed well. In the past he had consumed excessive amounts of alcohol, but since the syncope of 1935 he had taken only a few glasses of wine daily.

The patient on admission was too stuporous and unco-operative to permit of an adequate examination. The positive neurologic findings consisted of a left hemiparesis and anisocoria, the right pupil being greater than the left and reacting poorly to light. Of significance in the general physical examination was the transient absence of the left radial and brachial pulse. This was apparently due to a local condition of the artery, for on return of the pulsation and throughout the course of the patient's illness the pulse was weaker and the systolic blood pressure lower on the left than on the right. The blood pressure was within normal limits.

On March 3 the patient was more alert. It could now be determined that in addition to the left flaccid hemiplegia, which was more marked in the arm, there was a slight left hypesthesia. The deep reflexes were decreased throughout, though somewhat more active in the left extremities than in the right. The Babinski and Chaddock signs were positive bilaterally. Of most interest, however, was the patient's response when his left extremities were held up in his line of vision, and he was asked what they were. He identified the arm and leg as limbs but denied that they were his. To the question, "Whose are they?" he would reply, "The doctor's" or perhaps, "Yours." Asked directly, "Aren't they yours?" he responded, "Not mine." When the right extremities were held up, he identified them immediately as his own; and when he was asked concerning the strength of his left arm and leg, he responded correctly that he could not move them (he was not aware of them).

The laboratory findings were essentially negative. The Wassermann test taken in 1928 had been negative. Study of the blood at that time showed 5,450,000 red cells and 14,800 white cells, with 70 per cent polymorphonuclears, 24 per cent lymphocytes, 2 per cent eosinophiles, and 4 per cent baso-

philes. An electrocardiogram revealed left axis deviation and evidence of myocardial impairment.

The course of the patient's illness was short. He became stuporous again on March 4, developed bronchopneumonia, and died on March 6.

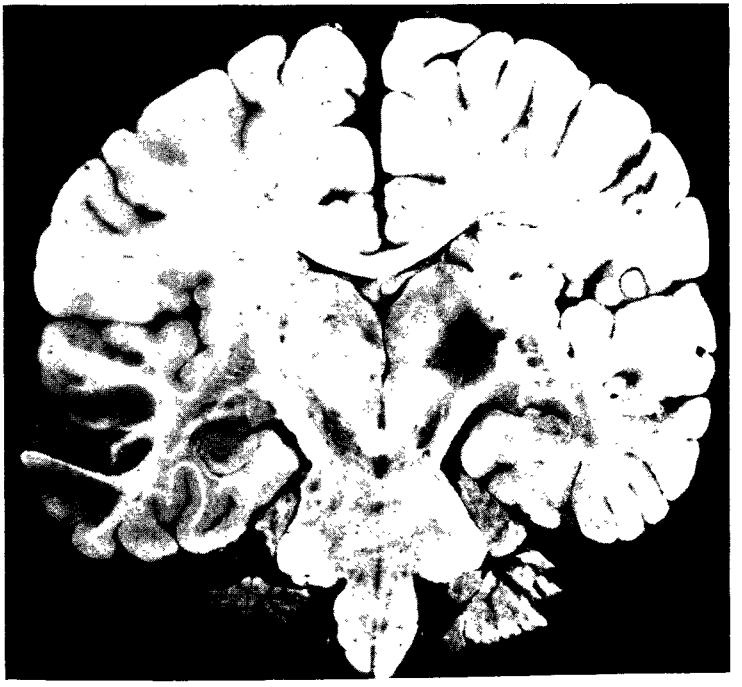

FIG. 47. Case of J. M. A section showing softening in the thalamus and extreme posterior portion of the internal capsule.

Autopsy showed the cerebral blood vessels not materially sclerosed, but the leptomeninges were somewhat thickened irregularly. There was a small subarachnoid hemorrhage over the left temporal lobe, more over the superior and middle convolutions. Below this was found a hemorrhage in the second temporal convolution, 3 cm. in length and 1.5 cm. in height. In order to show most clearly whether the thalamus was involved the brain was sectioned coronally. In the pars triangularis of the left frontal lobe in a small sulcus there appeared a circumscribed calcified nodule measuring 5 by 7 mm. in diameter. It was bordered on the mesial side by a small gelatinous bluish mass, 2 by 10 mm. A similar but smaller nodule was found in the head of the left caudate nucleus.

A section passing through the red nuclei, thalamus, and posterior portion

of the corpus striatum brought to light an embolic area of softening 8 by 13 mm. in the right retrolenticular internal capsule partly involving the thalamus. (Fig. 47). There was also softening in the semiovale just lateral to the caudate nucleus. Beside these two lesions there was another of the small calcified nodules already mentioned. This one was located in the parietal operculum and was 5 mm. in diameter. The hemorrhage described as in the temporal lobe appeared in cross section and was 1.5 cm. in each direction. In the section passing through the middle cerebellar peduncles a small portion of the softening in the internal capsule appeared as did the hemorrhage in the temporal lobe. In addition there was embolic softening of the entire superior cerebellar surface on the left due to involvement of the superior cerebellar artery. There were no further lesions.

Microscopic examination showed the small calcified nodules to be cysts with walls of connective tissue and with lymphocytes not only in the regional blood vessels but also scattered diffusely. The calcareous particles were found chiefly in the walls and in the structural content of the cysts and appeared in the form of small round globules. There was very little effect on the immediately surrounding brain substance. Dr. Cyril B. Courville was of the opinion that these were parasitic and probably cysticerci. The causes of the hemorrhage and the source of the emboli were not determined.

Comment. It was obvious that the cystic lesions were in no wise responsible for the disturbance of body scheme. Neither could the hemorrhage in the left temporal lobe nor the cerebellar embolism account for it in any way. The conclusion is drawn that the softening in the retrolenticular internal capsule was the sole cause, especially as this conclusion agrees with the site of the lesion in other reported cases.

(Case of Ives and Nielsen, 1937.)

15. *Delusion in the sphere of body scheme affecting the left arm. Left homonymous hemianopia, hemiplegia, and hypesthesia. Loss of position sense and allochiria on left. Left Babinski. Autopsy showed extensive softening of left parietal lobe and centrum.* (Los Angeles County Hospital, No. 398–645.)

P. O'B., age 66, was admitted to the hospital on Jan. 20, 1937, complaining of sudden weakness of the left extremities and of the feeling that his left arm did not seem like his own. He had been under treatment for coronary disease since November of 1934.

During the examination on admission, the patient co-operated well and was mentally alert, relating his history and volunteering the information that his left arm felt like someone else's "stuck on." Positive neurologic findings included a left homonymous hemianopia, pupils that were slightly irregular and which reacted better on convergence than to light, a left hemiplegia, with

involvement also of the face, tongue, and palate, and a left hypesthesia. Position sense was absent on the left. Painful stimulation of the left arm was referred to the left chest, but stimulation of the leg was correctly localized. The deep reflexes were decreased throughout, but slightly greater on the left, where there were positive Babinski and Chaddock signs. Physical examination revealed an enlarged heart, moderately decompensated, with auricular fibrillation. He had a hypertension, the blood pressure being 220 mm. systolic and 120 mm. diastolic pressure. His temperature was within normal limits. Urinalysis was not indicative of renal disease. The Wassermann test was negative.

Course: The patient gradually became more stuporous. On January 25, five days after admission, when asked to identify his left arm held up before him, he said that "someone is substituting this arm for my left arm" and continued to remark that, "my wife rubbed this arm, but it wasn't my arm." He developed bronchopneumonia and died January 27.

Examination of the brain showed a small amount of atherosclerosis of the basilar vessels. On the surface the entire right temporal lobe with the exception of the uncus was softened, the anterior 5 cm. being amorphous. The entire lower portion of the right frontal lobe involving the second and third frontal convolutions were markedly softened and this softening extended backward to converge at a point in the angular gyrus. There was also a small area of softening in the anterior part of the right occipital lobe. On section this proved to be lateral to the optic radiation. Further examination of the softened frontal area showed it on cross section to extend deeply to involve the lenticular nucleus but not the thalamus.

Comment. The term anosognosia was suggested in 1914 by Babinski to denote lack of recognition of hemiplegia. His patients distinctly did not lack recognition of their limbs; they believed not only that they had limbs but that the limbs were well and functionally intact. They attempted to walk with the use of the paralyzed extremities and fell in their efforts. In 1925 Barkman gathered all cases on record with autopsy findings and found that they all showed lesions affecting the right thalamus.

In the reported cases of amnesia for the limbs and delusion of absence of the limbs the lesions have been above the thalamus, in some cases superficial. The cases here reported do not exactly substantiate this conception as the thalamus was actually affected to some extent by the lesion in the preceding case (of J. M.). He could not have anosognosia because he could not be expected to recognize paralysis in limbs which he denied. However, the cases are striking in showing how specific delusions may be caused by local cerebral lesions.

16. *History of syphilis but negative serology. Apoplectiform seizure followed by right homonymous hemianopsia, disorientation in space, transient loss of recognition of objects. No paralysis. Gradual improvement.* (Los Angeles County Hospital, No. 474–886.)

M. B., aged 52 years, was admitted to the hospital Jan. 2, 1936 complaining of disorientation. He gave a history of penile lesion thirty-nine years before, for which he had been treated with intravenous injections periodically for some years. There was no other relevant history.

He stated that he had been employed as a salesman of women's ready-to-wear clothing. On that day, while on an outing in his automobile a distance of 70 or 80 miles from the city, he was suddenly taken with a severe headache, dizziness, and a feeling of weakness. For a moment everything went black before him; then his vision slowly cleared on the left side so that he could see, but he had still not regained ability to see on the right side. After a rest he drove his car home, but because he lost his way on the familiar road many times, it took him five hours to return, although it had taken him only two and a half hours to drive the distance the first time. From the time he reached his home until he was seen at the hospital there had been no essential change in his vision.

Physical examination showed a well developed and nourished Jewish man clearly conscious and co-operative. He had a moderate arteriosclerosis, although his blood pressure was only 140 systolic and 90 diastolic. No source of embolus could be found. There were no other findings worthy of note in the general physical examination. The neurologic examination showed a complete right homonymous hemianopsia without changes in the fundi. The other cranial nerves were unaffected. General sensation, motor strength, and cerebellar function were all normal. The deep reflexes were normal even for laterality and no pathologic reflexes could be obtained. Abdominal and cremasteric reflexes were also normal.

The spinal fluid was under an initial pressure of 175 mm. of water, but upon withdrawal of 8 cm. it fell to 100 mm. Jugular compression gave prompt rise, and release gave normal fall. The fluid was clear, showed 4 cells per cmm., and the serology on this and on the blood was negative for syphilis.

Doctors Richard Carter and Elinor Ives made a diagnosis of thrombosis of the left posterior cerebral artery.

With this diagnosis one should expect the Charcot-Wilbrand syndrome or at least disorientation in space. When examined for this, the patient was found to be entirely disoriented for direction and relationship of geographical landmarks in the community, even about his own home. He had forgotten whether the street upon which he lived was directed north and south or east and west.

He was unable to give directions for going to a place a few blocks from his own house. He did not even know how to get from his own abode to a well-known mountain pass a few blocks away, although he had driven along this road often. In describing his own home he did not know whether steps led up to it or not, did not know whether the front door was near the middle or on one side, did not know which way to turn after reaching the top of the stairs, etc.

Further, he was unable to name colors shown him, although he was able to select nearly any color asked of him. He therefore had a complete optic aphasia for colors but only a little actual agnosia for them. During his stay in the hospital he was able to recognize all objects shown him, even pointing out from the window an automobile racecourse where he had attended races. However, he was unable to tell from memory whether the boulevard upon which the racecourse faced was paved or unpaved.

He was able to read and to write and he drew a picture of a bird on request.

At the time of presentation the patient volunteered to give a more detailed history than that given previously. His history was as follows:

"I had gone to Murietta Hot Springs for the day, and early in the afternoon, while going through a door, I felt something like a shot in my head and everything went black. Then after just a couple of seconds things began to clear on the left side; so I could see again there, but still I couldn't see on the other side, just like now. I told my friend I wanted to go for a little walk because I didn't want him to know what had happened; so we went and got a drink. There we met some people who talked to me and called me by name, but I didn't know any of them. They said I knew them well. They came to us from the right side; so I had to keep turning around to see them, and I saw them one at a time, but I didn't know any of them.

"I decided to drive back home; so my friend and I went to get my car. I had a lot of trouble finding it, because I could not tell which one was mine. Instead of driving home I drove back to another place, and there I passed out and slept for about three hours. After this I started home and drove all the way, because I could see all right straight ahead. But instead of it taking me two and a half hours, it took me five hours, because I struck a lot of bumps on the road, and I lost my way many times. I was very familiar with the road but still I kept on getting lost. After reaching home I went to bed, and the next morning I had a terrible headache."

When he was asked how he distinguished the red from the green lights, he said, "Well, when you are used to driving, the car about drives itself. I guess I must have read the letters or watched the other cars." When tested for ability to calculate, it was found that he could do ordinary arithmetic.

Subsequent developments: In about two weeks his fields of vision began to

widen out to the right, and the patient had moments when he could revisualize certain roads in his own neighborhood. Three weeks after the onset of the illness the patient's visual fields (to a large test object) had returned completely. However, he had scotomata, and there was still considerable defect to a small test object.

On Jan. 21, 1936, the patient was again examined. There were still defects in his fields of vision, but the most marked defect was the persistence of his disorientation in space. He still believed that one of the most important streets in the neighborhood (Vine Street) ran in an easterly-westerly direction instead of north and south. He was still unable to state how to get from Vine Street to Western Avenue, a distance of about a mile in a straight line. The parallelism of these streets did not seem correct to him. He was unable to tell the direction in which a well-known planetarium was situated relative to a certain street corner, although it was almost directly east. He was similarly disoriented for directions and spatial relations of prominent places in the heart of the city. He explained that whenever he tried to remember these things, there was "something missing."

Comment. This case remained without autopsy, yet it is an excellent clinical illustration of the Charcot-Wilbrand syndrome. The loss of ability to recognize objects (his friends and his automobile) was of short duration, but the disorientation in space remained for at least two months, at which time he disappeared from observation. The defect remained after the fields of vision had grossly cleared.

17. *Complete visual agnosia of the subcortical type. Autopsy.*

Smith Shallen, aged 61, came to the hospital after a "dizzy spell" and a fall in which he did not lose consciousness and after which he did not see well to the left.

Physical examination showed that he was blind in three quadrants. The only area in which he had light perception was the upper right quadrant. He had a marked arteriosclerosis.

Introductory. The patient talked well, responding to all questions and answering intelligently. He gave a good history. The aphasic defect seemed limited to visual agnosia.

Visual agnosia. In the remaining quadrant the patient failed to recognize anything, not even his brother. He said he saw shadows moving before him. When he was shown an ophthalmoscope light, he said he saw the light. When shown two separate ophthalmoscope lights (both well concentrated), he said they were brighter than they were before, but he still saw only one. This "one" he could not localize in space. On attempting to touch it he groped near it. When he was handed a lighted flashlight, he could not tell where the hand

ended and the light began. He was unable to name colors but distinguished differences.

When asked to describe scenes he had known, he did so very well. He also described how letters look but could not read anything. He was not asked to read by touch, as he was quite ill and tired.

It was concluded that he had a complete visual agnosia.

Acoustic agnosia. The patient named a watch by sound and recognized voices.

It was concluded that he had no acoustic agnosia.

(He had no astereognosis.)

Writing. He wrote spontaneously and on dictation without looking. When asked the address of a certain hotel located at First Street and Pine Avenue, he wrote, "1 and Pine Ave." He also wrote figures on request.

It was concluded that he had no agraphia.

We were never able to complete our examination, as the patient died the same night from a pulmonary embolus. Autopsy showed bilateral lesions of the occipital lobes due to occlusion of portions of the posterior cerebral arteries. The angular gyrus was spared. The lesion extending between the posterior horn of the lateral ventricle and the cortex of the angular gyrus separated the left calcarine area from the cortex of the region of the angular gyrus. This is in harmony with the pathologic findings in the 9 cases gathered by Henschen from the literature.

(Case of Nielsen and Von Hagen.)

18. *Loss of recognition of objects and disorientation in space. Right-handed person. No aphasia. Thrombosis of right posterior cerebral artery.*

Elizabeth A., a white woman of 72 years, was admitted to a psychopathic ward because of "loss of visual function" and inability to take care of herself. Her husband had sworn out a complaint because he thought her trouble must be mental.

Physical examination by the intern showed a severe hypertension; blood pressure was 230 systolic, 130 diastolic (mm. of mercury). The heart was enlarged downward and outward and there was a systolic murmur at the apex. She developed pneumonia almost immediately and died in seven days. Autopsy showed malignant nephrosclerosis, cardiac hypertrophy, and bronchopneumonia.

The brain (Fig. 48) showed old small vascular cysts in the left putamen and head of the caudate nucleus and a smaller one on the right side of the putamen. However, the essential lesion was a severe shrinkage of the right occipital lobe most severe on the inferior surface, and this lesion was several months old.

The legal warrant was then studied and it was found that the complaint was disorientation in space and inability to recognize objects by vision alone. As this corresponded to the symptoms expected in case of destruction of the

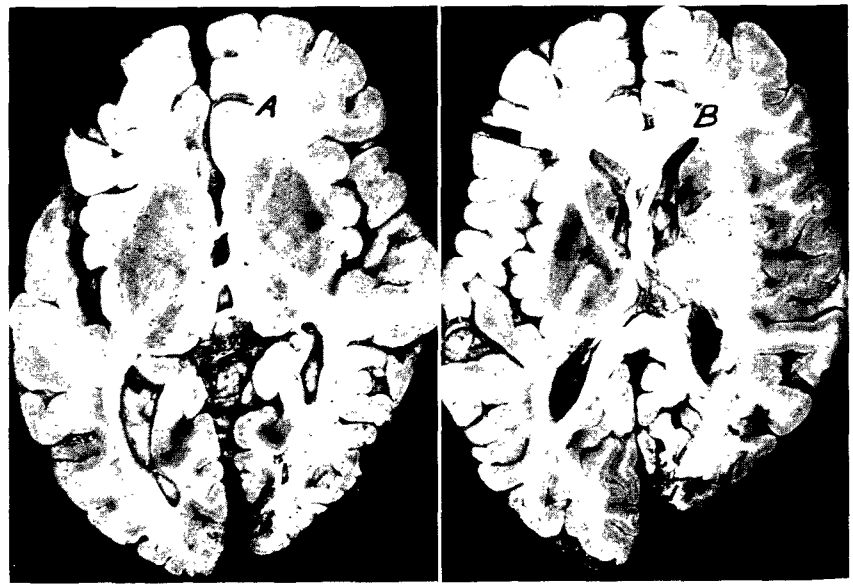

FIG. 48. Case of Elizabeth A. *A* is 1 cm. lower than *B*. The two horizontal sections show complete destruction of the right occipital lobe. The small cystic lesions in the caudate nucleus and putamen seen in *B* are interesting in view of the total lack of aphasia.

major occipital lobe the husband and the sister-in-law of the patient were interviewed concerning details of the history. The following is a record of the facts obtained:

The patient had always been right-handed and there was no record of left-handedness in the family. She had been essentially well up to the age of 62 when she had two "light strokes" which left her with paralysis of both arms so that the husband had to feed her and take care of her in every way for several months. During this time there was no aphasia, and after recovery from the paralysis of the arms the patient was about as well as before.

Her good health continued until the age of 72 when she had "some sort of stroke" which affected her vision. This occurred about three to four months before her death. It did not render her blind "because she could see what went on about her" but her behavior gave the husband the impression that she was mentally disturbed because she appeared not to recognize what she saw and could not find her way about the house. On a number of occasions after a conference with his sister about the matter he tried the expedient of

not going to her aid whenever she called but insisting that she get up and wait on herself.

However, such methods did not work because the patient became lost, would bump into things and fall, and she would then lie on the floor or on the ground until help was given to her. She complained of not being able to find her way back to bed or to the bathroom. The husband finally used the system of pulling her bed to the bathroom door whenever she needed to go. He then helped her just a few feet to the toilet and back to the bed.

The patient could not feed herself during the last four months of life. The husband stated that she did not recognize the difference between food on the plate and her glass of milk nor could she tell what to take with her fork or spoon. He knew she was *not* blind because she avoided knocking things over and reached correctly with her hands; she merely acted as though she did not recognize what she saw.

When visitors came she was more lively and entered into conversation, but her disability in finding her way about the house continued and friends became convinced that she was peculiar mentally.

At no time had she had any difficulty with speech or comprehension of spoken language but she was not able to write, except her name, after the "stroke" which affected her vision. Neither could she read except occasional words and she found newspapers and magazines uninteresting.

Comment. Here then is a case of a right-handed woman who, as a result of destruction of the right occipital lobe, developed visual agnosia for objects and disorientation in space. The bizarre symptoms led her family to assume a mental disturbance and have her committed as insane.

(Case reported by Nielsen in the *Bulletin of the Los Angeles Neurological Society* 3:135.)

19. *Ideational apraxia, ideokinetic apraxia, apraxia of the mouth, apractic agraphia, semantic aphasia, paraphasia.*

Charles Somerv., aged 56, a business man, had been well in the past. The first signs of trouble appeared one month previously when he began to have difficulty in performing his usual bookkeeping arithmetic. This trouble increased until his wife was notified that he was too uncertain in his work for him to be of any use at his place of business. He became constantly more "forgetful" (ideational apraxia?) and did such queer things that his fellow workmen thought he was "not right mentally."

Physical examination did not give localizing signs. There was bilateral Babinski sign and a very slight weakness of the members on the right side of the body. There was no evidence of choking of the disks.

Introductory. "How do you do?" The patient did not answer. His wife said

he had trouble talking, to which the patient replied, "Yes, we have trouble talking." When asked, "Can you think of the words all right, or do you have trouble saying them?", he said, "We have trouble *staying* them more than anything else."

Visual agnosia. He clearly identified objects about him. When he was asked to tell the examiners the use of objects he could not do so, but he used them correctly for the most part. He struck a match correctly and blew it out. When asked to read the sentence, "I am getting better," he read correctly "getting better." At the same time he remarked, "I suppose if I was getting over next to it, I could probably get it next." He probably meant, "If I were closer to it, I could probably see it better." There was a distinct paraphasia. He read correctly other test words.

Acoustic verbal agnosia. His constant response to spoken questions showed that he did not have acoustic verbal agnosia. When asked, "How long have you been sick?", he said, "Oh, a couple of weeks." On the other hand, he had a distinct semantic aphasia as shown by the care with which the examiners had to put their questions and the repetition which was necessary.

