THE GENESIS AND DISSOLUTION

OF

THE FACULTY OF SPEECH

A CLINICAL AND PSYCHOLOGICAL STUDY OF APHASIA

BY

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TO MY TEACHER

CHARLES L. DANA, M.D.

AND TO MY FRIEND

LIGHTNER WITMER, Ph.D.

TO WHOM I OWE MY INTELLECTUAL AWAKEMENT
THIS LABOR OF A YEAR'S LEISURE IS

AFFECTIONATELY DEDICATED

PREFATORY NOTE.

The MS. of this monograph left the writer's hand in April, 1897. Since that time several important contributions have been made to our knowledge of aphasia. The author regrets that time and opportunity have not been granted him to give them the consideration and discussion they merit. Only verbal changes have been made in the text since its completion, but here and there a footnote has been added to call attention to some of the more important facts and striking claims set forth by recent writers.

47 West Thirty-Eighth Street, New York, Christmas, 1897.

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THE FACULTY OF SPEECH.

CHAPTER I.

Disorders of Intellectual Expression, Known as Aphasia.

Introduction.

The possession of the faculty of speech distinguishes the human being from even those animals which stand next to him in biological relationship. Man is in consequence inclined to reflect upon its possession, and to speculate concerning its nature and origin. In days when philosophic speculation addressed itself principally to *a-priori* assumptions of the nature and interrelations of unanalyzed mental phenomena, the function of speech, and that, too, in its fully developed form, was regarded as an essential attribute or inherent faculty of the human mind, manifesting itself with the perfected completeness of a Minerva springing from the brows of Jove. From the vagueness, often the vagaries, of such speculation, there was little relief until attention was directed to the development of speech in the infant and to its dissolution from disease. Though of recent development, labor in these two fields of observation has extended our knowledge of the genesis of speech and thereby given a basis for the interpretation of its significance. The genetic method

has rendered relatively a small service compared with the pathological method. In this judgment I am not unmindful of the value of many painstaking observations, such as those of Preyer and of Baldwin, that have aided in determining the antecedent factors of voluntary articulation and other forms of mental expression; but it has been chiefly from the study of those disorders of speech included under the term aphasia, made by many students of human language, physicians, physiologists, and psychologists, that real knowledge of the faculty of speech has been acquired.

In introducing the term aphasia, Trousseau first applied it to the condition in which there was inability to express thought in words; in this narrow sense it was used for some time. When the part played by language in the communication of ideas began to be studied, it was not long before it became apparent that this application of the term was wholly inadequate; and that a word was needed not only to connote restrictedly an inability to create phonetic symbols, but to include as well the inability to express ideas or to manifest states of consciousness by signs of all sorts, of which speech occupied only the first rank in importance. Disturbance of the power to express a condition of mind manifests itself in speech, writing, pantomime, gesture, drawing, instrumentation, symbols, colors, attitudes, etc. To this end the term asymbolia was suggested by Finkenburg. It was improved upon by Kussmaul, who substituted the word asemia, which admitted easy paronymization and the adjective asemic, and which signified literally the meaning that was desired. Nevertheless, the term aphasia, sanctioned by time and

consecrated by usage, has not been supplaced by these technically better constructed terms. On the contrary, aphasia has been given a wider significance, a significance that attaches to it to-day, namely, the total inability or partial disability of an individual to make outward expression of thoughts, feelings, or other states of consciousness, whether such disability result from interference with the formation of the mental content or in the emission of it.

Aphasia has gone through many evolutionary stages. After the first wave of its universal recognition had subsided, it came to be looked upon as a symptom with an established seat and an accompaniment of definite lesions. As the literature of the subject grew and the cases with anomalous symptom complexes became more numerous, it was seen that they did not fit in with the simple conceptions of those who stood sponsors for its original recognition. This, coupled with the apparent ambition of almost every one who wrote on the subject to adopt a new nomenclature, soon robbed the symptom of its attributed pristine simplicity, and showed it to be in reality one of the most complex subjects with which the physician and psychologist had to deal.