Apraxia. He had a great deal of difficulty showing his teeth and protruding his tongue on request. When he was shown several times, he still had considerable trouble. He could not feed himself or even bite an apple when he wished. On the other hand, he did other and more complicated acts better. He lighted a match, blew it out, cut out a square of paper with the scissors, and did other things fairly well.

When he set about to do anything of his own accord, a marked ideational apraxia manifested itself. He was allowed to dress himself, and he did it as follows: With marked slowness and in an absent-minded way he attempted to put on his trousers. He inadvertently got one leg caught over the suspenders. He did not discover this for some time and could not understand why he could not get his foot into the leg. When he finally discovered it, he correctly arranged the garment. He put his trousers on while standing up, but did not place his suspenders over his shoulders. His trousers fell down around his feet; so he pulled them up but still did not fasten them. They fell down many times, and each time he merely pulled them up. As he attempted to put on his socks he remained standing. He took one sock and his necktie in one hand and proceeded to put on the sock without raising his foot off the floor. With his trousers around his feet he had considerable difficulty. However, he finally did get his socks on. He had the same sort of trouble with all garments.

When the examiner was seated before him and asked him to imitate all acts performed, he did in a general way everything correctly but clumsily with

the left hand and everything incorrectly with the right. The same applied to the lower extremities. When asked to place the right foot on the seat of a chair, he failed to raise it high enough. He did well with the left foot.

We note here three types of difficulty in the sphere of apraxia. In the first group of tests the patient showed apraxia of the mouth and this is of the ideokinetic type. In the second group of tests he showed ideational apraxia. In the third group he showed ideokinetic apraxia.

Aphasia on the higher level (*semantic aphasia*). He had a serious semantic defect. He grasped ideas very slowly and only those of the simplest sort. It was necessary to repeat questions three or four times very directly and forcibly before he succeeded in grasping their significance. (He obviously was using the minor area of Wernicke.)

"What is your name?"	"What is . . ."
"What is your name?"	(No answer.)
"What is your name?"	"Why after a . . ."
"What is your name?"	"I guess that . . ."
"What is your name?"	"Will they, will they, will . . ."
"Are you sick?"	"No." (Shakes his head.)
"Got a headache?"	"No." (Shakes his head.)
"Hold up your hand."	Does it.
"Put your hand on your knee."	He puts it down and says, "You'll have to put this . . ." Finally after some repetition on the examiner's part, he said, "I haven't got any."

He was unable to tell the time. When we suggested it was 2 o'clock, he agreed. When we suggested it was 3 o'clock, he again agreed. But he would not agree that it was 4 o'clock.

Writing. He was absolutely unable to write. We learned from his wife that this was the first difficulty discovered when the illness began. His writing with either hand did not even show semblance to lines written for him. He was just as completely unable to copy or to draw. He did not even get the direction of the lines right. Neither could he write from dictation. When we tried to determine whether he could revisualize letters, we met with so much paraphasia that we could not determine the point.

Autopsy showed multiple tumors of the brain metastatic from the lung.

(Case of Nielsen.)

20. *Ideokinetic apraxia.*

Nick Macry, aged 30 years, a white laborer. Onset over a period of eight months with weakness of the right foot, "unwillingness" to talk, loss of visual acuity, then petit mal and grand mal attacks.

Introductory. "How do you do?" No answer. The patient appeared to be in a stupor, but this did not seem to be verified by his performance when aroused. He then talked in an angry tone of voice, but only in monosyllables. He was not paraphasic but answered appropriately.

Visual verbal agnosia. The patient recognized objects about him, and tried to dress and undress himself. It was, however, impossible to get him to pay any attention to written matter. His relatives stated that they never saw him read.

It was concluded that the patient probably had visual verbal agnosia, but the point could not be established.

Acoustic verbal agnosia. When aroused the patient talked and answered properly. The examiner asked him, "Do you have headache?" He did not answer, but when the relatives said "Yes," he turned and said "No."

It was concluded that he did not have acoustic verbal agnosia.

Apraxia. The patient could not undress himself. He tried to unbutton his clothes but never succeeded in getting anything done. He acted like a child who had not learned how to do it. When he lay on the examining table, he turned and rolled like an infant and would have fallen off if not carefully watched. He was involuntary to urine and feces. When he attempted to dress himself, he put his arms into the legs of the underwear and could not proceed. When he was asked to sit down, he walked across the room as though uncertain whether he was acting properly. When he finally reached the chair, he walked up to it and did not know how to turn around but bent his knees while facing it. When this was not successful, he turned halfway around, decided that was wrong, turned all the way around to the proper attitude, bent his knees a little but again decided that was wrong, and never succeeded in seating himself.

The patient had choked optic disks and other neurologic signs of tumor of the brain. He was referred to the neurologic surgeon but the case was inoperable. At autopsy there was found a large diffuse subcortical glioma coming to within 1 cm. of the surface, nearly filling the semiovale on the left side. It reached from the anterior pole of the lateral ventricle nearly to the occipital lobe.

20. *A remarkable case of apraxia of the entire body except the left lower limb.*

Sarah Turney came to the hospital able to say only, "You, you, you" to everything.

Examination from the standpoint of agnosia was begun. The patient paid no attention to anything said to her, except to say the eternal "You, you, you." She paid no attention to anything written. She carried out nothing asked of her, either spoken or written.

It was concluded that she had a complete acoustic verbal agnosia, but we shall see how wrong this conclusion was.

Having decided that she understood nothing that was said and anticipating no co-operation, we proceeded to recheck the physical examination made by the intern. When the left lower limb was raised to test the achilles tendon reflex, the patient did not put the limb down. As a joke we said, "Put it down." She immediately did so. When we asked her to raise it, she did so. This was repeated several times. To make certain that she really understood, we said, while the limb was still raised, "Leave it up now." She did so. We then asked her to do things with the other members, but without the least result. When we showed her what to do, she was able to perform with the left lower limb *only;* all the other three members were apractic. She seemed by her expressions to understand in general all that was said to her and in her presence.

21. *Isolated agraphia.*

Winfield Lo., a white man of 51 years, came to the hospital with his first cerebral vascular accident, weakness of the lower right face, and flaccid paralysis of the right upper extremity.

He had full comprehension of spoken language. He answered correctly and immediately such questions as "What is the capital of Ohio?", "How do you spell Sacramento, New York, Constantinople?", "Where do you work?" However, he had considerable dysarthria, and one had to listen carefully to understand him. Calculation was correct. He named correctly objects shown to him and was able to read aloud handwriting and print without trouble.

The essential disability was confined to writing. All efforts were of necessity made with the left hand. When handed a pencil and asked to write whatever he chose, he made a series of lines like a septic fever curve. When asked what he intended to write, he replied, "Insufficiency." But he could not write it. He was then asked to write New York. He made a few unintelligible marks resembling letters. He was dissatisfied with his performance but unable to correct it. He next announced he would write the word inflation. What he wrote was illegible.

Copying was also impaired. He copied very poorly the words New York and still less well Philadelphia. He attempted to copy each letter exactly as it was presented to him, either in print or handwriting. He copied figures other than writing much better, such as a conventional birdhouse and geometrical figures.

Nine days later he was presented with blocks for building words, each block containing one embossed letter. He announced that he was going to build up the word INFLATION but he made "NLATION." He was unable to build up LOS ANGELES until he was given just the correct letters for it (Fig. 49). He

then succeeded. In the same way he built for CALIFORNIA, "CILORFINA." As he was able to copy figures which were not symbols, the defect was not due to loss of sense of direction.

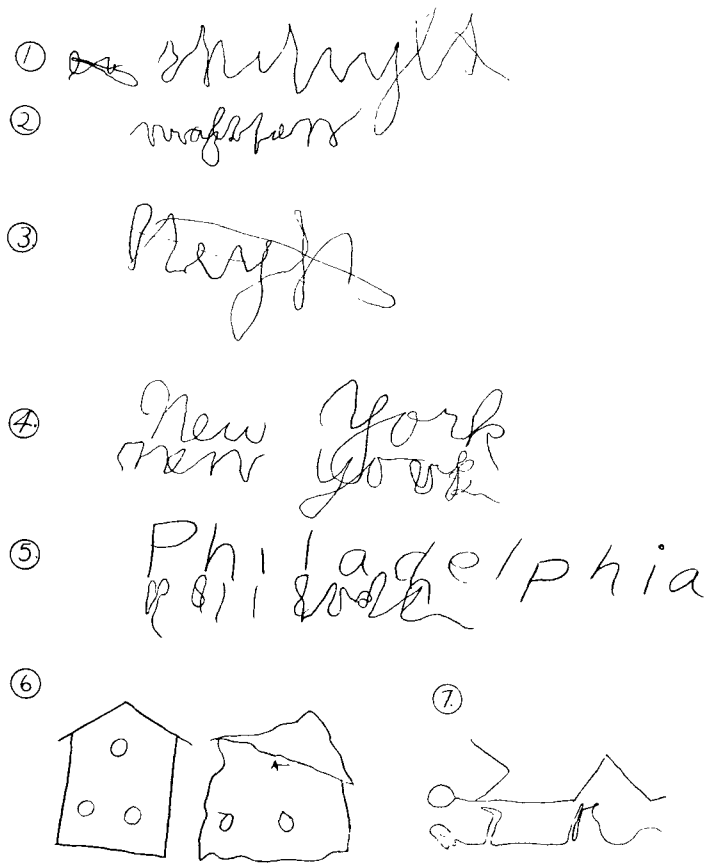

FIG. 49. Case of Winfield Lo. (apractic agraphia). 1. Written after patient announced he was going to write *insufficiency*. 2. Written after patient announced he was going to write *inflation*. 3. Written when he was asked to write *New York*. 4. Copy of *New York*. 5. Copy of *Philadelphia*. 6. Copy of the conventional birdhouse. 7. Copy of "reclining figure."

It is important here to note that the patient was able to spell any word asked of him. In other words, he could "visualize" words before attempting to write them. Yet whether he visualized them or saw them on paper before him, he was largely unable to make the letters. This is apractic agraphia.

We had further evidence of this fact from his attempt at building words

with blocks. This capacity was defective but not completely lost. See the illustration, Fig. 49.

22. *Marked disturbance of attention. Perseveration. Loss of spontaneous speech. Apractic disturbances of ideokinetic type. Death. Autopsy showed hemorrhage destroying left thalamus.* (Los Angeles County Hospital, No. 505–335.)

Helen M., aged 43 years, was admitted July 6, 1936, unconscious, in which condition she had been found in bed that morning. Her family had known for four years that she had a mild diabetes and hypertension.

Examination showed the patient still unconscious. The heart was greatly enlarged and blood pressure was 250 systolic and 140 diastolic. The spinal fluid was so bloody that its hemoglobin content was 20 per cent. There was sugar in the urine, and the blood sugar was 370 mg. per 100 cc. A few hours after this work had been done the patient regained consciousness and a neurologic examination became more satisfactory. Meningeal signs were prominent (bloody spinal fluid) and there was bilateral weakness of the abducens nerves. There was paralysis of upward gaze and a right hemiparesis.

On the following day the patient was clearly aware of her surroundings but was dull mentally and responses were slow. She named objects incorrectly, calling a dry electric cell "downpour" and a flashlight "watertight." However, she named a match correctly and when shown a match box she read correctly the words "Pacific Match Company" on it. Further examination of her speech faculties showed that her trouble was chiefly due to perseveration and paraphasia. When asked to name a watch held to her ear she said first, "Four seven," then "It's a downpour of rain." She used the reply "Four seven" on many occasions, but by repeated correct replies to some questions it became clear that she recognized objects shown her. She named correctly a bunch of keys, a bottle of water, a silver half dollar, and a ten dollar bill. When shown a comb she said, "I can count my hair with it."

On the third day in the hospital she clearly understood what was said to her but to an incomplete degree and very slowly. Sometimes she did not reply at all to questions. The pupils were sluggish in their reaction to light and paralysis of upward gaze continued, though downward gaze was good. The weakness of the right limbs was still present. Diagnosis was made of a lesion in the left thalamus.

On the fourth day the clinical picture was well defined. The principle disturbance was one of attention. She could not be made to protrude her tongue on request, demonstration, or urging. When stimulated she turned about, patted her hair, rubbed her nose, etc. When she was given a lighted match she readily blew it out. When the box of matches was handed to her

she took two matches in her fingers and had some difficulty in releasing one. Having obtained one she closed the box, then reopened it. She turned it correctly in her fingers but held it up in mid-air. When the box was approximated to it she tried to light the match without contact with the box. After some time she did reach the box but did not strike with force sufficient to light. When it was lighted in her fingers she held it aloft and did not seem to know what to do. When told to blow it out she did so without trouble; she then grinned at the examiner. Throughout the examination she never spoke, but as the examiner left and said, "Good-bye," she replied "Good-bye."

When tested for reading it was found that she could read but that she was totally disinterested. She read silly statements without the slightest emotional response and made no effort to carry out written commands. She answered all questions when possible with nods or shakes of the head if she answered at all.

The patient died of bronchopneumonia. Autopsy showed a hemorrhage into the left thalamus which had ruptured into the third ventricle. It extended into the upper portion of the mesencephalon as would be expected from the paralysis of upward gaze.

Comment. This case is highly instructive in showing the relation of the thalamus to the various parts of the cortex. The whole language area and the cortex concerned with ideation for movement (ideational plan) is dependent upon its association with the thalamus in order to perform its functions. It is noted that as only one thalamus was out of function with persistence the examiner could obtain the patient's attention.

(Case reported by Nielsen in the *Bulletin of the Los Angeles Neurological Society* 6:1.)

23. *A left-handed man presented delusion of body scheme, homonymous hemianopia, achromatopsia, and confusion of laterality from a left-sided lesion. There was no aphasia but partial crossed temporo-occipital dominance.* (Los Angeles County Hospital, No. 738–276.)

Donald O'H., a white man of 69 years, was admitted Oct. 31, 1940, following a sudden loss of consciousness which occurred as he attempted to rise from his bed in the morning. The only history obtainable from friends was to the effect that he had been well except for a known hypertension.

On admission some hours later the patient was found to be clearly conscious but completely paralyzed on the right side. As he comprehended language and spoke well he was asked about handedness. He explained that he had always been left-handed.

Examination revealed a blood pressure of 180 systolic, 120 diastolic, and a pulse rate of 88 per minute. His heart was considerably enlarged to the left and its rate was very irregular. The lungs were clear, the abdomen large and obese.

Neurologic examination showed the optic disks pale, the vessels full and bordered by silvery streaks. There was a complete right homonymous hemianopia. The pupils were 3 mm. but irregular in contour and fixed reflexly. There was a tendency to conjugate deviation to the left but this disappeared when the head was passively or reflexly turned. There was a partial ptosis of the left eyelid and a moderate exophthalmos on the left. The lower right face was weak as a part of the right hemiplegia and the tongue was protruded to the right. The deep reflexes were absent on the right, active on the left. All the usual pathologic pyramidal reflexes were present on the right; the Babinski sign was also present on the left. Sensation was diminished on the left side of the body and face.

On the second day a good study of the mental state was possible. There was no aphasia, the patient being in complete possession of comprehension of spoken and written language. He could even write with the left hand. On the other hand he had the delusion that his right hand did not belong to him but to the examiner (a symptom which could be expected on the basis of left-handedness) and yet he had hemiachromatopsia as though he were right-handed. While he failed to recognize colors he recognized all objects shown to him.

As for the elements of Gerstmann's syndrome he had no finger agnosia but was confused as to laterality. He did not know right from left with reference to his own ears and eyes and to the hands and fingers of the examiner. Calculation was not tested. As he could write, he did not have constructive apraxia. There were no other forms of apraxia.

The spinal fluid was clear and colorless, under a pressure of 180 mm., and the cell count and globulin content were normal. Serology was negative for syphilis and the urine was normal.

On the third day the patient suddenly went into coma and died in a few hours. A diagnosis was made of thrombosis of the left posterior cerebral artery with some involvement of the middle cerebral.

The brain was examined after hardening in formaldehyde solution. The left temporal, posterior inferior parietal, and occipital lobes were completely disintegrated. They were softened from thrombosis but in addition there was a massive hemorrhage from the proximal portion of the posterior cerebral artery at the base so that the inferior surface of the left side was filled with clots of blood. It was necessary to cut away the inferior portion by a horizontal incision in order to obtain a section for a photograph which would show relations.

Comment. This case presents evidence of crossed occipitotemporal dominance by virtue of confusion of laterality and achromatopsia from a lesion

of the left occipital lobe in a left-handed man. He was right-brained as shown by the absence of aphasia, even absence of finger aphasia; yet his left occipital lobe was dominant for recognition of colors and laterality. It is striking indeed that the right occipital lobe was unable to assume certain functions after destruction of the left.

(Case of Friedman and Nielsen, 1941.)

24. *Right hemiparesis and hemihypesthesia, semantic alexia, and simultanagnosia. Recognition of objects, colors, still pictures, and symbols preserved. Autopsy: thrombosis of left posterior cerebral artery and softening of the left thalamus.* (Los Angeles County Hospital, No. 24–929.)

William C., a white man aged 68 years, was admitted to the neurological service Sept. 15, 1940, without any available history. As he was unable to give an account of himself and as he died without our establishing contact with relatives, no history ever became available. His attention could be obtained for answers to questions but his memory of his illness was impaired.

General examination showed a well advanced state of arteriosclerosis. The blood pressure was 190 systolic and 100 diastolic, the pulse rate 60 per minute. The heart was somewhat enlarged to the left, the lungs were clear.

Neurologic examination showed the pupils to be 3 mm. in diameter and fixed to light as well as on convergence. The optic disks could not be seen. The fields of vision to gross examination showed a right homonymous hemianopia. The eyeballs moved well in all directions without nystagmus. There was nothing worthy of note concerning the remaining cranial nerves.

The patient had a right hemiparesis affecting limbs and lower face and also a right hemihypesthesia. The left limbs were moved voluntarily and intelligently without apraxia. There was a considerable degree of dysarthria but his speech was entirely comprehensible.

There was some degree of aphasia. While he could "read off" letters, figures. and words, he had no comprehension of their significance. This constitutes semantic alexia, or, according to old terminology the visual components of transcortical sensory aphasia. He was oriented in space, recognized and named colors correctly, recognized and named correctly the laterality of limbs and fingers of himself and of the examiner, and was able to draw geometrical figures as requested. This demonstrates that he had neither auditory agnosia, motor aphasia, nor finger agnosia.

Simultanagnosia was shown by his failure to recognize action in pictures which showed action while he was able to recognize the elements of the pictures. He named men and animals and inanimate objects (there was no visual agnosia for objects) but failed to grasp the implication of action as shown by postures of the persons represented in the pictures.

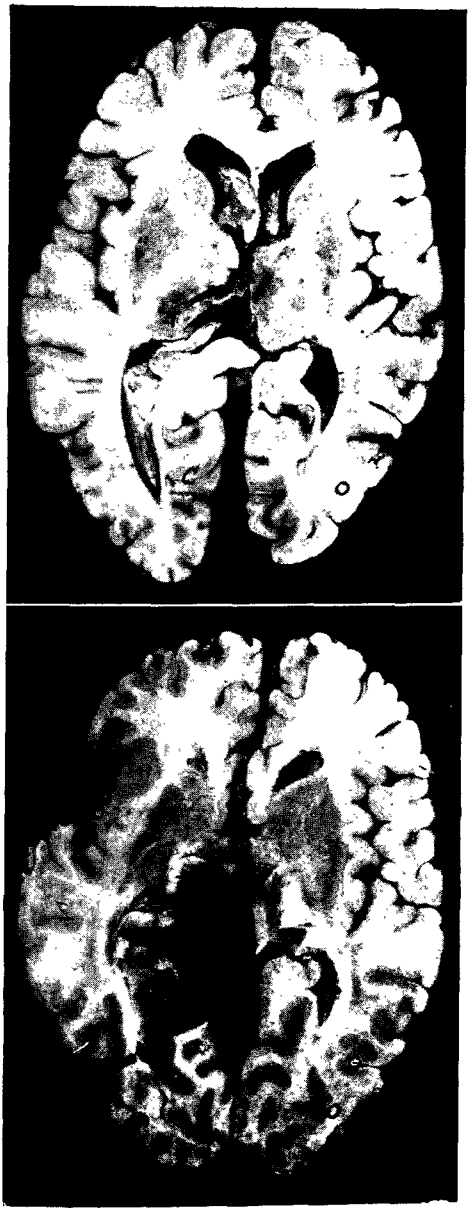

FIG. 50. Case of William C. The upper figure is cut at a higher level than the lower. It shows destruction of the left thalamus (*T*) by softening and also involvement of the left calcarine area (*C*). The lateral convex portion of the right occipital lobe (area 19) is involved (at *O*). In the lower figure the entire inferior surface of the temporo occipital region is softened. The brain stem is absent because it was destroyed by hemorrhage.

The diagnosis of the cerebral vascular accident rested between occlusion of the angular gyrus branch of the left middle cerebral artery and occlusion of the posterior cerebral artery. The latter was decided upon on the basis of the simultanagnosia and absence of the syndrome of Gerstmann.

The patient developed pneumonia and died in two days. The brain was examined after two weeks' hardening in formaldehyde.

Marked arteriosclerosis and an aneurysmal dilatation of the circle of Willis were found. The left posterior cerebral artery was occluded and the left cerebral peduncle almost entirely destroyed. Horizontal sections showed softening of the left cuneus and the entire lingual gyrus. The inferior surface was so disintegrated that it could not be held together after horizontal sectioning (see Fig. 50). The inferior portions of the left areas 18 and 19 of Brodmann were destroyed but the convex portion of both was preserved. The cortex of area 19 was destroyed on the right side. The left thalamus showed advanced softening in its posterior portion. The left superior cerebellar artery was also thrombosed.

(Case of Friedman and Nielsen, 1941.)

25. *History of cerebral anoxemia with stupor. Later the sudden development of delirium followed by hemiplegia and hemihypesthesia. Loss of sense of position and of localization in the left upper extremity. Persistent denial of the paralysis. Delusions referable to body scheme. Gradual improvement of anosognosia.* (Los Angeles County Hospital, No. 505–073.)

V. L. F., a Negress 48 years of age, was admitted to the hospital Nov. 3, 1936. According to members of her family she had complained of lassitude and fatigue for about three days. The morning before admission she had complained also of blurred vision and at about 4 P.M. she had developed a headache which persisted four or five hours. At the end of this time she suddenly became delirious. Restlessness was marked and her movements were said to be in-co-ordinate because she had spilled water in the bed when attempting to drink. She became quiet in a few hours and it was then noted that her left side was paralyzed.