Former and current nomenclature fostered the obscurity in which the subject of aphasia has been and is still enshrouded. After aphasia had been recognized as a symptom indicative of localized lesion in the brain, the use of the words "ataxic" and "amnesic" aphasia, the one to indicate the aphasia of impaired articulation, the other to indicate loss of memory of the word, stood obstinately in the way of ready comprehension of the speech disturb-

ances attending given cases. Such classification, however, did not offer material hindrance to those who desired to study the subject of aphasia seriously and scientifically, nor yet did the diagrammatic portrayal which held sway for a long time.

As time went on, students of the anatomy and physiology of the brain began to unravel the intricacies of its architecture and the mysteries of its functions; and it became apparent that diagrammatic methods, which necessitated allocating certain functions to definitely and sharply localized areas and the connection of these areas, the one with the other, by individual fibres, as well as the representation of such connection in the incoming and outgoing pathways, were inimical to the findings of science. It will be shown in the chapter on the psycho-physiological conception of speech, that the entire speech area, *i.e.*, all that part of the cortex whose functional integrity must be preserved for the production of speech, whether it be concerned in the reception of impressions or in the emission of them, is practically a sensory area. Study of the anatomy of the brain, particularly by the aid of the embryological method, which Flechsig has made so uniquely his own, has thus been of service in interpreting the symptomatology of aphasia.

A glance at the literature register appended to this monograph,[1] and a perusal of the chapter devoted to the history of the subject, will show that delay in establishing a satisfactory explanation of aphasia cannot be attributed

[1] There is appended to the original manuscript of this essay, now in the archives of the College of Physicians of Philadelphia, a bibliography of aphasia which aims to indicate the literature of the subject up to January 1st, 1897.

to dearth of reports of cases. In fact, the bare report of a simple case of aphasia is to-day even considered by many writers to be a dignified procedure; and, although it has been a sort of covenant among medical writers that, as soon as the symptom complex of a disease became universally recognized, nothing was to be gained by putting on record bare reports of cases that offered not the slightest difficulty in their interpretation and that contributed nothing to the elucidation of the problem to be solved, recalcitrant covenanters are numerous where aphasia is concerned. To show that this is not an exaggeration, I may refer to an instance of recent date in which the president of a most learned and dignified European medical society cited a simple case of subcortical motor aphasia, which he referred to as "quite remarkable," as a contribution to "A Discussion on Aphasia" which had been opened by a savant who is known in every medical community and who, in closing the discussion, disposed of the simple case in a few simple words.

I would not be understood to say that the study of the simplest cases of aphasia is not of value. It is of paramount importance, and especially study of the morbid anatomy. It is in such cases that the relation between cause and effect is most distinctly traceable. Bare clinical reports have made the literature vast and they have made it a literature that is not of very great value. Comparatively few cases have been observed in which careful, accurate autopsies were made and intelligently reported. This, coupled with the varying nomenclature that has been used by different writers and the different value put upon words, has served to make the subject of aphasia appear

much more intricate than it really is, and the possession of a knowledge of it more difficult than the phenomena warrant.

That comparatively few cases carefully observed, properly reported, and accompanied by details of post-mortem findings, go far to unravel many of the apparent mysteries of aphasia has been shown by the contributions of Wernicke, Dejerine, Vialet, Redlich, Wyllie, Elder, and of many others.

Aphasia is a term used to indicate any disturbance or perversion of intellectual expression. The significance of the term has expanded from the time when its application was used to designate a defect in the verbal expression of an idea, until now it includes all defects or disorders of intellectual expression, whether such disorders be the result of disarrangement or destruction of the receptive or of the emissive components of speech mechanism, or of anything which may be employed as the analogue of speech. Thus a person who, despite the integrity of the peripheral speech mechanism, is unable to utter his own name, or to give expression to thoughts which arise in the usual way, has aphasia. If he is incapable of making known his thoughts by the employment of some equivalent of spoken words, such as writing of any sort or by expressive mimicry or pantomime, he likewise has aphasia. Moreover, a person has aphasia who, with the extra-cerebral apparatus intact, is unable to understand the language in whose use he has been trained; does not even recognize, although he hears, the sounds of the most familiar nature and words to which he has for a lifetime been accustomed, such as his own name; although he may

be able to read, is unable to write voluntarily or from dictation, or to express his thoughts by words, by symbols, or by pantomime.

Yet even these shortcomings do not comprise all that is meant by aphasia. If a person with normal ocular apparatus looks upon a printed or written page, and the symbols there with which he has previously been entirely familiar convey no meaning to him in the form of approximate thought or idea, such person has aphasia, even though he may understand all that is communicated orally to him, and though he may himself be able to express his thoughts (incompletely and defectively however) by spoken and written words.

Thus it will be seen that aphasia, in the broad usage of the term, may be the result of conditions by which the patient is unable to part with the expressive equivalent of an idea which has been properly formed. This failure is not confined to words, but includes all modes of expression. Or it may be caused by any conditions that interfere with the reception of impulses or stimuli that enter into the genesis of ideas used in the construction of internal or external language. As movement in some form is requisite for the manifestation of any and all expressions, defect of this is the condition to which the term motor aphasia is applied, a condition which is equally well expressed by the term aphasia of emission. In the second form of aphasia, the sufferer is unable to adapt receptive communications and make them fit the idea represented by the verbal symbol, auditory or visual; that is, he has lost the faculty of adapting the complement of the word to his own idea. It matters not whether these words be

spoken or written, or communicated by some equivalent, such as music and pantomime. In a general way, this is the aphasia of reception, or sensory aphasia.

Motor aphasia, or aphasia of emission, which was described by Broca as aphemia, and by many writers after him as ataxic aphasia, may be divided into as many forms as there are habitual avenues of exteriorizing thoughts. Ideas are usually exteriorized by spoken words, by written words, by symbols, and by pantomime. Thus, we have aphasia of articulation, or logaphasia; and aphasia of writing, agraphia or logagraphia; asymbolia, and asemia. Aphasia of reception, or sensory aphasia, is also made up of a number of constituents, the two great divisions being auditory aphasia or word deafness, and visual aphasia or word blindness. Each form of aphasia admits in turn of further subdivision. The understanding of the subject of aphasia depends upon a comprehension of the development of the powers of language and thought. It has seemed to me necessary, therefore, to say something of the conditions under which these are developed, before proceeding to the clinical side of aphasia, although it would be manifestly out of place in a monograph of this kind to trace the evolution of speech in detail from the time of the child's birth to the period when the speech faculties have reached their highest development. Before proceeding to this psychological consideration, however, it seems to me desirable to present as succinctly as possible a consensus of the opinions furnished by physiology, psychology, pathology, and anatomy as to the location, interconnection, and relative importance of the different speech centres; or, to be more explicit, the areas by virtue of whose integrity

a person is able to adapt sensations coming through the special senses to the idea represented by such sensations, or to adapt and produce words to ideas which are conceived by him. After a brief consideration of the history of aphasia I shall take up the subject in the manner indicated. I am aware that this mode of attack may not commend itself, at first sight, to the physician who seeks information of the clinical phenomena of aphasia alone, but I hope that it will to him who would understand the genesis of speech, as well as the phenomena of its dissolution.

Classification of Aphasia.

The externalization of thought requires:

I. (*a*) The production of movements through the musculature of the thorax, the larynx, the tongue, and the lips, and the co-ordination of the respiratory, vocal-cord, lingual, and labial movements. (*b*) Integrity of the musculature involved in writing, generally the muscles of the right hand, but sometimes the muscles of the left hand, and very rarely those of any mobile part of the body, such as the foot, and hence positing specialized motor areas in the opposite cerebral hemisphere. (*c*) Integrity of the musculature of the face, arms, and hands, and to a lesser extent of other muscles of the body which produce pantomimic expression.

II. The sense organ of hearing, which is capable of being stimulated differentially by sounds that differ in timbre, pitch, intensity, and duration, and possessing a musculature which is reflexly stimulated so as to bring about a certain degree of accommodation or adaptation of

a sense organ to sounds that differ in the above fashion, and a conduction tract leading from the labyrinth to the oblongata, thence to the internal geniculate body, and to the cortex in the anterior region of the first temporal lobe.

III. A sense organ of vision capable of differential response to form, to magnitude, and to distance, with a musculature possessing a high degree of accommodative adaptability to such different stimuli, and a conduction tract which passes directly to the external geniculate body, thence to the anterior quadrigeminal body; a larger bundle, however, passing directly from the external geniculate body into the pulvinar of the thalamus, and thence to adjacent parts, ultimately reaching the cortex of the occipital lobe in the vicinity of the calcarine fissure.