The patient had previously been admitted to the hospital (Oct. 13, 1936) because of uterine fibromata. Surgery on October 14 had been rather prolonged and the patient while under ethylene anesthesia had stopped breathing and had gone into shock. She had been resuscitated with considerable difficulty and had remained somewhat stuporous.

Neurologic examination on October 15 showed her to be in an irritable, restless state of coma, unable to talk. There was no paralysis present but the upper extremities tended to assume a flexed position while the lower extremities remained extended. The deep reflexes were increased on the right

and the Babinski sign was bilaterally positive. A diagnosis of cerebral anoxemia was made by the author. The patient improved rapidly and became clear mentally two days later. She was discharged October 28. Neurologic examination at this time showed no evidence of organic disease of the nervous system. Past history was otherwise irrelevant.

On examination Nov. 3, 1936, the patient was found to be well nourished. Blood pressure was 116 systolic and 76 diastolic. The abdominal wound had not yet healed completely. There was conjugate deviation of the head and eyes to the right. The visual fields were grossly normal. The pupils were irregular and reacted poorly to light though promptly on convergence. There was a flaccid left hemiplegia. On the left side the deep reflexes were diminished, sensation was impaired, and there was a positive Babinski sign. Passive movement of the left upper extremity caused the patient to complain of pain in the left shoulder. When the sole of the left foot was stroked she complained of pain in the knee and on the left side of the body. Painful stimuli were correctly localized to the left knee and ankle but the degree of pain suffered seemed more marked than the stimulus would normally warrant. Perception of position and localization was absent in the left upper extremity.

The patient was conscious and moderately co-operative. She was oriented for person, place, and day of the week but not for date. She did not remember the details of her admission to the hospital. When asked about previous hospitalization she stated that she had been in the old hospital in 1934 when a 15-pound tumor was removed. (There was no record of this.) She raised her right hand regardless of which hand she was asked to show. She did, however, differentiate the right from the left side correctly. The patient had no knowledge of her paralysis. When questioned for the reason of admission to the hospital she stated that her tongue was thick but that her sister thought she had had a stroke. The patient did not agree, saying, "I could walk to New York." To the question, "There's nothing the matter with you now, is there?" she replied, "No, I want to walk." The patient stated that she believed she could move the fingers of her left hand if she tried, and then asked the examiner if he could "see them wiggling." When requested to move her left leg she suggested that the doctor step away from the foot of the bed as he might be kicked. Again being told to raise the left hand she said, "I never seem like I can raise it." After her hand was raised and dropped several times it was suggested that she must be paralyzed. She said, "It acts like it." It was suggested that maybe she had had a stroke, to which she replied, "Maybe is right." Later she said, "Since you say you think I'm paralyzed I believe I am," but on being asked, after a few moments, "Do you believe you are paralyzed?" she replied, "No."

For several days after admission the patient expressed delusions concerning the left side of her body. She usually identified her left hand held up before her as her own but occasionally stated that it belonged to her brother-in-law. When she touched her left elbow she called it someone else's knee. Speaking of her left extremities she said, "That's an old man. Stays in bed all the time." When asked if she minded, she replied, "Yes, I don't want any spirits in bed with me." (This phase of the case is another subject and is reported elsewhere.)

The patient improved gradually so far as the anosognosia was concerned. Examination on November 18 showed no return of function on the left side but the patient was clearly aware of her disability.

Comment. In this case there was no left homonymous hemianopia. Position sense was impaired only in the left upper extremity. The presence of anosognosia with motor and sensory disturbance on the left side of the body led to the clinical diagnosis of a thrombotic lesion involving the area of the right optic thalamus.

Barkman in his study advanced the idea that an initial disturbance of consciousness was necessary for the development of anosognosia. This had been noted in practically all cases reported in the literature except those reported by Babinski and Souques.

(Case reported by von Hagen and Ives in the *Bulletin of the Los Angeles Neurological Society.*)

26. *Onset of right hemiplegia with vertigo. Hypertension. Delusion referable to body scheme for a few hours. Right homonymous hemianopia. Impaired sensory functions on the right including loss of position sense. Allochiria and allachesthesia on the right. Denial of paralysis for three days. No improvement of hemiplegia.* (Los Angeles County Hospital, No. 524-804.)

M. B., a white woman 57 years of age, was admitted to the hospital Nov. 10, 1936. The history given was that at about 3 A.M. on the day of admission she had awakened with a feeling of nausea. She arose to go to the bathroom and in reaching for her slippers became dizzy and fell to the floor. She managed to get up and walk to the bathroom where she again fell and subsequently was assisted to bed by neighbors. The dizziness persisted for several hours but had subsided by the time she reached the hospital.

Past history was irrelevant except for attacks of dizziness at varying intervals for two years. These were associated with exertion and consisted of a feeling of falling backwards and rotation of objects to the left. Hypertension had been noted two years before.

The intern's examination showed the patient to be conscious and alert. Blood pressure was 190 systolic and 120 diastolic. There was paralysis of the right upper extremity and weakness of the lower extremity.

During the evening of November 10 the patient thought her daughter was in bed with her because there was a strange arm across her chest. (Oddly enough her right upper arm was at her side.) She denied being paralyzed.

The patient was examined by Dr. Elinor Ives November 11 and was found to be right-handed. She complained that her right arm felt peculiar and stated, "Last night I thought my arm was on the floor and that someone else's arm was across my chest. I thought someone was in bed with me but I didn't know who it was." On being asked when she decided that this was untrue, she stated that she realized it was her own hand when she pinched it. She said that her right upper arm felt "waterlogged and heavy as though it had gone to sleep." The right leg also "gets numb and goes to sleep." Asked if she could move the leg, the patient answered that it wasn't paralyzed like the arm. The leg could not be moved at all. When asked if she could walk on it, she admitted her inability, and to the question, "Does the weakness of the right leg keep you from walking," she answered in the affirmative.

There was a right homonymous hemianopia. Extraocular movements were well performed. Both extremities on the right were paralyzed and there was weakness of the right lower face. On protrusion, the tongue deviated to the right. The deep reflexes were hyperactive throughout, more on the right, and there were bilateral pathologic plantar responses. There was impaired perception of pain and touch on the right side of the body. Position sense was also lost on the right. Allochiria and allachesthesia were marked. The patient was able to calculate. When shown the written request, "Raise two fingers," she read it aloud and executed the request correctly. There was finger aphasia (inability to name her fingers).

The patient was re-examined November 13. She would not admit that she had had a stroke and was paralyzed. However, when her disability was strongly stressed she agreed that her right side was paralyzed "but not as bad as some people think." Further delusions of body scheme were not expressed.

The denial of paralysis was present for three days after which time the patient admitted being paralyzed. Her condition otherwise remained the same and she was discharged Nov. 26, 1936.

Comment. This is the first report of a case of anosognosia in right hemiplegia. The case serves to show that, while the denial of paralysis is nearly always associated with a lesion of the minor side, the rule is not inviolable. There is undoubtedly some relation between this syndrome and language.

(Case reported by Von Hagen and Ives in the *Bulletin of the Los Angeles Neurological Society.*)

27. *Previous cerebral vascular lesion. Hypertension. Impaired memory. Left hemiplegia and hemihypesthesia. Denial of paralysis of left lower extremity.* (Los Angeles County Hospital, No. 229-049.)

E. M. C., a white woman 76 years of age, was admitted to the hospital Dec. 11, 1936. She was unable to give a coherent history but correctly stated that her memory was poor. She volunteered the information that she had high blood pressure and had had a stroke but she "didn't know when."

On examination the blood pressure was found to be 199 systolic and 75 diastolic. The visual fields were normal to the usual gross tests. The pupils were equal, slightly irregular, and reacted sluggishly to light. There was a left hemiplegia, the upper extremity being spastic, the lower flaccid. On the left side the deep reflexes were increased, a positive Babinski sign was elicited, and perception of pain was diminished on the left side of the body, especially in the lower extremity. There was also impairment of touch perception on the left side. Position sense was not determined.

The patient showed a marked impairment of memory, yet she was alert and co-operative. She was disoriented for time and place. She recognized that she was paralyzed in the left upper extremity but always denied paralysis of the lower limb. When asked to move both legs she moved the right vigorously. When asked, "Are they both moving?" she replied, "I think so."

She was discharged from the hospital unimproved on December 19 and died eight days later in a sanitarium. Autopsy was not performed.

Comment. Although this patient was somewhat demented she was alert and co-operative. The mental defect alone could not account for the appearance of anosognosia as the patient was always aware that she was paralyzed in the upper extremity. It should perhaps be noted here that the sensory impairment was more marked in the extremity in which paralysis was denied. This case is also slightly different from the usual in the absence of hemianopia.

(Case reported by Von Hagen and Ives in the *Bulletin of the Los Angeles Neurological Society.*)

28. *Mental deterioration for one year. Sudden onset of left hemiplegia with left hypesthesia. Position sense intact in left upper extremity. Conjugate deviation of head and eyes to the right. Left homonymous hemianopia. Disorientation for time and place. Denial of paralysis.* (Los Angeles County Hospital, No. 493–887.)

M. H., a white woman aged 60, was admitted to the hospital Jan. 22, 1937. According to the record of the admitting physician the patient stated that she had awakened during the previous night unable to move the limbs on the left side of her body.

History revealed that the patient had been acting queerly for about a year. She had been a patient in the hospital during April of 1936. At that time she had stated that there was nothing wrong with her. She had been co-operative and well oriented. No motor weakness or loss of sensory function had been

demonstrated. The deep reflexes had been increased on the right and there had been bilateral positive Babinski signs. The blood Wassermann had been negative. A diagnosis of cerebral arteriosclerosis had been made.

On examination the following day the blood pressure was 185 systolic, 90 diastolic. There was a spastic left hemiplegia most marked in the upper extremity. Bilateral pathologic toe reflexes were elicited. Perception of touch and pain was impaired on the left. Also painful stimuli were not well localized on the left side of the body. Position sense was intact in the left upper extremity, absent in the lower. There was conjugate deviation of the head and eyes to the right and a left homonymous hemianopia.

The patient was conscious and co-operative but disoriented for time and place. Right and left sides of the body were correctly identified but paralysis was denied. Numerous attempts were made to convince the patient that she had a left hemiplegia but she persistently maintained that she was not paralyzed and could move her left arm.

The patient was re-examined January 27. She was co-operative but still disoriented for time and place. She continued to maintain that she was not paralyzed, although when it was pointed out to her that her left arm did not move when she so desired, she became somewhat uncertain. However, in a few minutes she again returned to her claim that she was not paralyzed.

Frequent examinations were made until she was discharged February 5. At all times she was consistent in her denial of paralysis although it was often pointed out that it was impossible for her to maintain the left extremities in any elevated position. The argument was always unsuccessful, and her condition upon discharge was unchanged. She died in a rest home March 7, 1937. Autopsy was not obtained.

Comment. In this case it may be pointed out that the sense of position was intact only in the left upper extremity. There was also a definite history of mental deterioration. Barré reported a case similar to this in that position sense was impaired in only one extremity. He maintained that of all sensations which play a part in this condition the sense of position is most important.

(Case reported by Von Hagen and Ives in the *Bulletin of the Los Angeles Neurological Society.*)

29. *History of a "stroke" nineteen years before. Hypertension. Left hemiplegia, and hemihypesthesia. Impaired position sense on the left. Conjugate deviation of head and eyes to the right. Left homonymous hemianopia. Disorientation for time and place. Lack of recognition of left hand but not of left foot. Denial of paralysis.* (Los Angeles County Hospital, No. 544–486.)

E. S., a Negress aged 75 years, was admitted to the hospital March 16, 1937.

There had been a gradual onset of weakness beginning in the left arm on the morning of March 15 and extending to involve the lower extremity later in the day. Some thickness of speech was noted immediately and the patient had been unable to speak since that evening.

Nothing of significance was noted in the history except that the patient had suffered a "stroke" nineteen years before from which she had completely recovered. Hypertension had been present for a number of years.

The patient was obese. Blood pressure was 230 systolic and 100 diastolic. There was conjugate deviation of the head and eyes to the right. Examination of the visual fields revealed a left homonymous hemianopia. The pupils were equal, and while slightly irregular, reacted quickly to light. There was a left flaccid hemiplegia including the lower face, and the tongue was not well protruded. The deep reflexes were increased on the left and there was no response to plantar stimulation. There was a loss of touch perception on the left side of the body. Pain sense was impaired and the patient referred painful stimuli on the left foot to the left lower abdomen and from the palm of the left hand to the medial aspect of the arm. There was impaired position sense on the left side.

The patient was somewhat stuporous and slow in her responses. The tongue was protruded and the eyes closed upon request. She was disoriented for time and place. When asked for her name the patient said, "Some call me Sexton." When her left hand was elevated and brought into her field of vision she failed to recognize it as her own. However, she identified her left leg correctly. At no time did she complain of being paralyzed, but on the contrary, stated that she could move her left extremities as well as the right.

The patient was examined several times during her first week in the hospital. The outstanding feature noted at all times was the denial of paralysis in spite of repeated attempts to lead the patient to recognize her disability.

Comment. Here we again have the development of anosognosia without any evidence of an initial loss of consciousness. Otherwise the case is typical of those reported in the literature.

(Case reported by Von Hagen and Ives in the *Bulletin of the Los Angeles Neurological Society.*)

30. *Signs of metastatic abscess to brain. Stupor, left homonymous hemianopia and conjugate deviation of head and eyes. Left hypesthesia and hemiplegia. Unawareness of hemiplegia. Autopsy showed several cerebral abscesses, one involving right thalamus and immediately adjacent structures.* (Los Angeles County Hospital, No. 375–797.)

The patient, a white woman aged 31 years, was admitted to the hospital complaining of abdominal pain which had been present for four months and

quite severe for ten days. On admission there were signs pointing to general peritonitis but this condition subsided with conservative treatment. She continued to manifest a septic fluctuation of temperature, and a diagnosis of left subphrenic abscess was made. It was considered secondary to diffuse pelvic infection. A single unsuccessful attempt to drain the subphrenic abscess was made on February 20.

On the evening of March 15 the patient became irrational and groaned a great deal during sleep which had been induced by sedatives. The following day she was stuporous but could be aroused and was then co-operative and seemed mentally clear although she made remarks to her family which they considered unusual. On March 17 she complained of right temporal and occipital headache and stiffness of the neck. There had been some vomiting.

Neurologic examination on March 17 showed the patient to be somewhat slow in her mental reactions although she was able to co-operate with the examiner. The neck was moderately stiff and painful to anterior flexion. There was conjugate deviation of the head and eyes to the right, possibly because of left homonymous hemianopia. Pupils reacted fairly well on convergence but not to light. There was a left hemiparesis. The deep reflexes were small and equal but the right archilles tendon reflex was greater than the left. There was a positive Babinski sign on the left. Sensory tests showed impaired touch and pain perception on the left side of the body with marked impairment in the lower extremity. There was loss of sense of position in the left lower limb but not in the upper. Spinal puncture gave cloudy fluid without increase in pressure. The cell count was 1000 per cmm., and of these 90 per cent were polymorphonuclears.

When questioned concerning her symptoms the patient complained of headache and of feeling ill. She did not mention the impaired function of the left extremities. When asked directly if the left arm and leg were paralyzed, she either would not answer or replied, "No." Eventually, after her inability to move either the arm or leg on that side had been demonstrated to her she believed that the left arm was "a little weaker than the right."

She became progressively more stuporous and died on March 19, 1937.

An autopsy confirmed the diagnosis of pelvic infection and secondary subphrenic abscess on the left. The brain was not studied at the time but was immersed in solution of formalin for a period of two weeks, after which the examination was completed. There was a generalized flattening of the convolutions on both sides, but the right temporal lobe appeared larger than the left, and there was a herniation of the right cingulate gyrus. A fibrinopurulent exudate filled the basilar cisterns and on coronal section of the brain it was found also in the right lateral ventricle. There were several areas of

the brain, all on the right side, which were or had been the seat of infection. These were: (1) A circumscribed abscess filled with inspissated pus in the head of the caudate nucleus; (2) an abscess in the globus pallidus and ad-

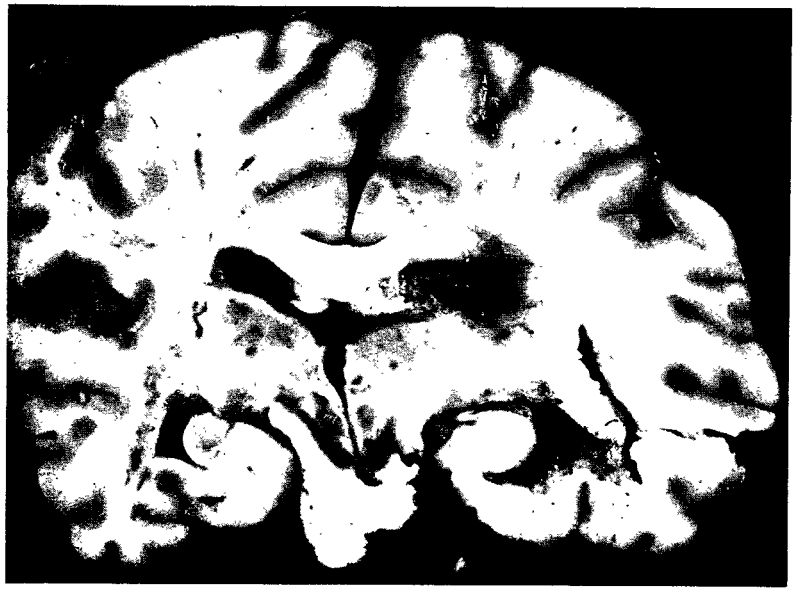

FIG. 51. Case of Von Hagen and Ives (1937). The dark lesion at the superolateral border of the right thalamus and the fragmentation of the adjacent border of the fornix is an abscess. Softening of the forceps major and of the hippocampus of the same side is visible.

joining internal capsule; (3) a large irregular area consisting of softening, disintegration, and purulent exudate involving the forceps major, the hippocampus, and the borders of the posterior horn of the lateral ventricle which contained pus; (4) an area of discoloration and softening in the crus of the fornix and the adjacent centrum and thalamus. (See Fig. 51.)

Sections were made through the thalamus extending from the softening of the centrum above to the disintegration of the forceps major below. The most anteriorly placed of these showed degeneration of the lateral inferior margin of the thalamus and internal capsule and infiltration with polymorphonuclears and round cells of this area which was adjacent to the disintegration of the forceps major. In the superior portion of the thalamus, adjoining the softening of the centrum, there was some cuffing of the blood vessels with polymorphonuclears and numerous round cells. The remander of the thalamus and internal capsule between these sites contained an increased number of oligodendroglia cells. The section made about 5 mm. posterior to this showed

a marked degeneration, which was relatively acellular, in the cerebral peduncle. There was degeneration with infiltration of polymorphonuclears, lymphocytes, and histiocytes in the lateral portion of the thalamus and internal capsule adjoining the purulent disintegration found in the forceps major. The body of the thalamus like that of the previous section exhibited only an oligodendrogliosis. But in the superior part of the thalamus there occurred a wide irregular border of fibroblastic reaction infiltrated with round cells and polymorphonuclears which resembled an inadequate attempt at capsule formation. This was somewhat more pronounced on the superomesial than on the lateral border. The blood vessel walls were swollen, the Virchow-Robin spaces filled with lymphocytes, plasma cells, and polymorphonuclears. The thalamus in this area obviously was unable to function because of the extensive pathologic change.

Comment. While there were several abscesses in this case, the small ones in the right caudate nucleus and globus pallidus were forward and could not cause the clinical picture of a thalamic lesion. The softening in the right forceps major and hippocampus is equally unrelated to anosognosia. The syndrome of anosognosia is therefore ascribed to the lesion shown in the illustration cutting off the thalamoparietal peduncle. This finding harmonizes with the conclusions of Barkman.

(Case reported by Von Hagen and Ives in the *Bulletin of the Los Angeles Neurological Society.*)

31. *Arteriosclerosis with hypertension. Delusions for one year. Sudden onset of total aphasia. Death six weeks later. Autopsy: softening in left supramarginal and angular gyri.*

J. C. F., a white man 87 years of age, was first seen on Sept. 12, 1937, because of expressed delusions that he was being poisoned. In giving the history, his wife stated that these manifestations were not new and that during the past year he had also had "attacks" of inability to speak. These periods of aphasia would usually last from one to four hours. Aside from these symptoms he had enjoyed fairly good health. When examined on this date his blood pressure was 180 systolic and 100 diastolic and the arteries were markedly sclerotic. The neurologic examination was essentially negative.

On the following day, as the patient attempted to get out of bed early in the morning, the nurse noticed that he could not speak. However, he could utter guttural sounds. From this time on, to October 30 when he died, his only recognizable words were uttered a week following the onset of aphasia. On that morning as the examiner entered his room he was greeted by the patient's wife and in the course of the conversation the name Abbott was used a number of times. Some minutes later the patient rose from the bed and

said in an explosive manner "Dr. Rabbit." He did not even answer "yes" or "no" to questions.

Neurologic examination then showed a slight weakness of the right side of

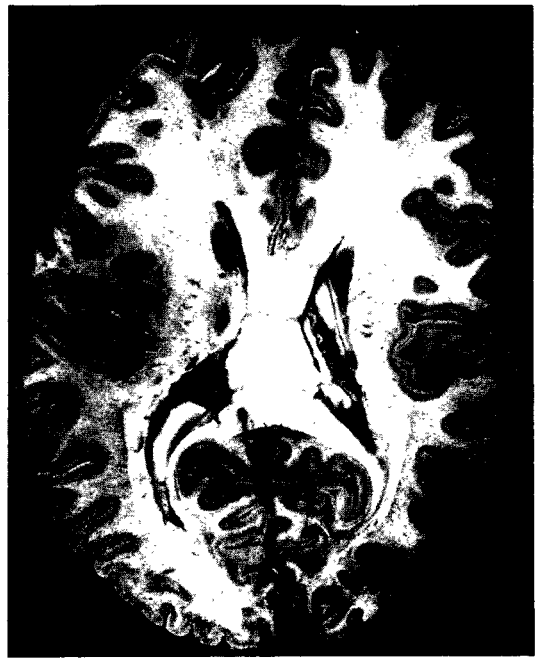

FIG. 52. Case of J. C. F. This represents a horizontal section which passed 1 cm. below the supramarginal gyrus and revealed subcortical softening of the left angular gyrus.

the face, and the right upper extremity. On repeated testing, occasionally a right Babinski sign was obtained. The deep reflexes on the right side were slightly hyperactive. The presence of bilateral cataracts made the study of the eye grounds difficult. Because of aphasia it was impossible to determine whether or not there were any defects in the visual fields. Prior to this illness he had been able to see well enough to get about the house and yard and to write his name. The examiner was never able to get him to show recognition of letters, words, symbols, or objects. Any attempt to have him write always proved futile; he merely ignored the pencil or let it fall. At times it seemed as though he comprehended spoken words as when he liked to have his minister read to him from the Bible. His abortive efforts at speech persisted until three days prior to his death (last examination).