IV. Sense organs or peripheral sensory nerves in muscles, in joints, and in adjacent parts which are capable of differential stimulation by different bodily positions and by differently executed muscular movements, and afferent tracts leading from these peripheral parts to the cortex, certainly to the cortex of the Rolandic region, the so-called somæsthetic area, and possibly to other locations.

V. Apperception, a term that is used to indicate the combination of central excitations with any incoming sensory stimulus before that stimulus arouses such excitation in the cerebral cortex as to bring into consciousness a completed perception.

I deem it expedient here to say a few words concerning the use of the term apperception, which, although thor-

oughly familiar to psychologists, is by no means so to physicians. Apperception is a factor that enters into all such processes as understanding, interpreting, identifying. It is not an element of consciousness, but a process that must intervene between the presentation of an object to consciousness and the disposition of such presentation. It cannot be maintained that there are locally separable centres of apperception and of sensation. Apperception involves past experiences acting on the cortex, leaving what are called memory traces. Every perception and every enunciation of speech involves the co-ordinate act of incoming sensory impulses and of these cortical memory traces. The adult consciousness never has a simple sensation which could be supposed to be the resultant of the activity of the ganglion cells of a distinctive sensory centre; but incoming impulses may evoke few or many of these memory traces, in some cases involving activities which represent memory traces that are the resultant of the actions of different sense organs. A study of aphasia teaches us that this central associating or co-ordinating power may become lost, and the patient's apperceptive powers be restricted, in some cases even to a state approximating simple sensation.

That an auditory impulse, passing up the acoustic nerve through the oblongata, through the temporal lobe, and thence radiating posteriorly, inferiorly, and dorsally, awakes in temporal succession these associated memory traces, cannot be doubted. Consequently at one point we may assume that we have a simple sensation. That point is certainly under normal conditions not higher than the initial auditory receptive cells in the middle portion of

the superior temporal convolutions. Whether a simple sensation or any consciousness at all is produced by an activity lower down cannot be determined.

A classification of aphasia is of much service in contributing to a ready understanding of the symptom aphasia. I shall endeavor to give a simple, natural classification that shall be in harmony with the interpretation of aphasia that is expounded in the chapter devoted to "Conception of Aphasia."

Personally, I am convinced that the time has come to make radical departure from the usual classification of aphasia. We should cease the use of such misleading terminology as ataxic aphasia, amnesic aphasia, etc., and discourage the attitude adopted by British writers, who use the term aphasia only for the condition that most writers call motor aphasia, and the term amnesia for sensory aphasia. To illustrate how confusing and misleading the nomenclature of the Britishers may be, I cite an instance of very recent occurrence. A Colonial physician read an excellent paper on "Motor and Sensory Aphasia" before the Royal Medical and Chirurgical Society. On rising to discuss it, a member of the society said that he thought the term aphasia was hardly justified! He would prefer to call the condition amnesia. It must be granted, I think, that a nomenclature of this kind has not sufficient in its favor to warrant us in retaining it.

I believe that aphasia should be classified as follows:

1. True Aphasia.—Aphasia of apperception. Due to lesion of any constituent of the speech region, the zone of language. It might be subdivided into (*a*) visual aphasia, due to lesion of the visual areas and centre; (*b*) auditory

Associative or Transcortical Aphasia.

I shall content myself with mere mention of this subdivision, as its symptomatology is in reality a part of sensory aphasia. Variation in the clinical picture is in accordance with the location of the lesion between speech areas in the zone of language. The lesion may be:

1. In the habitual pathway traversed by impulses going from the auditory to the visual area (the patient can hear a name but cannot write it from hearing; cannot write it from dictation; he has paragraphia).

2. It may be in the habitual pathway of impulses going from the visual area to the auditory area (the patient can see an object, but he cannot call up its name, because this requires the mediation of the auditory area).

3. A lesion that interrupts the habitual pathway that impulses take when going from the auditory to the seat of phonetic memories in Broca's convolution (the patient can hear, can interpret from hearing, but cannot talk correctly: paraphasia).

4. The lesion may interrupt the pathway taken by impulses going from the visual area to the auditory area (the patient is dyslexic, paragraphic, and slightly paraphasic).

Thus it will be seen that the two great divisions of aphasia are sensory aphasia and motor aphasia. As I have said previously, many British writers, such as Bastian, use the term amnesia synonymously with sensory aphasia, and a greater number of writers use the term aphemia, originally employed by Broca, as the specific term for the motor form of aphasia.

Both these forms of aphasia are so complex that it is nec-

essary to have recourse to a further subdivision; the most satisfactory basis for which will have reference to the seat of the lesion that produces the speech disturbance. Thus we have cortical sensory aphasia when the lesion is of the speech centre itself, and subcortical sensory aphasia when the perversion of the function of speech is due to a lesion of the sensory tracts leading to it. The same subdivision is made of motor aphasia, *i.e.*, cortical motor aphasia, and subcortical motor aphasia. The designations cortical sensory and cortical motor aphasia are often abbreviated to simply sensory aphasia and motor aphasia and the subcortical forms to *pure* sensory aphasia and *pure* motor aphasia (Dejerine, Mirallié, *et al.*), thus signifying that sensory and motor tracts alone are involved.

CHAPTER II.

History.

The real history of aphasia dates from 1861. In that year Broca presented an epoch-making communication to the Paris Anatomical Society, which seemed to prove that the morbid anatomy of aphasia was a lesion of the posterior part of the third convolution.[1] Before that time much speculation had been indulged in concerning the seat of the faculty of language and the location of a lesion that would interfere with its production. In the beginning of the second half of the nineteenth century two patients were admitted into the Bicêtre Hospital in Paris whose disorders of speech, thanks to the scientific zeal and clinical insight of Broca, surgeon to that institution, have contributed immeasurably to the understanding of speech in both health and disease.

The first of Broca's cases was an epileptic who had been for twenty years almost completely bereft of expressive speech, word production being limited to "tan, tan." The receptive faculties that contribute to speech were unimpaired, and he was able to indulge in pantomime. As

[1] Broca: "Remarques sur le siége de la faculté du langage articulé, suivies d'une observation d'aphémie." Bulletin de la Société Anatomique, August, 1861, p. 330.—"Nouvelle observation d'aphémie produite par une lésion de la moitié postérieure des deuxième et troisième circonvolutions frontales." Bulletin de la Société Anatomique, November, 1861, p. 398.

a slowly progressing right hemiplegia developed, intelligence waned and sight became dim. After death an examination of the brain revealed a widespread softening in the left hemisphere. The most ancient softened area, the one to which the aphasia was attributed properly, as this symptom had preceded the hemiplegia for many years, was confined to the middle and sylvian side of the frontal lobe. The second case corroborated in detail the conclusions drawn from the first. This patient understood all that was said, and, although articulatory production was reduced to three or four words, still he used these intelligently and discreetly. He was able to reckon, and in a general way intelligence was unimpaired. Autopsy showed a cavity confined to the posterior third of the second and third frontal convolutions, a region which soon became and is still known as Broca's area. The report of these two cases and the discussion to which it gave rise attracted widespread attention, and from that time dates the scientific history of aphasia.

At first many of the most eminent minds in the profession refused to believe that the emissive speech faculty was confined to the left hemisphere; indeed Broca himself looked upon the lesion in the left half of the brain in his two cases as merely accidental, and stated his conviction that wider experience would show that lesion of the corresponding part of the right hemisphere was as common as of the left. The French surgeon was scientific but not prophetic, for the scores of cases that were soon studied and reported showed that the lesion was almost invariably in the left hemisphere. From consideration of

the reported cases and from wider personal experience, Broca was led to offer in explanation of this seeming paradox of anatomical selection the hypothesis that the great majority of humanity are right-handed and therefore left-brained; that as the capacity for delicate motor dexterousness is most developed in the left brain, so are the exquisitely co-ordinated movements which subserve speech; an explanation which has formed the basis for other more comprehensive hypotheses. Indeed Voisin[1] had made an observation which seemed to demand such a conclusion; a patient with left-sided hemiplegia had no difficulty in interpreting language or in using language. Later he developed a right-sided hemiplegia and complete aphasia. It will be seen in further perusal of this monograph that the aphasia which Broca described and which he designated by the term aphemia, a word hostilely received by purists in etymology, as literally it means "infamy," is but a form or variety of the speech disturbances now included under that heading.