An autopsy (Fig. 52) revealed the cause of death to be coronary occlusion. Aside from this and the generalized arteriosclerosis, the principle findings

were in the brain. On the surface of the brain there was gross softening of the left supramarginal gyrus. Figure 52 shows a horizontal section which passed 1 cm. below the supramarginal gyrus, and revealed subcortical softening of the

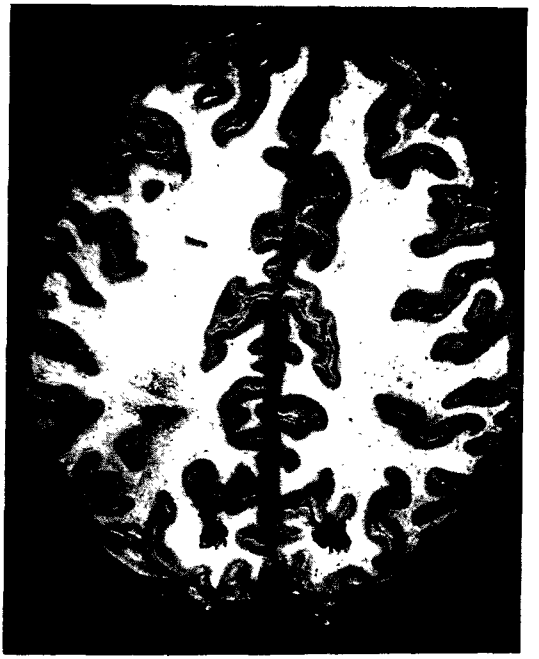

FIG. 53. Case of J. C. F. This section 1 cm. above that of Fig. 52 showed softening completely undermining the supramarginal and angular gyri.

left angular gyrus extending below the supramarginal gyrus. The lower section (Fig. 53), 1 cm. above the first, passing directly through the supramarginal gyrus, revealed softening completely undermining it and also the angular gyrus. These lesions extended well into the white centrum. In addition to these there were three older lesions, small healed hemorrhagic cysts, in each parieto-occipital pole (precuneus) and a more recent softening in the left frontal lobe extending deep subcortically above the basal ganglia.

It is clear that the temporal isthmus was softened.

(Case of Abbott, 1940.)

32. *Finger agnosia, agraphia, confusion of right and left, constructive apraxia, loss of ability to draw, disorientation in time, no aphasia; typical capsular hemiparesis. Autopsy showed metastatic carcinoma of the adrenal, with three lesions in the brain: the first in the left thalamus, the second (subcortical) at the border of the left angular gyrus and the second occipital*

convolution, and the third, a small lesion, lying parasagittally in the left forceps major.

H. L. D., a white man aged 49, was admitted to the general medical service of the Los Angeles County Hospital on July 6, 1936, because of pain and weakness in the lower limbs. He stated that he had been troubled with wheezing in the chest for two years but had not been ill until five weeks before, when he had been compelled to cease his ordinary work as a carpenter because of onset of intense pain in the lumbar region. The pain had been in the nature of a constant ache which spread down the limbs; after two weeks at home weakness had developed so that he was barely able to walk.

Approximately at the time of onset of weakness, he awoke one morning unable to speak as before. Difficulty with speech was of two types: inability to articulate clearly and trouble in finding the words he wanted. The latter symptom never became severe, but the thickness of speech progressed, so that at the time of examination it was with considerable effort that one could understand him.

Details of the medical examination are not given here, as they are not germane to the subject at hand. Autopsy revealed primary carcinoma of the right adrenal gland with generalized metastasis. The pain of which the patient had complained was due to involvement of the retroperitoneal nerves and lumbar plexuses.

Neurologic consultation was first requested by the intern, Dr. Louise Powers, who thought the patient had aphasia. His inability to write was corroborated by the resident physician, Dr. Elinor Ives, who urged more detailed study. It was my opinion at first that there was no real agraphia, as the disturbance of speech was clearly dysarthria and writing was difficult because of weakness of the right hand. Only when it was found that the patient was totally unable to write with the left hand was a detailed study made.

During the two weeks of examination there were no changes except in degree, for which reason one recording is sufficient. During that time the agraphia gradually increased to completion.

General motor function. There was slight weakness of the right lower part of the face, more marked in emotional expression than on voluntary movement. However, the upper limb, especially the hand, was more affected on the right. Although the patient could make a few marks with a pencil held in the right hand, he could not hold it tightly enough to write well. The paresis was specific, and not merely part of the general weakness of the body. He had severe dysarthria, but the difficulty in finding words of which he had complained at the onset was not noticeable.

General sensory function. There was general loss of sensation in the lower

limbs, more marked peripherally and shading off to a point above the knees. This affected the senses of touch, temperature, vibration, and position. Pain on passive motion was extreme. One could not test for a Kernig sign, because the heels could not be raised off the bed without severe pain. Passive movement of the hip joints was much more painful than that of the ankles and toes. There was no other objective sensory loss.

Except for weakness of the right lower part of the face there was no involvement of the cranial nerves. With the expectation of finding defects, we gave special attention to the visual fields, but they were normal.

General psychic function. The patient was remarkably alert and in general was clear mentally. This condition continued until two days before death. However, he fatigued rapidly, for which reason examinations of necessity were short. He was oriented clearly for place and person but not at all for time. This failure stood out in remarkable contrast to his general condition. On July 25 he said that the day was January 8. When the accuracy of this answer was questioned, he stated that at least it was the latter part of January. He did not know the day of the week, nor at 9 A.M. was he sure whether it was morning or afternoon.

His intelligence was above that of the average. He formerly had worked as a drafsman in the office of an architect and consequently had been able to draw and calculate well. He particularly understood plans and illustrations.

Writing. On July 17 Dr. Ives corroborated Dr. Power's statement that the patient was unable to write. He could not write even his own name or the word window on dictation. He made only unintelligible scrawls. He was able to copy slavishly if the sample was constantly before him. On the other hand, he would construct words on dictation with anagram blocks. He thus built up the words window and boat on dictation and the word bag spontaneously, but all with considerable hesitation and many errors, which, however, he went back to and corrected spontaneously. Three days later he also built up for me the words New York on request. At no time was he able to construct even a short sentence, either on dictation or spontaneously. When he found it impossible, he declared, "I am hopeless today." When asked to write 25 he made first a 5 and then placed a 2 before it.

Drawing and constructive apraxia. In spite of considerable experience with drawing, he was unable to draw a face or a hand. In the result of his efforts there was not the slightest resemblance to the object represented. His lines were placed one on top of the other, apparently without any sense of direction. The presentation of a sample made little difference. Although he had often drawn a roof with a dormer window, he could not do so on request. He was also unable to construct a square and a Y with matches, although he

succeeded to a fair degree in building a triangle and a T after a sample had been presented. It was necessary, however, for him to look at the sample constantly in order to do this.

Reading. There was never any disturbance in reading. The patient read whatever was presented to him and also the newspapers.

Recognition of objects. He recognized all objects shown him. He named correctly pictures of a baby, Uncle Sam, and a cat. He identified playing cards and even interpreted correctly action as shown in still pictures.

Apraxia. There was no apraxia except the defects in drawing and writing. He handled all objects about him without trouble and with dexterity in the left hand. With the right hand he had difficulty because of weakness.

Revisualization of images. The patient was able to some extent to revisualize former images. He described well-known public buildings correctly in some details but made flagrant mistakes in others. He did not know with what kind of roof his house was covered, in spite of his interest in architecture.

Orientation for right and left. The patient was entirely disoriented as to right and left. He could not point to parts of his body with either hand, except those in the mid-line. This applied not only to parts of his own body but to the limbs of the examiner as well. He was unable to tell to which side any given finger belonged.

Finger agnosia. He had lost identifiability of the fingers. This was a definite defect of recognition, not merely one of naming. It was also distinct from the question of laterality; i.e., mistakes were constantly made with reference to laterality, but the patient could not recognize the fingers even on one hand. At times he gave correct answers. Mistakes were made constantly with regard to the three middle fingers; the occasional correct answers referred to the thumb and little finger.

He was unable to show a given finger on request or to imitate finger postures. Vision did not guide him; at all times during this test he was allowed to look at his hands. There was also no astereognosis. Mistakes in naming fingers were not so constant as errors in presentation of a finger for which the examiner called. There was, therefore, less finger aphasia than finger agnosia. There was no difficulty in identification of toes.

Calculation. Calculation was faulty, but not absent. He gave correct answers to 4×7, $3 + 9$, and $9 + 5$, but 2×3 was 12, and $4 + 7$, 14.

Observations at autopsy are shown in the illustrations, Figs. 54 to 57.

(Case reported by Nielsen in the *Archives of Neurology and Psychiatry.*)

33. *Apoplectic onset of right hemiplegia and homonymous hemianopia. Paralysis not recognized by patient. Allachesthesia. Generalized autotopagnosia with consequent syndrome of Gerstmann. Loss of recognition of pictures and*

symbols with retained recognition of objects. Apractic agraphia. Autopsy: softening of posterior portion of left angular gyrus and its parasagittal extension in superior parietal lobule plus a superficial cortical lesion lateral to

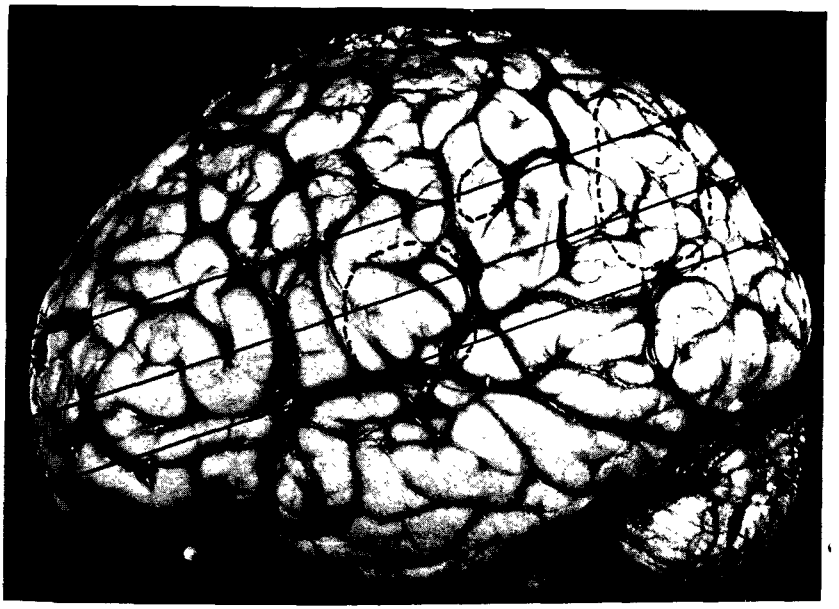

FIG. 54. Case of H. L. D. Photograph of the left side of the brain. The parallel lines indicate the planes of section resulting in the views of Figs. 55, 56, and 57. The lesions seen in the sections have been projected on the surface and then outlined in ink. (Figures 54, 55, 56, and 57 from the *Archives Neurology and Psychiatry*, 39:536 (March) 1938.)

occipital pole and subcortical destruction of calcarine area on left side. Also softening of precentral gyrus, hand and arm area left side. (Los Angeles County Hospital, No. 490–502.)

Charles W. H., an obese white man of 71 years, was admitted on Apr. 3, 1936 because of symptoms due to an inguinal hernia. His family history was to the effect that his father had died at 84 years and his mother at 70 years of carcinoma of the stomach. Four brothers had died of unknown causes, while two sisters remained living and well. He had been married but had had no children and, at the time of admission, was separated from his wife.

The patient had been a locomotive engineer earlier in life. As recently as 1932 he had done light manual work. His medical history was essentially negative. In childhood he had had the usual acute infectious diseases and also smallpox. He had never had rheumatic fever or renal disease, nor had he had any operations or accidents.

Inquiry brought out the fact that he had been troubled for two years with dizziness after exertion and with nocturia. It had been necessary for him to rise four or five times each night to void but there was no burning or other

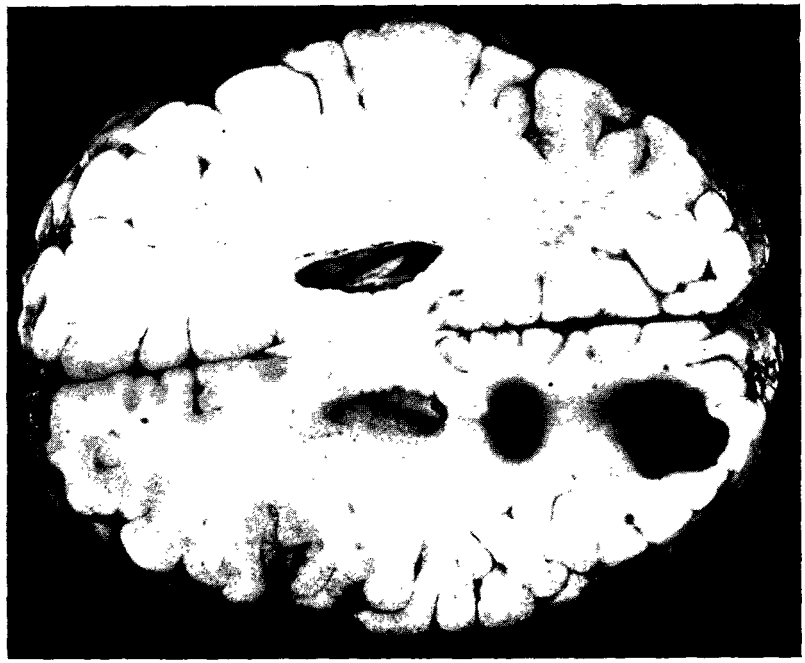

FIG. 55. Case of H. L. D. Highest section of Fig. 54. The larger lesion is located exactly at the border of the angular gyrus on the second occipital convolution. The smaller lesion is forward in the forceps major.

irritation. Sometimes, when walking or coughing, he had pain in the groin. He stated that a physician had told him two years before that he had high blood pressure for which nothing could be done, but recently he had had some carious teeth extracted on the advice of another practitioner.

Examination showed the patient to be an intelligent white man who looked senile but fairly robust. He weighed 191 pounds; body temperature was 98.8 F., pulse rate 76, and respirations 18 per minute. His blood vessels were tortuous and markedly sclerosed and his blood pressure 208 systolic and 120 diastolic. His apex beat was in the fifth interspace in the midclavicular line. Heart tones were of fair quality and there were no murmurs present. The chest was emphysematous, expansion about equal on the two sides. The teeth which still remained were in fair condition and the throat was normal. The abdomen was obese but negative for masses or fluid. Kidneys were not palpable. There

was an incomplete direct right inguinal hernia. The prostate was moderately enlarged, smooth not tender. Neurologically, the examination was entirely negative. There was no physical or serological evidence of syphilis. Blood

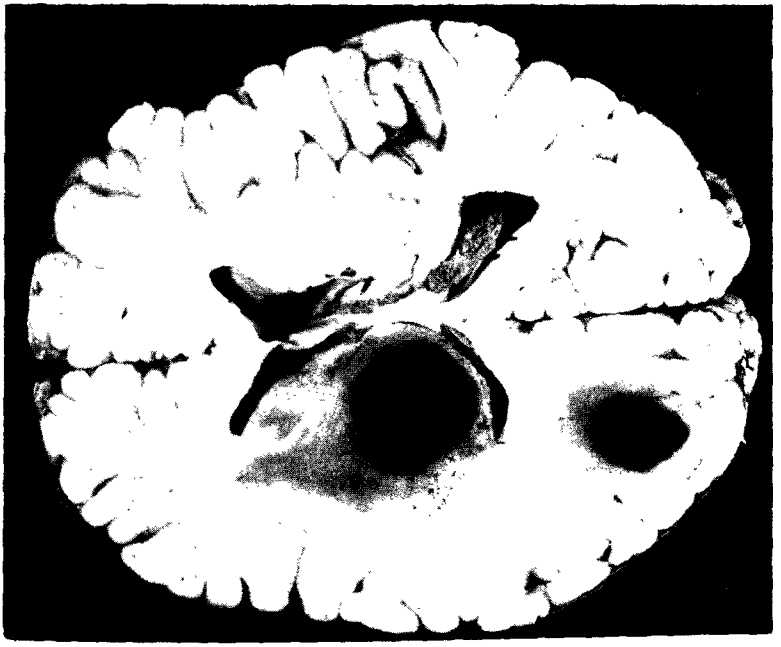

FIG. 56. Case of H. L. D. Middle section of brain in Fig. 54. The two lesions are obvious. The larger is in the thalamus.

count showed 5,210,000 red cells and 7000 white cells per cmm. The urine was normal considering the age of the patient.

After the usual preparation, the patient was operated upon and the hernia repaired. During his stay in the hospital there was no essential change in his condition and he was discharged on July 28, 1936, with a blood pressure of 192 systolic and 116 diastolic.

On June 17, 1937, the patient was readmitted because of a right hemiplegia of two weeks' duration. Although he had been struck by a streetcar a month before admission, there had been no material injury and the paralysis which was present had appeared two weeks later while the patient was walking on the street. He was not convinced that he was paralyzed but admitted having trouble with his gait.

The general physical condition was essentially like that on previous admission except for the hemiplegia. Neurologic examination showed the

pupils to be unequal, the left distinctly larger. Both reacted quite well to light and the extraocular muscles were normal. There was a faccidity of the limbs of the right side, affecting more the upper than the lower limb.

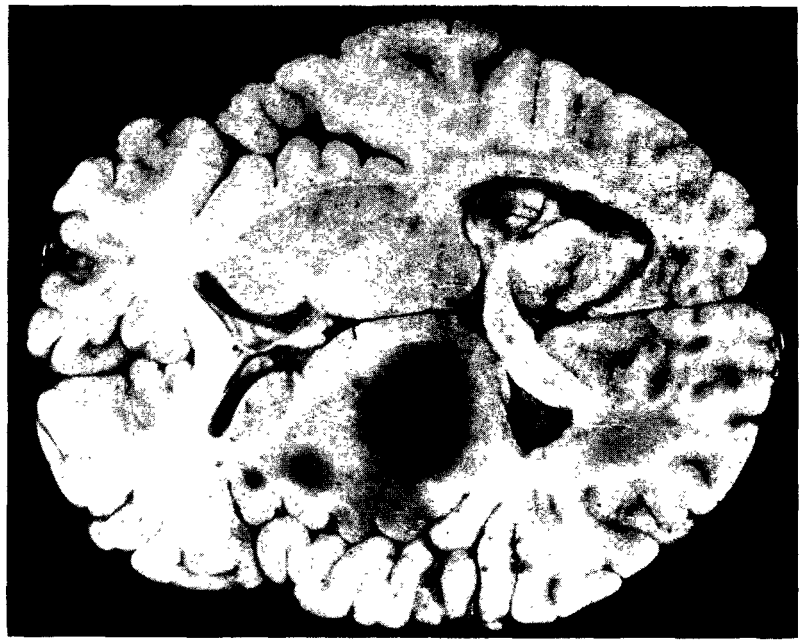

FIG. 57. Case of H. L. D. The lowest section of brain in Fig. 54. The large lesion is in the thalamus. There is also a suggestion of two tiny lesions in the putamen and insula respectively.

All deep reflexes were increased on the left (normal) side and greatly diminished but not absent on the paralytic side. The Babinski sign was present bilaterally. Sensation was unimpaired. It was difficult to determine whether hemianopia was present; there was some suggestion of it. The patient denied his hemiplegia.

For the next two days the patient continued emphatic in his denial of the hemiplegia. He gave various reasons why he could not bring about action in the hand or simply said he could not get it to come up, but to him it was not paralyzed.

On June 18 he was examined by Dr. Elinor Ives but he was unable to give a history. He said he had come to the hospital just for fun; that there was nothing the matter with him. He was co-operative but disoriented. He believed it was Monday when it was Friday. He knew he was in Los Angeles but did not recognize the hospital building. When asked whether it was a

school, hospital, or hotel, he said "school." The condition of the fields of vision could still not be determined accurately. There was no papilledema. The left pupil was the greater; both were irregular and reacted only fairly well to light. The patient looked spontaneously to both sides but could not be made to converge. There was a slight right central facial weakness and the tongue was not well protruded. There was not any specific loss of sensation in the face or body except possibly a slight diminution of position sense on both sides of the body (this was open to question because of the patient's mental state). There was some resistance of the neck to anterior flexion but not to rotation. There was a complete flaccid paralysis of the right arm and weakness of the right leg. At this time Dr. Ives also made an examination of the body scheme, the record of which is combined with further study below.

On June 21, Dr. Clarence W. Olsen made some observations as follows: The patient was conscious but fatigued easily. He looked mostly toward the left and he had a complete right homonymous hemianopia. Whenever he was under emotional stress, a gross rhythmic tremor of the left arm developed. The patient had a paralysis of the right arm and weakness of the face and leg but could not understand what was wrong. While he was unable to move the arm, still he did not consider it paralyzed. His failure to recognize fingers was all out of harmony with his ability to recognize other objects. The eyegrounds were negative, but the patient was unable to name or select colors on request. Tactile localization was greatly impaired.

When he was seen by the author on June 22, a remarkable clinical picture was observed. If one tested his mental acuity by means of his ability to recognize objects one found it keen. There was not the slightest hesitation. On the other hand, the patient was totally unable to recognize pictures. He called the face of a man a horse and other pictures of entirely different objects he called men. In spite of many tests, we were unable to determine any order in this failure to recognize illustrations. He seemed equally at a loss pertaining to human beings and inanimate objects.

He was next tested for ability to recognize fingers. In this field there was a complete loss. Not only his own fingers but also those of the examiner were all alike to him. (In this testing we told him that the objects presented were fingers, asking, "What finger is this?") When he was unable to name them we asked him to demonstrate his own fingers as requested. This he was equally unable to do. The question then arose whether he was confused as to right and left. It was found that he had no idea of laterality. Occasionally he named the side correctly as though by guess. We now followed up the idea of the Gerstmann syndrome and tested for writing. He had a complete agraphia. The writing was, of course, done with the left hand but this did

not explain the defect as he failed even to simulate letters. The fourth element of the Gerstmann syndrome, acalculia, was then tested. Somewhat to our surprise the patient was able to make simple calculations such as 3×4, 3×9, and $21 + 4$. If the calculations became complicated he failed, but there was no more failure than one could ascribe to his fatigue and general illness.