Broca's communication opened a new era in the understanding of speech. It was, however, no less epochal than that which marks the direction of Wernicke's master mind toward a solution of the problems of aphasia in the early years of the seventh decade of this century. Without taking upon myself the invidious task of selecting the name of the one who, next to Broca, has made the most material contribution to the subject of aphasia, it may be said that Broca and Wernicke should be accorded equal parental rights in the interpretation of the complexity of

[1] A. Voisin: "Observation de perte de la parole." Bulletin de l'Académie de Médecine, p. 1,241, 1862.

symptoms included under the term aphasia to-day. Speech disturbances had been recognized clinically and studied even so far back as the time of ancient Grecian writers, who used the term anandia to signify loss of speech; but the first records of serious attempts to study the faculty of speech from the viewpoint of its pathology were made by Bouillaud in France in 1825, by Jackson in this country in 1829, by Dax in 1836, and by Lordat in 1843. The latter designated the disturbance of speech production by the word alalia. Many years before this the brilliant, misguided Gall had located the faculty of speech in the supra-orbital convolutions. Gall died in 1828, and there can be no doubt that his teachings, which, it should never be forgotten, were based in part upon carefully made dissections of the brain, had much to do with arousing the interest manifested in these questions at that time and immediately following.

Bouillaud advanced the opinion that the anterior extremity of the frontal lobes, despite the contentions of Gall, was the seat of the faculty of speech. He was supported in this contention by Pinel, Grandchamp, and Belhomme, the latter of whom presented to L'Académie Royale de Médecine a memoir on the "Localization of Speech in the Anterior Lobes of the Brain," in which by a reference to ten cases he endeavored to prove that the cerebral organ which regulates speech is seated in the anterior lobes of the brain.

Early in the century, Lordat[1] seemed to have an

[1] Lordat: Revue Périodique de la Société de Paris, December, 1820, p. 317.

astonishingly accurate conception of aphasic speech disturbances as they are understood to-day, for he distinguished between a loss of memory of words, which he designated "amnésie verbale," and inability to pronounce or produce words which are the result of thought, a condition called by him "asynergie verbale." He also recognized the speech defect which is now called paraphasia, and he applied to it the term "paramnésie verbale." The Germans were in the field early and made several contributions to the study of aphasia, among them being those of Bergmann in 1849, Hasbach in 1852, and Nasse in 1853, but none of these was more than a report of cases analyzed and criticised.

From English observers no contributions of signal importance, supported by convincing anatomical proof, were made prior to the publication of Broca's report. Naturally such astute clinicians as Prichard, Crichton, Winslow, and Watson did not allow the striking and withal not uncommon symptom to escape their notice and remark, but their contributions did not materially advance the knowledge of the subject. In our own country the observations of Jackson have already been mentioned, and to them must be added those of Rush,[1] Hun,[2] Bigelow, and others.

The history of aphasia, dating from the time of Broca's first contributions made in 1861 and in 1865, up to the present day, if written in detail, now at the end of the century, would be more replete with references than the history of any other symptom or disease of the nervous

[1] Rush: "Practice of Physic," Philadelphia, 1846.
[2] American Journal of Insanity, 1851.

system. Mention of important contributions alone covers many pages, while to cite the authors who simply reported cases corroborating or denying the anatomical seat of aphasia as stated by Broca, and confirmatory of certain forms or types of the disease, would consume an enormous amount of space and be without particular value except to the bibliographer. In the text, I shall therefore mention only a few authors of the different countries who have made such contributions to the subject as are destined to last as long as the science of medicine. To the storehouses of information erected by these writers every one who essays to write on aphasia must go.

The most important early American contribution made to the subject of aphasia was that of Seguin,[1] who in 1867 published an elaborate essay detailing a number of cases personally observed and adding a critical review of a large number gathered from the literature. In this essay he showed that out of a total of two hundred and seventy cases of hemiplegia with aphasia, two hundred and forty-three were paralyzed on the right side of the body. This corresponds practically to the ratio of right-handed to left-handed persons. Several years later Amidon[2] made an important contribution to the pathological anatomy of sensory aphasia, which included a critical analysis of several typical cases reported by European writers.

[1] Seguin: "A Statement of the Aphasia Question, together with a Report of Fifty Cases." Quarterly Journal of Psychological Medicine, New York, 1868, vol. ii., pp. 74–179.

[2] Amidon: "On the Pathological Anatomy of Sensory Aphasia." New York Medical Journal, January 31st, 1885, vol. xli., pp. 113, 181.

In England the labors of Russell,[1] Gairdner,[2] Bastian,[3] Broadbent,[4] Ogle,[5] and Bateman[6] contributed largely to our present knowledge; in fact the names of these physicians are landmarks in the history of aphasia. Ogle coined the word agraphia, although the condition was described first by Marcé. Gairdner insisted that a patient with motor aphasia writes at least as badly as he speaks, and that the patient wholly unable to speak is also incapable of writing. Bastian, to whose interpretation of clinical phenomena psychology and neurology owe so much, was the first to recognize the relationship of defective auditory perception to speech production. He introduced the word "jargonaphasia" to indicate the conglomeration of syllables, words, and phrases strung together without order or meaning which is the striking feature of certain forms of sensory aphasia. Broadbent was the first to point out, in 1872, the occurrence of the condition which is now known as word blindness, and although very few of the hypotheses advanced by this writer are accepted to-day, I believe, by scarcely an individual aside from a few personal adherents, his writings have been of great service in furthering an understanding of some aspects of aphasia.

[1] James Russell: British Medical Journal, 1864.

[2] Gairdner: "The Function of Articulate Speech, with Observations on Aphasia," Glasgow, 1866.

[3] Bastian: St. George's Hospital Reports, vol. ii., p. 95; British and Foreign Medico-Chirurgical Review, vol. xliii., 1869, pp. 209, 236, 470, 492.—"The Physiology of Thinking." Fortnightly Review, 1869.

[4] Broadbent: "Cerebral Mechanism of Thought and Speech." Medico-Chirurgical Transactions, vol. iv., 1872.

[5] Ogle: "Aphasia and Agraphia." St. George's Hospital Reports, vol. ii., pp. 83–121. Medico-Chirurgical Transactions, vol. liv., 1871.

[6] Bateman: "Aphasia or Loss of Speech in Cerebral Disease." London, 1868; Revised Edition, London, 1890.

In Germany the work of Wernicke,[1] Grashey,[2] Lichtheim,[3] and Freud[4] has done much to extend the conception of the term aphasia and to give rational explanations of some of its subdivisions. Wernicke particularly, in 1874, furnished a basis for the conception of sensory aphasia. He pointed out that many cases recorded as anomalous examples of aphasia could be explained on the ground that there are several varieties, and that the most typical aphasia can occur with lesion of other parts of the brain than Broca's convolution. He showed clearly that lesion of the first temporal gyrus produced a symptom complex constituting what he called sensory aphasia, and he portrayed the individual on whose ears the words and sentences of his mother language fall like those of a wholly unknown tongue, and to whose eyes the letters that he formerly read with an ease akin to instinct are as hieroglyphs. In short, he interpreted the phenomena of word deafness and word blindness and made them readily comprehensible by parallelizing them with the phenomena

[1] Wernicke: "Der aphasische Symptomcomplex," Breslau, 1874.—"Aphasie und Geisteskrankheit." Deutsche medicinische Wochenschrift, p. 445, 1890.—"Lehrbuch der Gehirnkrankheiten," Berlin, 1881.—"Grundriss der Psychiatrie in klinischen Vorlesungen," 1894.—"Aphasie und Anarthrie." Deutsche medicinische Wochenschrift, p. 163, 1882.—"Ueber die motorische Sprachbahn und das Verhältniss der Aphasie mit Anarthrie." Fortschritte der Medicin, No. 1, pp. 1, 405, 1884.—"Replik auf Dr. Kussmaul's Entgegnung die Aphasie betreffend." Fortschritte der Medicin, Berlin, 1883, pp. 313, 316.—"Die neueren Arbeiten über Aphasie." Fortschritte der Medicin, ii., 1885, p. 24.

Wernicke and Friedländer: Fortschritte der Medicin, Berlin, 1883, No. 6, p. 177.