Other elements which have been found associated with the Gerstmann syndrome were then considered, such as the ability to draw. He was completely unable to draw even so simple a figure as a square or a rough circle. There was no semblance whatever in the lines he produced; yet he persisted in his belief that he had drawn the figures asked. His knowledge of symbols was then tested and it was found that he was totally unable to read even letters or figures. He could spell words asked of him but could not recognize the letters when he saw the words he had just spelled. Up to this point, then, we had established as elements associated with or constituting the Gerstmann syndrome finger agnosia, disturbance of right and left, agraphia, and drawing defect. Acalculia was not present. There was, however, in addition alexia on an agnostic basis. Of symbols other than letters or figures he recognized a dollar bill and a playing card except for its color. When he mistook a queen of hearts for a queen of spades we tested for color sense and found that he had a complete achromatopsia.

A review of the findings to this point gave rise to the suspicion that parts of the body were far more difficult for the patient to recognize than other objects. Further study of this brought out the fact that he failed not only to recognize which finger or which hand was presented but he failed to recognize *any* portion of the body. He recognized an object held in the hand before him, but if no object was held he did not recognize the hand. Neither did he recognize the face, or features of it as the nose, the ear, or the chin. Occasionally as through a fog of confusion he suddenly named something correctly, e.g., on being stuck on the right arm with a pin he said, "right arm." When this happened he always named the area instantly; if he had to give thought and consideration to the answer he was wrong. He named a shoe correctly though it was applied to the foot at the time. His apparent allachesthesia continued.

It was now clear that we were dealing with an unusually complete autotopagnosia. This was a broader failure than mere finger agnosia or even that defect plus disturbance of right and left. Since the patient was unable to recognize any portion of the body, he certainly could not tell right from left. And since he could not recognize hands he certainly could not be expected to identify fingers. Also, as he could not recognize his limbs, it is not strange that he could not determine the presence of paralysis or at least that he could not determine what was paralyzed. We can therefore restate our case as

follows: The patient suffered from generalized autotopagnosia, apractic agraphia, agnostic alexia, visual agnosia for pictures, achromatopsia, right hemiplegia, and right homonymous hemianopia.

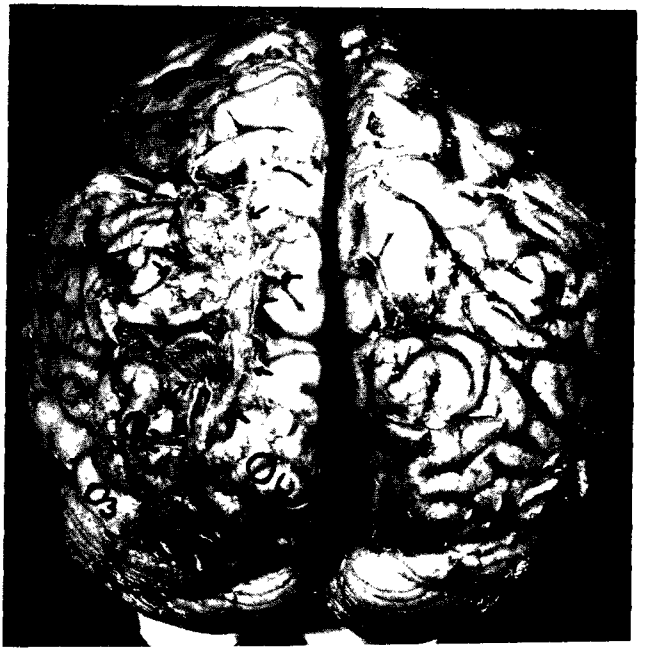

FIG. 58. Case of Charles W. H. Occipital view of the uncut brain. The arrows point to the margins of the softening. *O1*, *O2*, and *O3* are placed on the corresponding convolutions.

The patient failed rapidly during the following day and could not be tested for estimation of distance, stereoscopic vision, or sense of depth. He died two days after the completion of the study. Because of the history of injury by a streetcar a month earlier, the autopsy was performed by the coroner who made a few sections of the brain from the mesial surface and then referred it to us for further study. Our sections were of necessity made as extensions of those already started.

The uppermost section (Fig. 59) shows softening in the precuneus leaving a little less than 1 cm. thickness of the mesial portion spared. The lateral-most point of softening reaches laterally 3 cm. from the sagittal plane. The lesion extends forward from the posterior cut surface 3 cm. This softening is in the superior parietal lobule.

The second section (Fig. 59) parallel to the first passes directly through the angular and supramarginal gyri. It reveals a salient of softening com-

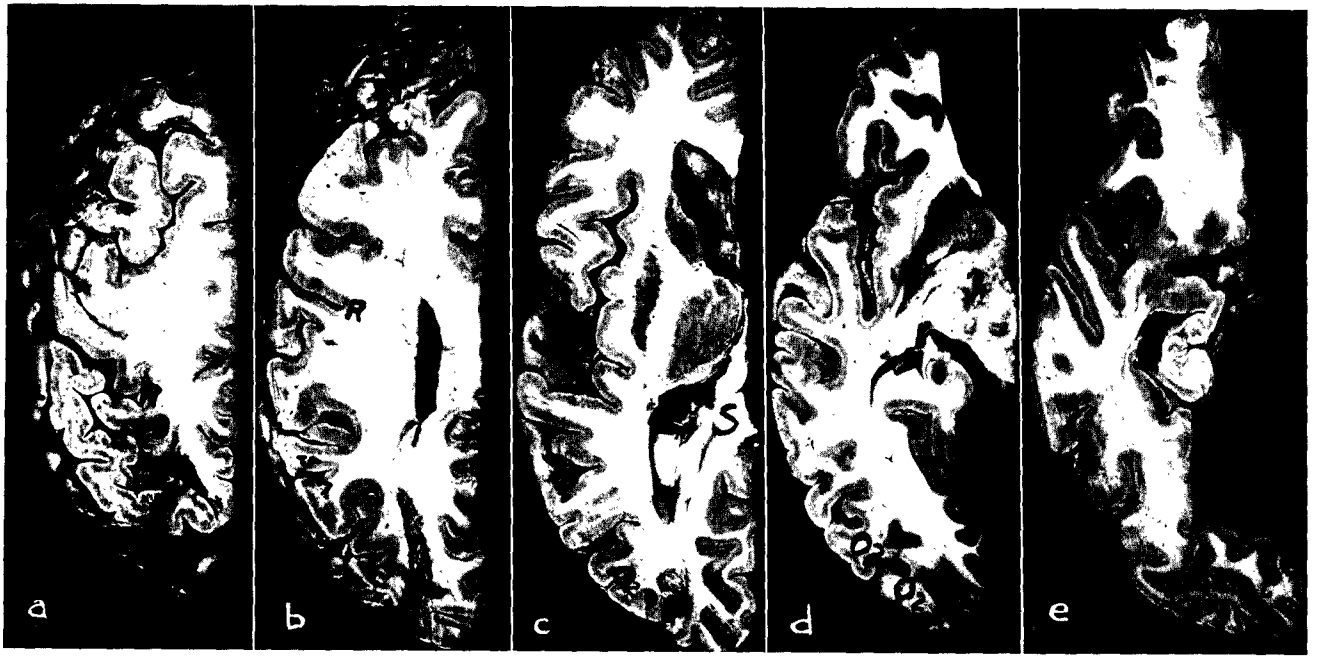

FIG. 59. Case of Charles W. H.

a. Uppermost section showing the softening in the left parieto-occipital region. *R* is placed at the fissure of Rolando.

b. Second section 14 mm. below the first. *R* is again placed at the fissure of Rolando and the two small dark lesions are seen in the prerolandic sulcus. On microscopic examination these are seen to be disintegrated areas of softening. *SM* represents the supramarginal gyrus and *A* the angular gyrus. The salient of softening is seen to extend inward, and adjoining the lateral ventricle there is softening so that the supramarginal as well as the angular gyrus is entirely undermined.

c. This section, made through the convolution of Broca and the center of Wernicke, shows the lesion mesial to O_2 (second occipital convolution). It is seen that fibers of the splenium (*S*) of the corpus callosum can still reach O_2 so that impulses from the right calcarine area could stimulate O_2 of the left side.

d. This section shows cortical destruction of the mesial portion of O_2 (where the mesial mark O_2 is placed) while the lateral portion is intact.

e. This section shows the lateral portion of O_3 (third occipital convolution) spared. In the mesial portion there is cortical destruction.

pletely undermining the angular gyrus reaching forward exactly to the posterior border of the supramarginal gyrus, measuring 3.5 cm. from the posterior cut surface. The slender streak measures only 3 mm. in width. There is also softening independently in the posterior margin of the ventricle. Close inspection shows also two small dark areas in the anterior and posterior portions of the precentral gyrus which, on microscopic examination, are found to be areas of complete disintegration from softening more than a week in age. This is in the hand and arm area and explains the paralysis of the right upper limb.

The third section (Fig. 59) passes through Broca's convolution and Wernicke's center, both of which are intact. It also passes through the posterior margin of the angular gyrus mesial to which is the cuneus (of which the first occipital convolution is a portion) and this is found to be softened. This lesion affecting the optic radiation and subcortically the area striata explains the hemianopia.

The fourth section (Fig. 59) passes through the second occipital convolution and shows disintegration of the mesial portion of this area as well as small foci forward in the second temporal convolution. There are also dilated spaces from small but old lesions in the putamen. Microscopic examination shows these to be many months old and of no material interest in the case.

The fifth section (Fig. 59) shows the cortex of the mesial portion of the third occipital convolution affected by softening. The lateral portion is preserved.

The occipital surface view (Fig. 58) gives one a good idea of the extent of the softening and its relation to surface markings.

CONCLUSIONS

I. When an agnosia, apraxia, or aphasia appears there is either no performance in the sphere of function under consideration or there is faulty performance. In either case the major cerebral hemisphere is affected. Absence of performance means that neither hemisphere is functioning; faulty performance means that the minor hemisphere is working imperfectly.

II. A lesion in the isthmus of the major temporal lobe incapacitates the major language area in its entirety.

III. Agnosias which have a highly specific localizing value are:

1. Visual verbal agnosia without agraphia. The lesion affects the white substance beneath the cortex of the major angular gyrus. (There is practically always an associated hemianopia.)

2. Visual verbal agnosia with agraphia. The lesion affects the cortex of the major angular gyrus.

3. Visual agnosia for mathematical figures. The lesion affects the cortex or subcortex about the major interparietal sulcus.

4. Acoustic verbal agnosia. The lesion affects the paratransversal portion of the major superior temporal convolution (Wernicke's area.)

IV. Agnosias which have some degree of localizing value but not highly specific are:

1. Tactile agnosia (astereognosis). The lesion affects the thalamoparietal fibers of the opposite side at any point. (The lesion may be cortical or subcortical.)

2. Subcortical acoustic verbal agnosia. The lesion is usually bilateral (in both temporal lobes) but a large lesion in the major temporal lobe may cause it.

3. Geometric-optic agnosia. The lesion affects the major parieto-occipital region, cortically or subcortically. The major occipital lobe, however, is not necessarily ipsilateral with the major temporal lobe.

4. Visual agnosia for objects and pictures. The lesion affects either both occipital lobes or the major one plus the splenium of the corpus callosum. In most cases the lesions have been subcortical.

5. Visual agnosia for animate objects. The lesion affects either occipital lobe.

6. Visual autotopagnosia in general. The lesion affects the major occipital lobe.

7. Visual agnosia for fingers. The lesion affects the border between the major angular gyrus and the occipital lobe.

8. Optical disorientation in space. The lesion affects the major occipital lobe.

9. Hemiachromatopsia. The lesion affects the occipital lobe of the side concerned.

10. Acoustic musical agnosia. The lesion affects usually both temporal lobes at the anterior extremity. Occasionally only the major side is affected.

11. Visual musical agnosia. The lesion affects the major parieto-occipital lobe.

12. Acquired cerebral color blindness. The lesions affect the inferior portions of the occipital lobes of both sides, the poles escaping.

V. Apraxias which have a highly specific localizing value are:

1. Kinetic apraxia of the limbs (cortical motor pattern apraxia). The lesion affects the corresponding or the major precentral convolution.

2. Apraxia of swallowing. The lesion affects the major para-rolandic operculum or both sides identically.

3. Apraxia of (motor) speech. The lesion affects the fibers from the major convolution of Broca on their way to the ipsilateral precentral gyrus.

VI. Apraxias which have some degree of but not great localizing value are:

1. Ideokinetic apraxias. The lesion or lesions affect the corpus callosum or the subcortical region about the major supramarginal gyrus.

2. Amusia instrumentalis. Unless it is part of a general apraxia the lesion is in the third major frontal convolution.

3. Apraxia of visual finger guidance. The lesion affects the major parietooccipital region.

VII. Apraxias which have no localizing value are:

1. Ideational apraxia.

2. "Amnesic" apraxia.

VIII. Aphasias which have localizing value are:

1. Motor aphasias (already listed as apraxias).

2. Wernicke's aphasia. The lesion affects the major temporal lobe, especially the superior temporal convolution in its posterior one-half.

3. Aphasic alexia. (This form of alexia is to be distinguished carefully from agnostic alexia.) The lesion affects the major temporo-occipital area.

4. Amnesic aphasia. The lesion affects the major temporal lobe.

5. Formulation aphasia. The lesion affects the major temporal lobe, chiefly area 37 of Brodmann.

6. Jargon aphasia—formulation aphasia—paraphasia (see 5 above).

IX. Alexia must be analyzed into its elements. Alexia as a unit has no localizing value.

X. Agraphia has no localizing value unless it is the only symptom present. It may then be caused by a lesion at the border between the major angular gyrus and the occipital lobe or by a lesion of the major second frontal convolution.

XI. Certain syndromes have a high degree of localizing value.

1. Gerstmann's syndrome (of acalculia, confusion of laterality, "finger agnosia," and agraphia) results from a lesion at the border of the major angular gyrus on the occipital lobe.

2. Simultanagnosia of Wolpert (not an agnosia). It results from a lesion of the major occipital lobe.

3. Apperceptive blindness of Pick. This results from a diffuse cortical lesion of the major occipital lobe.

XII. The aphasias not specifically mentioned have no appreciable localizing value.

APPENDIX

EPITOME OF AGNOSIA, APRAXIA AND APHASIA WITH PROPOSED PHYSIOLOGIC-ANATOMIC NOMENCLATURE *

THE nomenclature of agnosia, apraxia, and aphasia has evolved in the manner of a straggling city and for the same reason, namely that independent workers have taken different viewpoints and have become interested in one phase of the subject at a time. The terminology is in some instances anatomic, in other physiologic, in still other psychologic. Certain terms have even arisen in the minds of pedants who have constructed hypothetical diagrams on the basis of which they have invented anticipated types of clinical manifestations which were named before they were observed.

Just as grammarians arise in a nation and systematize terminology to bring intellectual order out of the chaos of human expressions, so have organizers arisen in the study of aphasia. Their efforts have been laudable but unfortunately premature because of lack of anatomic-physiologic background for their conclusions. Kinnier Wilson stated in his excellent monograph of 1926 that not only was the subject not understood but the approach was not agreed upon. He envisioned an anatomico-physiologic nomenclature as the ideal to be striven for on the grounds that a psychologic terminology was too destructive to the subject of cerebral localization to be acceptable.

Through the great revival of interest in the subject of aphasia due largely to the enormous compilation and intensive study of Henschen and the immediately subsequent invaluable contributions of neurologic surgeons, notably Foerster, Penfield, McKenzie, Gardner, German, and others, so much information has accumulated during the last twenty years that Wilson's ideal seems about to be attained.

It is with considerable trepidation and fear of being considered presumptuous that I offer the present system of nomenclature. However, a study of the system will immediately make it evident that I am presenting exceedingly little by way of original terminology; I am rather organizing present knowledge. The terms agnosia, apraxia, and aphasia can be retained with slightly modified definitions. The greatest single change suggested is one of the concept of the function of the minor cerebral hemisphere in language. Even this concept is far from new as it was suggested by that great master Hughlings Jackson and elaborated considerably by Henschen. Pötzl's work in localization and Kleist's intensive studies are landmarks worthy of careful study.

* By J. M. Nielsen. Reprinted by permission from the *Journal of Speech Disorders*, June 1942.

THE FUNCTIONS OF THE MINOR CEREBRAL HEMISPHERE

It is customary to speak of the two cerebral hemispheres as being dominant and recessive, respectively. The terms are based on the conception that the one ordinarily takes the lead and leaves little or nothing for the other to do in the associative sphere of activity. It is tacitly understood that dominance does not apply to projection fiber systems or to the basal ganglia.

The term *dominance* or *dominant hemisphere* is expressive but inaccurate. The studies of many excellent authors on thousands of cases have shown that the relation of the so-called dominant hemisphere to the recessive is such that it can be much more accurately represented by the relationship of pilot and co-pilot on an airliner. In the beginning of their associated careers the pilot is slightly more expert than the co-pilot, but in case of sudden incapacity of the pilot the co-pilot can manage the ship almost equally well. The pilot does not dominate the co-pilot. I have suggested the use of the terms major and minor hemispheres as being more appropriate than dominant and recessive hemispheres because they suggest merely "more" and "less." The terms major and minor are already in wide use in industrial medicine relative to hands, feet, and eyes.

There is, however, an important discrepancy between the two relationships in our simile (the pilot–co-pilot relationship and the two-hemisphere relationship) which must be clarified. The pilot ordinarily turns over the management of the ship to the co-pilot during at least a short portion of each flight and the co-pilot therefore remains proficient. The major hemisphere ordinarily never turns over its functions to the minor but instead becomes more and more proficient while the minor misses opportunity for further training. It is capable, however, of rapid training when the necessity arises if the patient (and the minor hemisphere) are not too old and if they are in good general condition.

So far as the *language* function of the brain is concerned, another simile is much more accurate. Consider a hypothetical situation of a group of men traveling together through a foreign country. Only one, the leader, has studied the language of the foreign country and is expert in it. The followers have not studied it. The group travels for some months, when suddenly the leader is killed. The proficiency of the followers to understand and speak for themselves will depend entirely on their individual independence and initiative. Some followers will have learned nothing, some will have acquired a short vocabulary and will have learned to understand a few simple phrases. Others will have read, listened, and spoken for practice with a view to preparedness. A very few will have become proficient.

That is the case with the major and minor cerebral hemispheres. They start through the world together almost equally incompetent. In some instances, when the major is destroyed the minor hemisphere will have developed its engrams so crudely that it does not function at all. In other instances it will be able to formulate language slightly, understand a little immediately, and improve rapidly with training. In a very few instances it will have established its engrams so well that when the major hemisphere is suddenly destroyed, it will be able to assume the functions of language.

When, in this simile, minor cerebral hemispheres were compared with individuals they were actually compared with major cerebral hemispheres, because the indi-

viduals in the illustrations were using their major hemispheres. While there is a hereditary determinant to make a certain hemisphere the major one, there is no essential difference between the functional capacity of major and minor hemispheres at birth. This has been shown repeatedly by instances of severe trauma to the major hemisphere early in life. In such cases the minor hemisphere invariably takes over the function of the major with such precision and dexterity that observers do not notice any disturbance of language. Handedness does not develop until the age of about 9 months to a year. Even when it begins to manifest itself the superiority of the major does not appear great. This is proved by the fact that injury to the major hemisphere at the age of 4 or 5 years causes only transient aphasia. Whenever a minor hemisphere is called upon to take over a function it at first fatigues with astonishing rapidity. As it becomes trained it develops endurance rather than specific capacity.

"Majority" and handedness.

The common hereditary tendency to right-handedness is actually a tendency to left-brainedness; the brainedness comes first. The handedness can be shifted by training early in life but the brainedness, as a unit, does not shift with it. Well-meaning mentors frequently change left-handed individuals to right-handedness at so early a time in life that the subject may not be aware of it at all. This accounts for some instances of right-brainedness in right-handed persons. Aside from these occasions, however, there are instances of left-brainedness in left-handed persons. Ipsilaterality of handedness and brainedness occurs, according to the figures of Chesher, in about 6 per cent of all persons. Hereditary right-brainedness, as shown by the fallible index of left-handedness, occurs in about 10 per cent of all persons. Because of these discrepancies between handedness and brainedness it is never safe to lateralize a cerebral lesion on the basis of aphasia alone. Unequivocal lateralizing signs based on projection fiber systems must be relied upon.

THE ELEVEN SUBDIVISIONS OF A CEREBRAL HEMISPHERE

Up to this point, to facilitate clarity, the discussion of function of the minor cerebral hemisphere has centered about it as though it were a unit. Extensive research, however, has shown that the following subdivisions must be considered separately:

(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 (area 39 of Brodmann).
(9) Area 18 of Brodmann.
(10) Area 19 of Brodmann.
(11) Convolutions of Gratiolet.

These eleven areas are all association areas, i.e., they are not areas of cortex concerned immediately with reception or transmission of impulses over the projection

fiber tracts. The eleven areas occur in pairs, of course, but the degree to which the major in each case is superior to the minor is not the same for all pairs. Disregarding for the present the exceptional cases one may state the case for each area in the average person as follows:

The prefrontal lobes, so far as their intellectual functions are concerned, either establish no unilateral superiority or establish it to a small degree. They seem at times to divide the various functions of the lobes between them, this being manifest by the appearance of certain symptoms regardless of which frontal lobe is affected. But in any case either right or left prefrontal lobe seems capable of assuming the necessary function of both after a short period of training (1).

Area 18 of Brodmann (area parastriata) is concerned with recognition of objects and pictures. The evidence available at present indicates strongly that either the right or the left becomes the major during the first year of life and that the selection is purely a matter of chance. In most persons the degree to which either becomes the major is relatively slight and destruction of the major one leads to only temporary disability of recognition of objects. In a few persons (relatively speaking) the degree of superiority of one becomes so great that its destruction leads to visual agnosia for objects lasting for years. In most instances the major area 18 is ipsilateral to the major temporal lobe (when the major temporal lobe is determined by language function). In a few instances the right remains the major because of an early fixation even though the major temporal lobe becomes established on the left side (2).

Area 19 of Brodmann (area peristriata) is concerned with revisualization of former images. It follows the rules of lateral superiority described as governing area 18 (2).

The three groups described (prefrontal lobe, area 18, and area 19) are not concerned with language and this fact probably underlies the weakness of unilateral superiority. There is much evidence that the superiority of the occipital lobes actually shifts, when it does shift, with the establishment of language centers (second year of life). The lowest function which can be called language is music. It will be seen as we proceed that the areas concerned with vocal, instrumental, and auditory music perform their function almost equally well on the two sides.

The *pars triangularis of the third frontal convolution* (area 45 of Brodmann) is concerned with all of the functions of vocal music and the playing of musical instruments. Injury to it causes apraxia for these acts if it causes any symptom. But it has been abundantly verified that the minor side when called upon takes over the function with great ease and that in only rare instances is there any functional defect after injury to only one side (2c).

The *anterior extremity of the superior temporal convolution* (area 38 of Brodmann) is concerned with musical auditory recognition and interpretation. It receives impulses from the transverse gyri of Heschl which act as centers of primary auditory perception. Destruction of area 38 of either side usually produces no disturbance of musical comprehension because the other side takes over the function. Destruction of the area bilaterally destroys the auditory musical sense (2c).

Wernicke's area (areas 41 and 42 of Brodmann) is concerned with interpretation of spoken language. Comprehension of spoken language is next in scale above comprehension of music and in harmony with this fact the degree to

which the major side ranks above the minor is next in the scale. After removal of the major temporal lobe the average patient is able to comprehend about five or six questions addressed to him. He (his minor temporal lobe) then fatigues. After a year he usually understands a great deal of what is said and after two years one can as a rule hardly tell in ordinary conversation that anything has been removed. The right has assumed the function (3).

Area 37 of Brodmann is concerned with formulation of language. This function is really the one which has been called internal language. Destruction of area 37, or in some cases even relatively slight damage to it, results in inability of the patient to find his words and formulate his sentences. The degree to which the major side is superior is almost identical with that described for the area of Wernicke (4).

Formulation of language is a complex cerebral function dependent on several subsidiary functions. An area of cerebral cortex essentially identical in extent with area 37 of Brodmann is of the utmost importance for formulation of language, either written or spoken. Difficulty with word finding occurs in some cases of angular gyrus lesions, in perhaps one-half of the instances. The same difficulty occurs in a few instances of lesion of the posterior portion of the superior temporal convolution, i.e., when the lesion is behind Wernicke's area. Lesions of Wernicke's area itself interfere with formulation of language to such an extent (as a rule) that the patient does not speak.

These facts explain the views of Kleist that reauditorization of a word is necessary to formulation of language. He calls area 37 of Brodmann an acoustic-psychic sphere.

Kleist states that the word sound is not simply apperceived acoustically and then immediately connected with the concept of the object, but it first appears in consciousness. He says that something is meant with that speech sound, that it designates something without the individual's becoming conscious of what specific object is meant. While all this is true, there is nevertheless much more to the determination of the meaning of a word than the process described above. Some words cannot be distinguished by their sound alone or by their appearance alone. Some words alter their meaning according to context. The expression "they read" cannot be pronounced until context is added. We need to know whether the tense of the verb is present or past. And one cannot distinguish between "red" and "read" by the sound alone. The endless punning heard in entertainment is possible because of these peculiarities (imperfections) of language.

Because of these facts I cannot conceive of area 37 of Brodmann as being merely an acoustic-psychic sphere. It is an area in which knowledge is co-ordinated for significance of words and expressions and in which grammar, rhetoric, and syntax are considered in the formulation of language. The area is in constant association with all of the other areas concerned with language and its separation from the other areas by organic lesions may give rise to defects of formulation of language.

Lesions of this area provoke as their most constant symptom amnesic aphasia. The area for formulation of language is chiefly area 37 of Brodmann but this area is so trained by association with Broca's convolution, Wernicke's area, and the angular gyrus that in some instances a lesion of one of these cripples the function of area 37 sufficiently to cause it to "resign" and turn over the function of formula-

tion of language to the identical area of the minor side. The result is amnesic aphasia. I believe that this disturbance should be called formulation aphasia. (The term "formulation" need not be negative so long as it is followed by "aphasia.") Whether misuse of words, use of wrong words (commonly called paraphasia), or the more severe forms called jargon aphasia or agrammatism result depends on the degree to which the minor side takes up the function. The minor side is usually capable of assuming to some degree the function of formulation of language, but, until it has had time for training, it functions imperfectly. The most pronounced defect is seen in loss of ability to associate objects and their names.

Broca's convolution contains engrams representing the physiologic basis of memory of how to use the vocal organs for the production of words. These engrams are formed simultaneously with, or very shortly after, those of Wernicke's area. The major is superior to the minor in about the same degree as already stated for Wernicke's area, i.e., far superior. Destruction of it in adulthood leaves the patient, in the great majority of cases, able to say only a few words, which he uses for all responses. Usually several years are required for training of the minor side; but as a rule, if the general cerebral function is of good quality, the minor side becomes nearly as proficient as the major had been (8).

The convolutions of Gratiolet, located between the angular gyrus and the second occipital convolution (zone between areas 19 and 39 of Brodmann), are concerned with the body scheme. As the naming of parts of the body, especially naming of fingers and designation of laterality, includes phases of language, disturbances of the body scheme follow the rules for language areas rather than those for the nonlanguage areas regarding laterality. The major is superior to about the degree described for the area of Wernicke, i.e., greatly superior, as the syndrome occurs with great regularity from lesions of the major side (5).

The frontal writing center and the *angular gyrus* are concerned with the most highly specialized of language functions, emission and reception, respectively, of written and printed language. The writing center at the foot of the second frontal convolution is essential to the memory of handwriting movements. In the majority of cases it cannot function without close association with the convolution of Broca. It cannot, of course, function in writing unless the language formulation area (area 37 of Brodmann) and the area of revisualization of the written word (angular gyrus) are intact and in communication with it. The major writing center is far superior to the minor in most cases. A long period of training is usually necessary for the minor to function at all well. Unless the destruction of the major occurs early in life, the minor rarely attains a degree of perfection comparable to that which the major had enjoyed. Conversion of a left-handed person to right-handedness for writing establishes a writing center on the minor side (6).

The *angular gyrus* has a double function. Its cortex contains patterns of two types, one for recognition, the other for revisualization of the written and printed word. The patterns of both types are spread diffusely over the cortex and the superiority of the major is great. The minor angular gyrus in most instances has its engrams so crudely made that, after destruction of the major side in adulthood, many years are required for it to perform at all well. Unless the major is destroyed early in life, the minor rarely attains a degree of perfection comparable to that of the major.

The angular gyrus is trained in very different ways in different persons and consequently its degree of ultimate independence varies enormously. When a child learns to read he at first associates very intimately the engrams for the enunciated word (for the word spoken by himself) with the auditory memory engram of the word which he reads. This is obvious when one recalls that the child reads aloud; he *says* the word and he *hears* himself say it. During the formative period this association of the engrams of the angular gyrus with those of Broca's convolution is so intimate that a lesion of Broca's convolution prevents the child from even *recognizing* the written or printed word. If his training in reading does not advance beyond the point of enunciating (or at least retaining a habit of lip movements while reading) the angular gyrus never becomes independent of Broca's convolution. If the child is taught to read by simultaneous writing of the word the angular gryus may become and may remain dependent on the frontal writing center also. In such cases alexia results later in life from a frontal lesion. However, in the majority of educated persons the angular gyrus develops, by silent reading, an essential independence of the centers in the frontal lobe.

The situation is somewhat different regarding the continued dependence of the angular gyrus function on the area of Wernicke. In the area of Wernicke one "auditorizes" the word one is reading and this habit in most instances continues throughout life. For this reason, at any time of life, in the majority of cases, destruction of Wernicke's area, or destruction of the short cortical area between the angular gyrus and Wernicke's area, produces alexia by preventing not only interpretation but also recognition of the written or printed word. It is because of the elaborateness of the angular gyrus connections that the major becomes so strongly superior to the minor and that training of the minor, when the necessity arises, is extremely difficult (7).

In addition to these important facts about the functional capacity of the various cortical areas of the minor side there is another principle which has not been enunciated, but which is becoming more and more evident and which is of the utmost importance to the understanding of localization in agnosia, apraxia, and aphasia. This is the principle that a given area of cortex concerned with language may, after a minor lesion in it, either attempt to carry on its function, though crippled, or resign immediately. The question whether it continues to function after it is crippled or resigns depends on the facility with which the corresponding minor area can assume the function. Thus a strongly superior language formulation area (area 37 of Brodmann) may continue to function, though imperfectly, after a minor lesion, while the same area in another case with a similar lesion may resign and turn over the function to the minor area. There is no absolute criterion by which an examiner can determine which has happened in a given case. In the majority of cases the damaged area resigns its job. It is for this reason that the brain of a person who has been unable to formulate his sentences may show a tiny lesion in the area concerned or it may show a complete destruction of the area. The whole matter resolves itself into this question, "Can the crippled major area perform with greater ease than the normal but untrained minor area?" The individual in question will take the easiest course. With these facts before us we can see that a positive agnostic or aphasic symptom has a positive value in diagnosis but a negative one has little if any value. If the projection fiber signs and symptoms

indicate that an area concerned with aphasia is destroyed, but there is no aphasia, the negative finding of absence of aphasia has little if any weight in determining the extent of the lesion.

We now come to the question, "How complete must the defect be to constitute agnosia or aphasia?" If, following a lesion of the convolution of Broca, a person is able to speak but with stammering and hesitancy, some students might say that he does not have Broca's aphasia. But we know that from the standpoint of cerebral localization he does have a lesion and that the reason for his being able to talk at all is either that the crippled area is performing or the imperfectly trained minor convolution of Broca is doing the work which we observe. We should therefore say that the patient does have aphasia (apraxia of speech). If the patient, after such a lesion, speaks as well as he did before he either had two equally capable convolutions of Broca or it was the minor one which was destroyed, the minor one merely having been located on the side where we expected the major one to be. Our misinterpretation is due to the erroneous assumption that handedness is an infallible guide to brainedness.

We thus arrive at the principle that a disturbed function of language has the same weight in cerebral localization as a complete loss of the function. A corollary of this is that in the study of human cerebral function in the associative field (by the use of clinicopathologic material to which we are limited) it is impossible to determine the function of any area unless we confine our studies to *bilateral* lesions of the area in question. If this principle had been recognized earlier, millions of words could have been omitted from the scientific literature on aphasia and allied branches. If a given area is bilaterally destroyed we are certain of a functional defect. The determination of the functional defect by a clinical study is not difficult in the light of present knowledge.

APRAXIA

The function of the minor cerebral hemisphere in acts not pertaining to language is far greater than in language functions because handedness and brainedness are largely governed by the development of language. It is for this reason that apraxia is far rarer in the nonlanguage sphere than in the apractic aphasias.

In the precentral gyrus are located patterns of movement, physiologic patterns for co-ordinated motor performance. Physiologically speaking, on a par with these patterns are many others. Loss of function of these patterns has been called kinetic apraxia of the limb (limb-kinetic apraxia of Liepmann). I have suggested the term cortical motor-pattern apraxia because in many instances the limbs are not involved. Illustrations of this type of apraxia are: Broca's aphasia, dysarthria and dysphagia resulting from a lesion of the precentral gyrus of the mouth area, paralysis of voluntary lateral gaze with preserved reflex lateral gaze resulting from a lesion of the foot of the second frontal convolution, apractic agraphia resulting from a lesion of the frontal writing center, loss of finger movement with preservation of use of the upper limb as a whole resulting from a lesion of the precentral gyrus, avocalia (of Henschen) from a lesion of the pars triangularis of the third frontal convolution, and probably a few other instances of disorganization of movement resulting from lesions of area 6-a, 8. In all forms pertaining to language unilateral dominance is relatively strong; in the others dominance in most persons plays a minor role.

These various centers of performance of *movements* are in turn governed for the performance of *acts*. An arbitrary differentiation must be made between movements and acts, the latter consisting of a series of the former co-ordinated to the carrying out of a plan of action. There is no "center" for carrying out an act. Loss of the power to do so (in the absence of paralysis, ataxia, or dementia) is ideokinetic apraxia. Lesions of the corpus callosum or deep lesions of the major supramarginal gyrus are most potent but in any case we are dealing with a disturbance of associative function and not a cortical lesion.

There is relatively slight dominance of one hemisphere over the other in ideokinetic apraxia for which reason the condition is rare relative to aphasia and usually transient. Surgical severance of the corpus callosum does not cause apraxia. The ideational plan of action can usually be formed in either hemisphere for both lateral sets of limbs. Ideokinetic apraxia represents physiologically an interruption between the ideational plan and the patterns for execution of an act.

When the ideational plan itself is disturbed, i.e. when an ideational plan cannot be kept in consciousness long enough for its execution, the condition is called ideational apraxia. This condition is always due to a diffuse cerebral lesion, quite often a toxemia.

SELECTION OF TERMS

The reasons for the choice of each term will be much clarified by development of the physiology in brief and simple language. The function of area striata has been designated as *primary perception*. While it is true that there is no perception in the usual sense until the impulse reaches consciousness, and since the visual impression does not reach consciousness until impulses reach the diencephalon, the area striata does not really perform the function of perception. However, common usage has dignified the term primary perception, and it seems wise to keep it.

AGNOSIA

So far as the cortex is concerned, the next physiologic step above the level of primary perception is recognition and for this function area 18 of Brodmann is essential. Loss of the function of recognition resulting from an organic cerebral lesion and through one sense organ only is *agnosia*, and a distinction must be made between agnosias for each of three senses; olfactory and gustatory agnosias are not definitely known. Loss of the function of visual recognition is *visual agnosia*. From the standpoint of cerebral localization it is necessary to distinguish between recognition of animate and inanimate objects because one function is not infrequently lost without the other. And as objects have many attributes such as form, color, distance (dimension), and direction and as each of these attributes has been known to be lost separately through an organic lesion of area 18 (parastriata) we are compelled to acknowledge *general visual object agnosia, visual animate object agnosia, visual inanimate object agnosia, visual form agnosia, color agnosia, visual distance agnosia, and visual direction agnosia*. In the realm of animate objects, organic cerebral lesions cause agnosia separately for certain parts of the body, especially the fingers. We thus have *visual finger agnosia*. Visual agnosia for the entire body has occurred from a cerebral lesion and this has been described as autotopagnosia (referring to agnosia for topography of oneself). The term, however, should be qualified by the adjective visual, making *visual autotopagnosia*. The

lesion causing this condition is found in the occipital lobe, areas 18 and 19 of Brodmann of the major side, or both sides. All visual agnosias not concerned with language result from occipital lesions, but in order to have a consistent nomenclature an anatomic term must be added to each.

The localization of the lesions causing agnosias *in the sphere of language* is not so simple. The area of cortex essential to visual recognition of letters, syllables, words, and musical notes is in the angular gyrus. The similar area for mathematical figures is made up of the borders of the interparietal sulcus. (One little exception to these statements which must be made because of its importance in treatment of certain cases is this: when the infant first learns letters they are to it not symbols but objects. They are therefore stored as engrams in area 18 of Brodmann at that time. They may remain there ready for use and this explains the fact that adults who have had their angular gyri destroyed and who consequently are totally unable to recognize syllables or words, sometimes recognize letters and are thus able to spell words aloud and recognize them by their sounds.) Visual verbal agnosia, visual literal agnosia, visual numeral agnosia, and visual musical agnosia all result from cortical destruction of the corresponding cortical areas. But this is not the only possible site for the causative lesions because subcortical destruction in the same area or between the cortical areas in question and the occipital pole may cause it also. Moreover, in the cases in which the individual affected has established strong associations between the angular gyrus and Wernicke's area (which is the rule) or between the angular gyrus and the convolution of Broca (the exception) destruction of these associated areas may cause visual agnosia for letters, words, etc. In a given case one cannot tell by the agnosia alone where the lesion is. Other findings in the case give the clue. Thus hemianopia would indicate temporal, lower parietal, or occipital location; hemisparesis would indicate a capsular or frontal cortical lesion, etc. Because of these possibilities anatomic terms in the form of adjectives must be added to the physiologic descriptive term in designating the agnosia.

We thus have *visual parietal numeral agnosia, visual occipital numeral agnosia, visual angular literal agnosia, visual occipital literal agnosia, visual temporal literal agnosia, visual insular literal agnosia* (rare), *visual frontal literal agnosia* (rare), *visual angular verbal agnosia, visual occipital verbal agnosia, visual temporal verbal agnosia, visual insular verbal agnosia, visual frontal verbal agnosia, visual subcortical angular musical agnosia, visual musical angular agnosia, visual musical occipital agnosia,* and *visual subcortical angular verbal agnosia.* A corresponding series with the word *syllabic* substituted for *verbal* could be made but is hardly necessary. The distinctions between cortical and subcortical lesions of the angular gyrus with reference to agnosia are made clinically by the occurrence of simultaneous agraphia in the former (cortical) and its absence in the latter.

The auditory agnosias all result from temporal lesions. The reason that temporal lesions can cause visual verbal agnosia while angular gyrus lesions do not cause auditory verbal agnosia rests on the fact that the temporal lobe is trained in the infant and child for four to five years before the angular gyrus comes into function. The angular gyrus is usually dependent on the area of Wernicke but the area of Wernicke is independent of the angular gyrus.

Of auditory agnosias we have *general auditory temporal agnosia, auditory temporal verbal agnosia,* and *auditory temporal musical agnosia.* Auditory verbal ag-

nosia results from a lesion of Wernicke's area and occasionally from lesions in its immediate vicinity within the temporal lobe because in some cases the pressure or edema from a neighboring lesion causes the area of Wernicke to "resign." Auditory musical agnosia is rare because the minor side assumes the function easily. When it does occur it results from a lesion of the anterior extremity of the superior temporal convolution (area 38 of Brodmann). It is entirely useless to consider the site of the causative lesion in transcortical or subcortical sensory aphasia because the lesion is located in a position identical with that for ordinary auditory verbal agnosia. The same statement applies to paraphasia. Paraphasia and senseless repetition of sounds heard is simply due to imperfect performance of the minor side.

Olfactory and gustatory agnosias are still impossible of diagnosis either clinically or pathologically. It is doubtful whether they occur.

Tactile agnosia (astereognosis) may result from a lesion of the parietal lobe (usual) and very rarely from an occipital lesion. In the former case the *tactile parietal agnosia* is unilateral only and contralateral to a lesion of the postcentral gyrus, superior parietal lobule, or supramarginal gyrus. It is probable that the postcentral gyrus is the essential area concerned and that lesions in the other two areas cause tactile agnosia by edema or pressure. When tactile agnosia results from an occipital lesion it is invariably associated with a lesion of area 19 of Brodmann. The patient is unable to form visual concepts of objects by either the visual or tactile route and hence the tactile agnosia (astereognosis) is bilateral even from a unilateral occipital lesion. Such a clinical condition is now known as apperceptive blindness of the senile (Pick) but should be called *occipital tactile and visual agnosias*. This term indicates that the area for revisualization of former images is out of function and its removal is causative of the tactile agnosia (there are two simultaneous agnosias).

When the body scheme is disturbed (by a lesion of the gyri of Gratiolet) the patient may have separate tactile agnosia for the fingers. This should be called *tactile finger parieto-occipital agnosia*. If the entire body scheme is disturbed, *visual autotopagnosia* results.

In the use of the term agnosia, there is only one slight modification of its past usage that needs to be agreed upon, namely that agnosia should apply in any case to recognition from one sense organ only. Its misuse in the past has resulted from inaccurate thinking. Several unacceptable terms have thus arisen. We cannot logically speak of ideational agnosia because ideation goes far beyond recognition. Neither can we speak of loss of finger recognition as finger agnosia unless we mean either visual recognition or tactile recognition. If both are present we have two simultaneous agnosias before us.

The third step in sensory cortical function is that of reactivation of former engrams (images in the visual sphere, sounds in the auditory sphere, tactile reconstructions in the tactile sphere). This third step consists in bringing into consciousness memory from the past. We have been accustomed to speak of it as revisualization in the visual sphere and as recall in the other two spheres. The term recall is distinctly English and not at all specific for the function of cerebral function. The term amnesia is in universal use for forgetting, but forgetting is often a positive process, not merely a failure to remember but a relegation of something unpleasant to the subconscious. Moreover, all agnosias, apraxias, and aphasias are

forms of amnesia. There is an international term which means the process of reliving a former memory, namely reminiscence. The failure to effect *reminiscence* could be termed *irreminiscence* and the verb, now only colloquial in English, *reminisce,* can be dignified with usage. I therefore suggest that the function of the tertiary sensory cortex be called reminiscence. We can then speak of *visual, auditory, tactile, olfactory,* and *gustatory reminiscence* and *irreminiscence.*

The function of the tertiary visual cortex (area 19 of Brodmann) is, then, that of visual reminiscence. By means of area 19 (area peristriata) one relives former visual memories. It must be recalled that area 18 is the servant of area 17 and functions only through it (area 18 serves the purpose of recognizing images primarily perceived by area 17) and one cannot voluntarily stimulate it without allowing area 17 to function again by the patient's voluntary act of looking again. But area 19 is the card index to former memories in the service of all the rest of the brain. One can revive a former image by hearing an associated sound, by feeling an object which is associated in memory, by smelling or tasting a substance which can revive the memory or by seeing an object which has associated memories. But if one reactivates the engrams of area 19 by seeing, one works through area 18. The other four senses reactivate area 19 directly. Its reactivation is reminiscence.

For verbal or musical visual symbols the angular gyrus serves for reminiscence. For numerals the borders of the interparietal sulcus serve the purpose. For reminiscence of the body scheme the gyri of Gratiolet are essential.

Visual irreminiscence is manifest clinically by loss of power to revisualize objects and all attributes of objects such as form, color, direction, dimension and relative distances between objects or parts of objects. Loss of power to revisualize cardinal directions, street scenes, one's home, one's friends or one's body have all been observed following lesions of area 19 of Brodmann. We thus have: *form irreminiscence, visual occipital; color irreminiscence, occipital; distance irreminiscence, occipital; direction irreminiscence, occipital; visual verbal irreminiscence, angular; visual musical irreminiscence, angular; visual literal irreminiscence, angular; visual numeral irreminiscence, parietal; visual finger irreminiscence, parieto-occipital;* and when the body scheme is affected, *visual body irreminiscence, parieto-occipital.*

In the auditory sphere cortical areas concerned with reminiscence cannot be sharply separated from the areas for recognition. It seems that the portion of temporal cortex juxtaposed to the musical and general auditory cortices for recognition serve for reminiscence. It is probable that the area essential to the function varies considerably in different individuals. Clinically it is well known that *auditory musical irreminiscence, temporal; auditory verbal irreminiscence, temporal;* and *general auditory irreminiscence, temporal* occur. One may be unable, after a temporal lesion, to recall a tune but able to recognize it when heard, or one may lose the exact wording of a quotation while retaining a concept of the idea expressed and recognize the words when they are spoken.

APHASIA

Aphasia has been defined in the past as loss of the power to comprehend or express signs and symbols by which man communicates with his peers. It has been agreed that dementia, psychotic disturbances and dysarthria are excluded as causes

and that aphasia is always due to an organic lesion of the brain. We can simplify this definition somewhat by saying that *aphasia consists in organic disease of the cerebral memory engrams for language.* Either of these definitions makes aphasia embrace those agnosias and apraxias which have to do with language. I suggest that in any compound term embodying the word agnosia or apraxia the added term of aphasia is a redundancy and not advisable. We can reserve the use of the term aphasia in our nomenclature for designating "the loss of language association with." Thus *visual finger aphasia* means loss of power to associate the written terms for the fingers with the *object* (finger), *color aphasia* loss of power to associate the language symbol with the color. When one wishes to distinguish between the loss of power to associate the symbol and the loss of power to determine the *significance* of the symbol in terms of language, the adjective *semantic* introduced by Head serves nicely.

There are, of course, all degrees of semantic aphasia even within the present use of the term. So simple a term as the verb *to read* may have a great variety of significances. It may mean:

1. To pronounce the sound indicated by the written symbol without even the faintest understanding, just as I may "read" Welsh (of which I am ignorant).

2. To render the symbol in another language, as to say that this P is an R because the language is Greek.

3. To interpret the word symbol in its most elementary way as to say that that word (cat) means an animal like the one we have in the house.

4. To interpret the symbol in any degree of complexity as to say that "cat" means a domesticated, carnivorous quadruped of the feline family, or to interpret many pages of a legal citation as blackmail, or to interpret the scriptures. To read has even wider meanings as in "to read between the lines" or to read a latent content into a dream.

However, if we are to establish a practical nomenclature such a range of semantics must be discounted. We must be willing to accept an arbitrary distinction between simple recognition and recognition plus interpretation.

It will be necessary to distinguish carefully between the object and its symbol. Loss of *recognition* of the object or its symbol is agnosia for the object or the symbol. Loss of *significance* of the symbol is aphasia.

Inasmuch as all of the old motor aphasias are forms of apraxia it will be an improvement to call them apraxias, or apractic aphasias.

With these explanations we can proceed to elucidation of terms. It is practical to work out from the known agnosias to the aphasias, both anatomically and clinically.

An object may be recognized and comprehended without the patient's being able to establish the symbolic association by seeing or feeling the object. The patient says, "I know what it is but I can't name it." This is the classic amnesic aphasia. Inasmuch as the difficulty is one of language with reference to the object, this is properly called visual object aphasia and tactile object aphasia. In either case the lesion affects the association between the occipital area for visual recognition and the temporo-angular-formulation area or the association between the parietal lobe and the temporo-angular-formulation area. This area is in the posterior part of the temporal lobe, especially area 37 of Brodmann. Hence we derive the terms *visual*

temporal object aphasia or *tactile temporal object aphasia* and both are usually present in the same case. The same syndrome applied to colors need not be encumbered with the adjective visual since colors are recognized only by vision. We therefore select the term color aphasia but as the lesion may be occipital or temporal, we have *temporal color aphasia* and *occipital color aphasia*. Loss of ability to identify fingers by name is exactly parallel and is *visual finger temporo-occipital aphasia*. In this defect we have two categories, *visual temporo-occipital finger aphasia* and *tactile parieto-occipital finger aphasia*. When other parts of the body are affected the part may be named accordingly.

Gustatory and olfactory aphasias are at present unknown.

Auditory aphasia consists in the loss of ability to associate the name with the sound of an object such as loss of ability to name a typewriter by hearing its sound. It practically always accompanies auditory verbal agnosia, only one case having been reported in which it occurred separately. *Auditory musical aphasia* if it ever occurs is extremely rare. In auditory aphasias the lesion is always in the temporal lobe.

Semantic aphasia is aphasia of significance. It does not come into play relative to objects because by the time one has recognized the object, and has named it, its significance is known. Thus if one recognizes the sound of a saw and is able to name the object the significance is complete. This is not true with reference to language symbols. One may see a certain word or phrase and be able to select from a given copy all similar words or phrases and still be totally unaware of their significance. Or one may hear and recognize a word (as shown by ability to repeat it) and still be totally unaware of its significance. Such defects are *semantic aphasias*. The visual semantic aphasias may be described as alexias but they are still aphasias.

We have *visual semantic external capsular aphasia* (loss of significance of written or printed matter through removal of the practic component), *visual semantic temporal aphasia* (loss of significance of written or printed matter due to a lesion between the angular gyrus and Wernicke's area or in Wernicke's area itself), *visual semantic third frontal aphasia* (loss of significance of written or printed matter from destruction of the practic component), *auditory semantic temporal verbal aphasia* (loss of ability to determine significance of spoken language though able to recognize it as spoken language), and *auditory semantic temporal musical aphasia* (loss of ability to determine significance of the music though able to recognize it, type of auditory amusia).

There is also *formulation aphasia* or inability to formulate language (the old paraphasia or jargon aphasia).

THE APRACTIC APHASIAS

Inasmuch as the types of aphasia which have been called "motor" are all forms of apraxia and as apraxia is only one form of motor disturbance the term apractic aphasia is far more exact than "motor aphasia." It is necessary to retain the two designations "apractic" and "aphasia" because some apraxias do not concern language at all.

The motor patterns located in the convolution of Broca are engrams of the memory of how to make certain sounds with the organs of speech. Disease of these engrams causes motor pattern apraxia (the type which Liepmann called kinetic

apraxia of the limb but which in this instance does not affect a limb). As the impulses from these cortical engrams must reach the precentral gyrus in order to produce speech, it is feasible for a lesion between Broca's convolution and the precentral gyrus to render speech impossible. It is also possible for a lesion of the anterior portion of the insula to extend deeply enough (a few millimeters to a centimeter into its substance) to give the same syndrome.

Beside these mechanisms which can render speech impossible, there is the mechanism of preventing formulation of language. Lesions at the temporal isthmus, by destroying the cross-road of association fibers, can so interfere with language reminiscence that the patient has nothing to say, i.e., he has no speech ideation. For this reason when the general physical examination indicates a severe posterior temporal lesion and the patient is also speechless, it is not necessary to assume an apraxia of speech. The patient must have ability to formulate his language before he can send language-composition impulses to the motor area and the motor area must not be blamed for failure to dispatch impulses which do not reach it.

The apractic aphasias are, then, *frontal apractic aphasia* (the old Broca's aphasia), *insular apractic aphasia, frontal triangularis apractic amusia* (lesion of pars triangularis, which contains engrams for praxia of music), *second frontal apractic agraphia* (due to lesion of Exner's writing center at the second frontal convolution), and *third frontal apractic agraphia* (the agraphia accompanying apractic aphasia).

APRAXIA

The physiologic basis of the apraxias has already been discussed. It has also been pointed out that the motor aphasias, some types of agraphia, some disturbances of conjugate lateral turning of the eyes, and some cases of dysarthria and dysphagia, etc., are types of apraxia. There is no difference between apractic amusia and musical apraxia, or between apractic aphasia and aphasic apraxia; the selection of the term in a given case of that sort depends on one's viewpoint at the moment of discussing the subject. One point, however, must remain clear, namely that agnostic apraxia is an impossibility because apraxia based on agnosia is a form of agnosia. There cannot be a sensory apraxia of any sort. But we may have the reverse, apractic agnosia which is an agnosia resulting from destruction of a motor pattern (an apraxia) which prevents the sensory pattern from functioning. In such cases the anatomic term will clarify the idea in each case.

The apraxias will therefore embrace: *apraxia* (for any particular act or in general), *cortical motor pattern apraxia, ideokinetic callosal apraxia, ideokinetic parietal apraxia, ideational apraxia.*

AGRAPHIA

A separate discussion of the terms in agraphia is necessary purely for co-ordination of the elements. This is because agraphia may be due to an irreminiscence, to ideokinetic apraxia, to cortical motor pattern apraxia, or to lesions of engrams which the individual in question has, by training, intimately associated with the function of writing. We have *irreminiscence angular agraphia, ideokinetic apractic parieto-occipital agraphia, ideokinetic callosal apractic agraphia, formulation agraphia, third frontal apractic agraphia, second frontal apractic agraphia,* and *external capsular agraphia* (the fibers conveying impulses from the angular gyrus to the frontal

lobe for writing coursing in the external capsule). It will be evident from this that the subject of agraphia is the ultimate in difficulty of cerebral localization on the basis of it alone. Isolated agraphia occasionally results from a lesion of the foot of the second frontal convolution and from a lesion of the gyri of Gratiolet (border between the angular gyrus and the occipital lobe). *Isolated agraphia,* however, is exceedingly rare.

ALEXIA

Alexia (a type of aphasia in the broad sense) must also be the subject of correlation because it may result from lesions in any one of a great variety of locations and may be agnostic or semantic and may result from loss of motor patterns which in a given case have been associated with the patient's system of reading. Alexia may affect letters, words, musical symbols, and numerals. We have *occipital agnostic verbal alexia, angular agnostic verbal alexia, angular agnostic musical alexia, angular agnostic literal alexia, parietal agnostic numeral alexia, temporal agnostic verbal alexia, temporal semantic alexia, agnostic external capsular alexia, semantic external capsular alexia, agnostic third frontal alexia, semantic third frontal alexia,* and *tactile parietal agnostic alexia.*

It will be clear from this presentation that one must analyze with great care the reason for alexia in a given case. The causative lesion may be located anywhere from the occipital to the frontal lobe and it may be due to loss of recognition or loss of knowledge of significance of the recognized written matter.

ACALCULIA

Acalculia, the loss of ability to calculate, is also aphasia since it is a loss of language function. It results from a lesion of the parieto-occipital area.

AMIMIA

Amimia has been used as a descriptive term to designate inability of a patient to mimic the movements and gestures of the examiner. Amimia depends on a loss of either comprehension of the act (based on agnosia or on a higher mental function akin to "simultanagnosia") or an apraxia. While the term amimia may be a useful shorthand term for such a loss, it in no way is conclusive of the site of the causative lesion. The reason for the amimia must be analyzed.

AMUSIA

Amusia has been extensively used to indicate loss of the musical functions. It may be used as a convenient designation but its causative elements should be appreciated. It is based on agnosia for the symbols, aphasia for their comprehension, or apraxia for the execution of vocal or instrumental music.

Proposed Nomenclature, Definitions, Old Terminology, and Location of Lesions

1. Acalculia.
   Loss of ability to calculate with or without agnosia or loss of revisualization of symbols.

O.T. Acalculia.
Focus. The lesion affects the posterior parietal or parieto-occipital region.

2. Agnosia, auditory, temporal (general).
Loss of power to recognize sounds heard.
O.T. Auditory or acoustic agnosia, psychic deafness, Wernicke's aphasia.
Focus. The lesion affects the posterior portion of the superior temporal convolution.

3. Agnosia, auditory, temporal, musical.
Loss of power to recognize music as music by sound alone.
O.T. Musical agnosia, *psychic amusia.*
Focus. The lesion affects the anterior extremity of the temporal lobe.

4. Agnosia, auditory, temporal, verbal.
Loss of power to recognize words heard.
O.T. Auditory or acoustic verbal agnosia, psychic deafness, Wernicke's aphasia, subcortical acoustic verbal agnosia.
Focus. The lesion affects the posterior superior temporal convolution.

5. Agnosia, tactile, occipital.
Loss of power to recognize objects by the sense of touch alone even with the use of both hands. The loss is based on loss of revisualization (loss of visual reminiscence) and because of this loss the patient cannot form a visual image of the object by touch alone. The condition occurs in those particularly visual-minded.
O.T. Apperceptive blindness of the senile of Pick, ideational agnosia of Liepmann.
Focus. The lesion affects area 19 of Brodmann in its convex portion.

6. Agnosia, tactile, parietal.
Loss of power to recognize by touch alone objects which are recognized by the other senses, or in the other hand, as this agnosia is unilateral.
O.T. Astereognosis, tactile agnosia.
Focus. The lesion affects the parietal lobe and may be cortical or subcortical.

7. Agnosia, tactile, parieto-occipital.
Definition is identical with that of occipital tactile agnosia (5).
O.T. Apperceptive blindness of the senile of Pick, ideational agnosia of Liepmann.
Focus. The lesion affects area 19 of Brodmann and the posterior portion of the parietal lobe.

8. Agnosia, tactile, parieto-occipital, finger.
Loss of power to recognize one's fingers by the sense of touch alone.
O.T. Finger agnosia.
Focus. The lesion affects the convolutions of Gratiolet.

9. Agnosia, visual, angular, literal.
Loss of power to recognize letters by vision alone, due to a lesion of the angular gyrus.

O.T. Visual literal agnosia, literal alexia, part of Wernicke's aphasia.
Focus. The lesion affects the angular gyrus.

10. Agnosia, visual, angular, verbal.
Loss of power to recognize written or printed words by vision alone (embossed letters may still be read) due to a lesion of the angular gyrus.
O.T. Visual verbal agnosia, alexia, part of Wernicke's aphasia.
Focus. The lesion affects the angular gyrus.

11. Agnosia, visual, angular, musical.
Loss of power to recognize musical notes by vision alone, due to a lesion of the angular gyrus.
O.T. Visual agnosia for musical symbols, visual amusia.
Focus. The lesion affects the angular gyrus.

12. Agnosia, visual, frontal, literal.
Loss of power to recognize letters by sight alone, due to a lesion of the frontal lobe. Through destruction of Broca's convolution or of the frontal writing center at the foot of the second frontal convolution the motor associations (which the patient has maintained since childhood and which to him are necessary for visual recognition) and even visual recognition have become impossible. This is a rare occurrence.
O.T. Visual literal agnosia, literal alexia, part of Wernicke's aphasia.
Focus. The lesion affects the foot of the second or third frontal convolutions. That the lesion is not in the angular gyrus is determined by the presence in the case of apractic aphasia or other apraxia, especially cortical motor pattern apraxia.

13. Agnosia, visual, frontal, verbal.
Definition is identical with (12) except that words alone and not letters are effected.
O.T. Visual verbal agnosia, element of Wernicke's aphasia, alexia.
Focus. The lesion affects the foot of the second or third frontal convolutions. The explanation is identical with that given in (12). The reason that recognition of letters is preserved is that they are stored as engrams of objects in area 18 of Brodmann in many instances.

14. Agnosia, visual, insular, literal.
Loss of power to recognize letters by sight alone due to a lesion of the insula or pathways immediately subcortical to it. By destruction of tracts, which in such a case are necessary for association of the convolution of Broca or of the foot of the second frontal convolution with the angular gyrus, the angular gyrus cannot function alone in recognition of letters. This is a rare occurrence and is based on retained childhood associations.
O.T. Visual literal agnosia, alexia, part of Wernicke's aphasia.
Focus. The lesion affects the insula and probably of necessity the external capsule through which the association fibers go.

15. Agnosia, visual, insular, verbal.
The definition is identical with that of (14) except that the loss affects words and not letters.

O.T. Visual verbal agnosia, alexia, part of Wernicke's aphasia.
Focus. The explanation of the syndrome and its rarity are identical with those given under (14).

16. Agnosia, visual, occipital (general). This includes visual agnosia for animate and inanimate objects.
Loss of power to recognize objects or symbols by vision alone in spite of adequate visual perception (and with sufficient mental capacity in general) due to a lesion of the occipital lobe. The visual agnosias for symbols are called agnosias rather than aphasias because the term agnosia is more specific.
O.T. Visual agnosia (qualified), psychic blindness, mind blindness.
Focus. The lesion affects area 18 of Brodmann in its convex portion.

17. Agnosia, visual, occipital, musical.
Loss of power to recognize musical symbols by vision alone due to a lesion of the occipital lobe. There is nearly always, if not always, an associated homonymous hemianopia.
O.T. Visual musical agnosia, subcortical musical alexia.
Focus. The focus is subcortical (and perhaps also cortical) in the occipital lobe. The agnosia is caused by the fact that impulses from both areae striatae fail to reach the angular gyrus for recognition.

18. Agnosia, visual, occipital, literal.
The definition is identical with that given in (17) except that letters instead of musical notes are affected.
O.T. Literal agnosia, subcortical literal agnosia, literal alexia.
Focus. The focus and explanation are identical with that given in (17).

19. Agnosia, visual, occipital, numeral.
Loss of power to recognize mathematical symbols due to a lesion of the occipital lobe. There is usually an associated homonymous hemianopia.
O.T. Visual numeral agnosia, numeral alexia, psychic blindness.
Focus. The focus is in the occipital lobe so placed as to prevent impulses from reaching the borders of the interparietal sulcus from both areae striatae.

20. Agnosia, visual, occipital, verbal.
The definition is identical with that given under (17) except that words instead of musical notes are affected.
O.T. Pure visual verbal agnosia, pure word blindness.
Focus. The lesion is in the occipital lobe, subcortical (and often cortical). It prevents recognition of words by interrupting impulses from both areae striatae to the angular gyrus. It does not cause agraphia because the angular gyrus itself is spared. That is the basis for the old terminology "pure."

21. Agnosia, visual, parietal, numeral.
Loss of power to recognize mathematical symbols due to a lesion of the parietal lobe, either at the borders of the interparietal sulcus or subcortical to that area.
O.T. Visual agnosia for mathematical figures, figure agnosia.
Focus. The lesion is cortical on the borders of the interparietal sulcus or subcortical to that area.

22. Agnosia, visual, parieto-occipital, finger.
Loss of power to recognize fingers by vision alone, those of the patient or those of the examiner or both.
O.T. Finger agnosia.
Focus. The lesion affects the convolutions of Gratiolet.

23. Agnosia, visual, subcortical, angular, musical.
Loss of power to recognize musical symbols due to a subcortical lesion of the angular gyrus. There is usually an associated homonymous hemianopia.
O.T. Musical agnosia, visual agnosia for musical notes.
Focus. The lesion is subcortical to the angular gyrus and prevents recognition by interrupting impulses from both areae striatae to the angular gyrus. There is no agraphia for musical notes because the cortex of the angular gyrus is not affected.

24. Agnosia, visual, subcortical, angular, verbal.
The definition is identical with that of (23) except that words instead of musical notes are affected.
O.T. Pure visual verbal agnosia, alexia, subcortical visual verbal agnosia.
Focus. The lesion affects the subcortical region of the angular gyrus. There is no agraphia because the cortex is spared.

25. Agnosia, visual, temporal, literal (and verbal).
Loss of power to recognize letters and words due to a lesion of Wernicke's area or the small space between Wernicke's area and the angular gyrus.
O.T. Visual literal (and verbal) agnosia, literal (and verbal) alexia, Wernicke's aphasia.
Focus. The lesion either separates Wernicke's area in the posterior superior temporal convolution from the angular gyrus or affects Wernicke's area itself. It causes visual agnosia because in the great majority of cases the angular gyrus cannot function for recognition of symbols without association with Wernicke's area. This is because visual engrams are laid down in close association with auditory ones.

26. Agraphia.
A descriptive term when not qualified, meaning loss of the power to write.
O.T. Agraphia.
Focus. On the basis of agraphia alone it is usually impossible to make a focal diagnosis. See agraphia, isolated.

27. Agraphia, apractic, second frontal.
Loss of power to write due to a lesion of the writing center at the foot of the second frontal convolution. The patient usually is unable to write by any method, even by building words with blocks or by writing on a typewriter.
O.T. Motor agraphia, isolated agraphia.
Focus. The lesion is in the cortex of the foot of the second frontal convolution.

28. Agraphia, apractic, third frontal.
Loss of the power to write due to a lesion of Broca's convolution. There is an associated apractic aphasia.

O.T. Broca's aphasia, motor agraphia.
Focus. Broca's convolution is affected either cortically or subcortically. The agraphia results secondarily because most patients are unable to write unless they have the motor patterns for speech associated with their writing mechanism.

29. Agraphia, external capsular.
Loss of the capacity to write due to a lesion of the external capsule.
O.T. Motor agraphia.
Focus. The lesion affects the external capsule and causes agraphia because the impulses from the angular gyrus (where reminiscence of words occurs) fail to reach the writing center at the foot of the second frontal convolution.

30. Agraphia, formulation, temporal.
Loss of ability to write because of inability to formulate language, all due to a lesion of the temporal lobe.
O.T. Amnesic agraphia, paragraphia.
Focus. The lesion affects the posterior part of the temporal lobe. It may be small or large. It interferes with the function of the language formulation area.

31. Agraphia, ideokinetic apractic, callosal.
Loss of ability to write because of a lesion of the corpus callosum which causes ideokinetic apraxia.
O.T. Apractic agraphia.
Focus. The lesion is in the corpus callosum, usually in the anterior portion. Unless the lesion is a slowly progressive one the patient usually recovers ability to write.

32. Agraphia, ideokinetic apractic, parieto-occipital.
O.T. Apractic agraphia.
Focus. The lesion affects most often the subcortical structures of the general region of the supramarginal gyrus.

33. Agraphia, irreminiscence literal (or verbal) angular.
Loss of ability to write due to loss of ability to recall the appearance of letters or words, and caused by a lesion of the angular gyrus.
O.T. Agraphia.
Focus. The lesion affects the cortex of the angular gyrus.

34. Agraphia, isolated, parieto-occipital.
Loss of ability to write due to loss of knowledge of visual or visual memory guidance of hand movements and due to a lesion of the convolutions of Gratiolet.
O.T. Isolated agraphia (it being the only aphasic element in the case) as part of Gerstmann's syndrome.
Focus. The lesion affects the convolutions of Gratiolet at the border between the angular gyrus and the occipital convolutions.

35. Agraphia, isolated, second frontal, apractic.
Loss of ability to write due to cortical motor apraxia for writing only and due to a lesion at the foot of the second frontal convolution.
O.T. Motor agraphia, isolated agraphia.

Focus. The lesion affects the cortex at the foot of the second frontal convolution.

36. Agraphia, temporal.
Loss of ability to write due to crippling of the angular gyrus through destruction of its associative connection with Wernicke's area and caused by a lesion of the superior temporal convolution between the angular gyrus and Wernicke's area.
O.T. Agraphia accompanying Wernicke's aphasia.
Focus. The lesion affects the small area of cortex between the area of Wernicke and the angular gyrus (part of area 22 of Brodmann).

37. Alexia.
Descriptive term signifying loss of ability to read without reference to the physiologic cause except that it is due to a focal cerebral lesion. Sufficient visual perception and mental capacity are presupposed.
O.T. Alexia.

38. Alexia, agnostic, external capsular, literal and verbal.
Loss of ability to recognize words and letters due to a lesion of the external capsule and based physiologically on dissociation of the angular gyrus from Broca's convolution. This is a rare occurrence and is found only in persons who have retained childhood associations between vocal reading and visual reading.
O.T. Alexia, visual, verbal or literal alexia.
Focus. The lesion affects the external capsule.

39. Alexia, agnostic, literal and verbal angular.
Loss of ability to recognize letters and words due to a lesion of the angular gyrus.
O.T. Agnostic alexia, alexia, visual literal and verbal agnosia.
Focus. The lesion affects the cortex of the angular gyrus.

40. Alexia, agnostic, musical, angular.
Definition and physiologico-anatomic basis is identical with (39) except that musical symbols are affected instead of letters and words.
O.T. Musical alexia, visual musical agnosia.
Focus. The lesion affects the cortex of the angular gyrus.

41. Alexia, agnostic, numeral, parietal.
Loss of ability to recognize mathematical figures due to a lesion of the parietal lobe (borders of the interparietal sulcus).
O.T. Visual agnosia for mathematical figures, mathematical alexia.
Focus. The lesion affects the cortex of the parietal lobe at the borders of the interparietal sulcus.

42. Alexia, agnostic, third frontal, literal and verbal.
Loss of ability to recognize letters and words due to a lesion of Broca's convolution. This is a rare occurrence and is found only in those who have retained childhood associations between the angular gyrus and Broca's convolution as a necessity for recognition.

O.T. Visual verbal agnosia, alexia.
Focus. The lesion affects the cortex of Broca's convolution.

43. Alexia, agnostic, verbal, occipital.
Loss of ability to recognize words due to a lesion of the occipital lobe so placed that impulses from both areae striatae fail to reach the angular gyrus.
O.T. Pure visual verbal agnosia.
Focus. The lesion is in the occipital lobe and if unilateral affects also the splenium of the corpus callosum. There is nearly always an associated homonymous hemianopia.

44. Alexia, agnostic, verbal, temporal.
Loss of ability to recognize written or printed words due to a lesion of the temporal lobe. While the angular gyrus functions in recognition, in most cases it cannot function without association with Wernicke's area.
O.T. Visual verbal agnosia, verbal alexia. Part of Wernicke's aphasia.
Focus. The lesion affects the cortex of Wernicke's area itself or separates Wernicke's area from the angular gyrus.

45. Alexia, semantic, external capsular.
Loss of ability to determine significance of words, which are recognized, due to a lesion of the external capsule. In rare cases a lesion in this location even prevents recognition but usually interferes, if at all, with reading, only in determination of significance.
O.T. Semantic alexia, transcortical sensory aphasia.
Focus. The lesion affects the external capsule, usually also the insula.

46. Alexia, semantic, temporal.
Loss of ability to determine significance of words, which are recognized, due to a lesion of the temporal lobe. The physiologic basis is the requirement of the angular gyrus, in most cases, to have association with Wernicke's area in order to determine significance of what is read.
O.T. Semantic alexia, transcortical sensory aphasia.
Focus. The lesion affects the cortex of Wernicke's area or separates Wernicke's area from the angular gyrus.

47. Alexia, semantic, third frontal.
Loss of ability to determine the significance of words, which are recognized, due to a lesion of Broca's convolution. This form of alexia in a minor degree is common.
O.T. Semantic alexia, transcortical sensory aphasia.
Focus. The lesion affects Broca's convolution either cortically or subcortically.

48. Alexia, tactile, agnostic, parietal.
Loss of ability to read letters or words by touch, sufficient tactile perception presupposed, due to a lesion of the parietal lobes, cortical or subcortical.
O.T. Tactile aphasia, tactile agnosia for letters, astereognosis.
Focus. The lesion affects the parietal lobe opposite to the affected hand. It may be cortical or subcortical.

49. Amimia.
A purely descriptive term used to designate loss of ability to mimic gestures.

As amimia may be based on agnosia or on apraxia, the cause must be analyzed in each instance.
O.T. Amimia.

50. Amusia.
A purely descriptive term used to designate a disturbance of the musical sense, auditory, visual, or motor. Its subdivisions are classified as agnosias, apraxias, and aphasias.
O.T. Amusia.

51. Aphasia.
A term restricted to mean "loss of language association with." It includes agnosias and apraxias which are concerned with language. Thus "finger aphasia" means loss of the ability to name or comprehend the names of the fingers. Visual object aphasia means loss of ability to name objects seen, etc. Aphasias based on apraxia are apractic aphasias. The term is therefore narrower than the old expansive definition.

52. Aphasia, apractic, insular.
Loss of ability to speak based on apraxia and due to a lesion of the insula (probably chiefly the subcortical structures, the external capsule in particular).
O.T. Subcortical or association motor aphasia, anarthria of Pierre Marie.
Focus. The lesion affects the insula probably always subcortically.

53. Aphasia, apractic, frontal.
Loss of ability to speak due to a lesion of Broca's convolution cortical or subcortical.
O.T. Broca's aphasia, subcortical motor aphasia, transcortical motor aphasia.
Focus. The lesion affects Broca's convolution, cortically or subcortically.

54. Aphasia, apractic, musical, triangularis.
Loss of ability to sing words due to a lesion of the pars triangularis of the third frontal convolution.
O.T. Avocalia, motor amusia.
Focus. The lesion affects the pars triangularis of the third frontal convolution.

55. Aphasia, auditory, musical, semantic, temporal.
Loss of ability to comprehend played or sung music, which is recognized, due to a lesion of the temporal lobe. This is rare because of the facility with which the minor side assumes the function.
O.T. Auditory amusia.
Focus. The lesion affects the anterior portion of the temporal lobe.

56. Aphasia, auditory, object, temporal.
Loss of ability to associate the name of an audible object with the sound of the object, e.g. the patient cannot say "bell" when he hears the ring or "watch" when he hears the tick.
O.T. Amnesia aphasia.
Focus. The lesion affects the posterior part of the temporal lobe.

57. Aphasia, auditory, semantic, temporal.
Loss of ability to comprehend names of sounds in general in spite of recogni-

tion of the sounds, the disability being due to a temporal lesion.
O.T. Amnesic aphasia.
Focus. The lesion affects the temporal lobe.

58. Aphasia, color, occipital.
Loss of ability to name colors which are seen and recognized, due to an occipital lesion. The lesion breaks the association between the occipital area for recognition and the language formulation area.
O.T. Amnesic color blindness, optic aphasia for colors, visual aphasia for colors.
Focus. The lesion affects the occipital lobe close to the temporal lobe.

59. Aphasia, color, temporal.
This is identical with (58) above except that the lesion is in the temporal lobe close to the occipital.

60. Aphasia, formulation, temporal.
Loss of ability to formulate words and phrases into language.
O.T. Amnesic aphasia, paraphasia, agrammatism.
Focus. The lesion affects the posterior portion of the temporal lobe, chiefly area 37 and the posterior extremity of area 22 of Brodmann.

61. Aphasia, visual, semantic, external capsular.
Loss of comprehension of significance of written or printed words due to a lesion of the external capsule. This form of aphasia is based on the necessity of having association between the angular gyrus and Broca's convolution for full comprehension of written or printed words.
O.T. Semantic aphasia, semantic alexia.
Focus. The lesion affects the external capsule.

62. Aphasia, visual, semantic, temporal.
Loss of comprehension of significance of written or printed words due to a lesion of the temporal lobe. This is based on the dependence of the angular gyrus in association with Wernicke's area for comprehension of written language.
O.T. Transcortical sensory aphasia, semantic alexia, part of Wernicke's aphasia, Wernicke's aphasia.
Focus. The lesion affects Wernicke's area or the short portion of cortex between it and the angular gyrus (posterior extremity of area 22 of Brodmann).

63. Aphasia, visual, semantic, third frontal.
Loss of ability to comprehend significance of written or printed matter due to a lesion of Broca's convolution. This is based on the physiologic necessity for association of the angular gyrus with Broca's convolution for full comprehension of written language.
O.T. Semantic aphasia, semantic alexia.
Focus. The lesion affects Broca's convolution.

64. Aphasia, tactile, finger, parietal.
Loss of ability to name or comprehend names of fingers held by the examiner due to a lesion of the parietal lobe.

O.T. Amnesic finger aphasia, finger aphasia.
Focus. The lesion affects the parietal lobe usually in the region of the supramarginal gyrus. It interrupts the association between the parietal lobe and Wernicke's area.

65. Aphasia, visual, finger, temporo-occipital.
Loss of ability to name or comprehend the names of fingers, seen by the patient, due to a lesion of the temporo-occipital area.
O.T. Finger aphasia, visual finger aphasia, visual agnostic element of finger agnosia, amnesic aphasia for fingers.
Focus. The lesion affects the temporo-occipital area and interrupts association between the occipital area for recognition (area 18) and Wernicke's area.

66. Aphasia, visual, temporal, object.
Loss of ability to name objects seen, though they are recognized, due to a lesion of the temporal lobe.
O.T. Amnesic aphasia, anomia.
Focus. The lesion affects area 37 of Brodmann or the posterior extremity of area 22, in general the posterior portion of the temporal lobe. It interrupts communication between area 18 and Wernicke's area for which reason the auditory memories of the names cannot be associated with the memory of the object seen.

67. Apraxia (for any particular movement, act, idea, or apraxia in general).
The term is only descriptive until qualified. Apraxia is loss of ability to perform as desired or as requested, through loss of memory of how to perform. This loss of memory in turn is due to destruction of cerebral engrams. Ataxia, paralysis, and dementia must always be excluded as causes.

68. Apraxia, cortical motor pattern.
Loss of ability to execute a *movement* due to lesion of the cortical motor pattern. This type of apraxia is illustrated by Broca's aphasia, by agraphia as a result of a lesion in the foot of the second frontal convolution, by aphasia from a lesion of the precentral gyrus at the frontal operculum, or by apraxia of singing from a lesion of the pars triangularis of the third frontal convolution. The result is the same whether the cortical motor pattern itself is destroyed or whether its connections with the Betz cells, through which the projection produce the movements, are broken.
O.T. Kinetic apraxia of the limb, apraxia of lateral gaze, Broca's aphasia, subcortical motor aphasia, transcortical motor aphasia, apractic amusia, apractic agraphia, apractic aphasia, avocalia.
Focus. The lesion for each type is cortical if it destroys the motor pattern or subcortical if it prevents the organized plan of the movement from reaching the Betz cells where the projection fibers begin for execution of the movement.

69. Apraxia, ideational.
Loss of ability to carry out an idea due to a diffuse lesion of the brain affecting the ideational plan of action. The individual acts constituting the ideational plan can be executed without trouble but the entire ideational plan cannot be retained in consciousness long enough to be executed.

O.T. Ideational apraxia.
Focus. There is no single focus. The "lesion" is either a toxemia or is diffusely placed such as in multiple vascular softening, or multiple tumors.

70. Apraxia, ideokinetic, callosal.
Loss of ability to execute the plan of an act, the plan and the mechanism of execution both being undisturbed but the connection between them being broken, by a lesion of the corpus callosum. The loss may affect the fingers of the patient only.
O.T. Ideokinetic apraxia. Apraxia of finger demonstration, finger agnosia.
Focus. The lesion is in the corpus callosum, anterior for the face and upper limbs, posterior for the lower limbs, intermediate for the trunk. The apraxia does not remain unless the lesion is progressive.

71. Apraxia, ideokinetic, parietal.
This condition is identical with (70) except that the lesion affects the subcortical structures of the parietal lobe, most potently at the supramarginal gyrus. When it affects the minor limb it is the old sympathetic apraxia.

72. Autotopagnosia, visual, occipital.
Loss of ability to recognize parts of the body. It is essentially visual agnosia for the body and hence is part of general visual agnosia.

73. Irreminiscence.
A term coined to designate a disturbance of ability to recall, i.e. a disturbance of power of reminiscence. It is only descriptive unless qualified.
O.T. Revisualization, re-auditorization, loss of power to recall, amnesia of certain types.

74. Irreminiscence, auditory, musical, temporal.
Loss of power to recall into auditory memory musical compositions formerly known, due to a lesion of the temporal lobe.
O.T. Loss of musical recall.
Focus. The lesion affects the anterior end of the temporal lobe. (Irritation of the same area causes auditory musical hallucinations.)

75. Irreminiscence, auditory (general) temporal.
Loss of power to recall auditory memories in general due to a lesion of the temporal lobe.
O.T. Loss of reauditorization, loss of recall of auditory memories.
Focus. The lesion is a large one in the temporal lobe.

76. Irreminiscence, auditory, verbal, temporal.
Loss of power to recall the auditory memory of words due to a lesion of the temporal lobe. (Irritation of this area causes hallucinations of words.)
O.T. Amnesia aphasia, loss of recall of auditory memory of words.
Focus. The lesion affects the temporal lobe, chiefly area 37 of Brodmann. It is the underlying factor in some cases of disturbance of language formulation.

77. Irreminiscence, color, occipital.
Loss of ability to recall visual images of colors. In some instances the patients recall all colors as being black. (Irritation of this area causes hallucinations of colors.)

O.T. Loss of revisualization of colors.
Focus. The lesion affects the occipital lobe, area 19 of Brodmann, especially the lateral convex surface, cortical or subcortical.

78. Irreminiscence, direction, occipital.
Loss of ability to recall directions, either the cardinal ones or as related to the body of the individual, due to a lesion of the occipital lobe.
O.T. Geometric-optic agnosia, disorientation in space.
Focus. The lesion affects the occipital lobe, chiefly area 19 of Brodmann on the inferior surface.

79. Irreminiscence, distance, occipital.
This condition is identical with (78) except that distance instead of direction, is affected.

80. Irreminiscence, visual, body, parieto-occipital.
Loss of ability to revisualize the body of the patient or of another, due to a lesion of the parieto-occipital lobe. (Irritation of this area causes visual hallucinations of living things.)
O.T. Disturbance of the body scheme, autotopagnosia.
Focus. The lesion affects the parieto-occipital region, either cortically or subcortically, usually within a centimeter of the mesial surface (area 19 of Brodmann).

81. Irreminiscence, visual, finger, parieto-occipital.
Loss of ability to recall visually one's own fingers or those of another due to a lesion of the parieto-occipital area.
O.T. Finger agnosia, visual agnostic element of finger agnosia, disturbance of the body scheme, part of the Gerstmann syndrome.
Focus. The lesion affects the border between the angular gyrus and the occipital lobe, the convolutions of Gratiolet.

82. Irreminiscence, visual, object, occipital.
Loss of power to recall visual images of objects due to an occipital lesion (irritation of this area causes visual hallucinations of objects).
O.T. Loss of revisualization of objects, disorientation in space, acoustic-optic agnosia.
Focus. The lesion affects area 19 of Brodmann, chiefly the inferior convex portion, cortically or subcortically.

83. Irreminiscence, visual, literal, angular.
Loss of ability to recall visual images of letters due to a lesion of the angular gyrus (cortex). This loss causes agraphia.
O.T. Loss of revisualization of letters.
Focus. The lesion affects the cortex of the angular gyrus.

84. Irreminiscence, visual, musical, angular.
Loss of power to recall appearance of musical notes or musical composition due to a lesion of the angular gyrus.
O.T. Loss of revisualization of musical symbols.
Focus. The lesion affects the angular gyrus.

85. Irreminiscence, visual, numeral, parietal.
Loss of ability to recall visual images of mathematical figures due to a lesion of the borders of the interparietal sulcus.
O.T. Loss of revisualization of mathematical figures.
Focus. The lesion affects the borders of the interparietal sulcus.

86. Irreminiscence, visual, verbal, angular.
Loss of power to revisualize written or printed words, due to a lesion of the angular gyrus. This causes agraphia.
O.T. Loss of revisualization of written or printed words, sensory agraphia.
Focus. The lesion affects the cortex of the angular gyrus.

87. Irreminiscence, visual, verbal, temporal.
Loss of ability to revisualize words, due to a lesion of the temporal lobe. This causes agraphia.
O.T. Loss of revisualization of written or printed words, sensory agraphia.
Focus. The lesion affects the portion of cortex between Wernicke's area and the angular gyrus (posterior extremity of area 22 of Brodmann). The syndrome is based physiologically on the dependence of the angular gyrus on the area of Wernicke in the function of revisualization of words.

CLASSIC NOMENCLATURE WITH PROPOSED NOMENCLATURE WHICH IS EQUIVALENT TO, IS EMBRACED BY, OR EMBRACES THE CLASSIC

(O.T., Old Terminology. P.N., Proposed Nomenclature)

O.T. Acalculia.
P.N. Acalculia.

O.T. Agnosia. The term according to some authors included loss of recognition through several sense organs simultaneously.
P.N. Agnosia. The term is now limited to loss of recognition through one sense organ only.

O.T. Agnosia, acoustic or auditory.
P.N. Agnosia, auditory, temporal, (general).

O.T. Agnosia, acoustic or auditory, musical.
P.N. Agnosia, auditory, temporal, musical.

O.T. Agnosia, acoustic or auditory, verbal.
P.N. Agnosia, auditory, temporal, verbal.

O.T. Agnosia, disjunctive (agnosia, ideational).
P.N. In the new light this is interpreted as a combination of visual and tactile agnosia in various combinations.

O.T. Agnosia, dissolutive (agnosia, ideational).
P.N. This is somewhat similar to disjunctive agnosia and is interpreted as a combination of agnosias.

O.T. Agnosia, finger, of Gerstmann.
P.N. This is subdivided into visual agnosia for fingers, tactile agnosia for fingers, and ideokinetic apraxia of fingers.

O.T. Agnosia, geometric-optic.
P.N. This is a part of visual agnosia for objects, geometrical relations being part of objects small or large. That which formerly was considered geometric-optic agnosia is also in part visual object irreminiscence.

O.T. Agnosia, gustatory.
P.N. Gustatory agnosia whenever this may become established as a recognizable fact.

O.T. Agnosia, ideational.
P.N. Various combinations of visual plus tactile agnosia.

O.T. Agnosia, olfactory.
P.N. Olfactory agnosia whenever this may become established as a recog nizable fact.

O.T. Agnosia, subcortical acoustic or auditory verbal.
P.N. Auditory verbal agnosia in which there happens to be good function of the minor cerebral hemisphere for formulation of language and for production of speech.

O.T. Agnosia, subcortical, visual, verbal.
P.N. Agnosia, visual, subcortical, angular, verbal.

O.T. Agnosia, tactile (astereognosis).
P.N. Agnosia, tactile, parietal.

O.T. Agnosia, visual (or optic).
P.N. Agnosia, visual, occipital (general).

O.T. Agnosia, visual for letters.
P.N. Agnosia, visual, occipital literal; or agnosia, visual subcortical or cortical angular literal; agnosia visual temporal literal, etc.

O.T. Agnosia, visual for mathematical figures.
P.N. Agnosia visual parietal, or parieto-occipital numeral.

O.T. Agnosia, visual, for musical notes.
P.N. Agnosia, visual occipital (or angular, or subcortical angular) musical.

O.T. Agnosia, visual, for objects or pictures.
P.N. Agnosia, visual occipital (general).

O.T. Agnosia, visual, verbal.
P.N. Agnosia, visual, angular, verbal.

O.T. Agrammatism.
P.N. Aphasia, formulation, temporal.

O.T. Agraphia.
P.N. Agraphia analyzed as to cause and site of lesion. See each heading beginning with "Agraphia."

O.T. Alexia.
P.N. Alexia analyzed as to cause and site. See each heading beginning with "Alexia."

O.T. Amimia.
P.N. Amimia as a descriptive term, but it has little significance until analyzed into terms of agnosia and apraxia.

O.T. Amusia.
P.N. Amusia. The term is to be analyzed in each case as to agnostic, apractic, or aphasic basis. Otherwise, it is purely descriptive.

O.T. Anomia.
P.N. Aphasia, visual, temporal, object.

O.T. Aphasia.
P.N. Aphasia. However, the term is restricted and redefined to mean "the language component of."

O.T. Aphasia, acoustic-optic.
P.N. Irreminiscence, visual, object, occipital.

O.T. Aphasia, amnesic.
P.N. Aphasia, visual, temporal, object; or Aphasia, auditory, temporal, object; or Aphasia, formulation.

O.T. Aphasia, Broca's.
P.N. Aphasia, apractic, third frontal.

O.T. Aphasia, jargon.
P.N. Aphasia, formulation.

O.T. Aphasia, motor.
P.N. Aphasia, apractic (with various anatomical locations of the causative lesions).

O.T. Aphasia, optic.
P.N. Aphasia, visual, temporal, object.

O.T. Aphasia, optic for colors.
P.N. Aphasia, color (temporal or occipital).

O.T. Aphasia, semantic.
P.N. Aphasia, semantic (qualified as to type and anatomic site of causative lesion).

O.T. Aphasia, sensory.
P.N. The term is too loose to have much significance and should be analyzed into its specific components.

O.T. Aphasia, subcortical motor.
P.N. Aphasia, apractic (qualified as to anatomical site of causative lesion).

O.T. Aphasia, transcortical motor.
P.N. Aphasia, apractic (qualified as to site of anatomical site of causative lesion). Its occurrence is based on the appearance of cases in which the minor hemisphere has excellent repetitive capacity.

O.T. Aphasia, Wernicke's.
P.N. As this is a complex syndrome dependent on agnosia, irreminiscence and aphasia the elements should be analyzed in each case.

O.T. Apraxia.
P.N. Apraxia. The defect should be analyzed in each case as to movement, act, and idea.

O.T. Apraxia, constructive (of Kleist).
P.N. This is based on visual irreminiscence of finger movements.

O.T. Apraxia, ideational.
P.N. Ideational apraxia.

O.T. Apraxia, ideokinetic.
P.N. Ideokinetic apraxia.

O.T. Apraxia, kinetic of limb.
P.N. Cortical motor pattern apraxia.

O.T. Apraxia, sympathetic.
P.N. Ideokinetic apraxia affecting the minor limb.

O.T. Astereognosis.
P.N. Tactile agnosia.

O.T. Autotopagnosia.
P.N. Visual autotopagnosia.

O.T. Avocalia.
P.N. Aphasia, apractic musical, triangularis.

O.T. Blindness, amnestic color of Wilbrand.
P.N. Aphasia, visual, temporal, object.

O.T. Blindness, apperceptive of the senile.
P.N. Simultaneous visual and tactile agnosia.

O.T. Blindness, cerebral, color, acquired.
P.N. Agnosia, visual color, occipital.

O.T. Blindness, psychic.
P.N. Agnosia, visual, occipital (general).

O.T. Charcot-Wilbrand syndrome.
P.N. Agnosia and irreminiscence visual, occipital.

O.T. Deafness, psychic.
P.N. Auditory agnosia and auditory semantic aphasia.

O.T. Deafness, verbal.
P.N. Auditory verbal agnosia.

O.T. Disorientation, optical, in space.
P.N. Irreminiscence, object, occipital.

O.T. Imperception (Hughlings Jackson).
P.N. Agnosia, visual, object, occipital.

O.T. Paraphasia.
P.N. Aphasia, formulation, temporal.

O.T. Simultanagnosia (Wolpert).
P.N. A complex disturbance of synthesis of visual impressions.

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