[2] Grashey: "Ueber Aphasie." Archiv für Psychiatrie und Nervenheilkunde, vol. xvi., 1885.

[3] Lichtheim: "Aphasia," Brain, January, 1885.—"Ueber Aphasie." Deutsches Archiv für klinische Medecin, 1884–85, p. 204.

[4] Freud: "Zur Auffassung der Aphasien," Leipzig, 1891.

of motor aphasia, which by this time had been put on a firm anatomical basis. Another very important service which this writer rendered was to give proper application to the term sensory aphasia and to submerge the term amnesic aphasia, which for a time had found its way into literature.

Aside from Broca and Wernicke, the three great names that stand out above those of all the others who early increased our knowledge of aphasia are Trousseau[1] in France, Hughlings Jackson[2] in England, and Kussmaul[3] in Germany, the latter having written what is to-day the soundest and fullest treatise on the subject of aphasia in any language.

In his exhaustive and philosophical consideration of aphasia, published in 1877, Kussmaul introduced the terms "word deafness" and "word blindness" to indicate the conditions described by Wernicke, and although the latter and others hesitated to accept some of the conten-

[1] Trousseau: "Discours sur l'Aphasie." Bull. Acad. Méd., February 25th, 1861; Clin. Méd. de l'Hôtel Dieu, vol. ii., 1865, p. 571; Bull. Acad. Méd., June, 1865.—"Leçons cliniques sur l'aphasie." Gazette des Hôpitaux, February and March, 1864.—"Paralysie progressive de la langue, des lèvres et du voile du palais." Gazette des Hôpitaux, January and February, 1863.

[2] Jackson (Hughlings): "Loss of Speech, Its Association with Valvular Diseases of the Heart and with Hemiplegia of the Right Side." Clinical Lecture and Report of the London Hospital, Lancet, ii., 1864, p. 604.—"On Affection of Speech from Diseases of the Brain." Brain, 1879–80, t. 2, pp. 203–323.—"On Aphasia with Left Hemiplegia." Lancet, 1880, p. 637.—"On Slight and Severe Epileptic Attacks with Auditory Warnings; Slight Paroxysms with Deafness and the Special Imperception called Word-Blindness; Spectral (Auditory) Words; Inability to Speak and to Write." Lancet, July 28th, 1894.

[3] Kussmaul: Article "Aphasia (Disturbances of Speech)." Ziemssen's Cyclopedia, vol. xii., Wm. Wood & Co.—"Entgegnung die Aphasie betreffend." Fortschritte der Medicin, Berlin, 1883, pp. 309–313.

tions of this writer, particularly those bearing on the individuality of word blindness and its relation to defective speech production, the experience of innumerable workers in the field of aphasia since that time has supported Kussmaul.

In 1881, Exner,[1] of Vienna, was led, from an analysis of the disturbances of intellectual expression attending cases of aphasia, to allocate a separate area of the brain in which are stored the motor memories of writing, destruction of which caused agraphia. This centre he placed at the base of the left second frontal convolution, and, although to-day no one gives credence to his method of investigation, his conclusions have been a point around which an active war has been waged from that day to this.

Although not so early in the field as others, the Italians have furnished a number of contributions of the greatest value. Of these mention must be made of the works of Seppilli[2] and of Banti,[3] both of a high standard of excellence.

In France the numerous and worthy contributions of Trousseau were ably followed up by those of Charcot, who in his inimitable way suggested an explanation of aphasia based apparently on a psychological analysis of speech, contrasting the acquisition of speech with the phenomena of dissociation occurring in disease. For a long time this theory seemed to clarify the atmosphere of any haziness

[1] Exner: "Untersuchungen über die Localisation der Functionen in der Grosshirnrinde des Menschen," Wien, 1881.

[2] Seppilli: "La sordità verbale od afasia sensoriale." Riv. Sperim. di Frenatr., Reggio-Emilia, 1884, pp. 94–95.

[3] Banti: "Afasia e sue forme." Sperimentale, Firenze, 1886.

surrounding the interpretation of aphasia, while at the same time it furthered the claim of aphasia to accurate anatomical localization. The contributions of Charcot[1] had a capable, lucid exponent and commentator in his pupil, Bernard.[2]

It is the first duty of the historian to be truthful and exact in the statement of facts, and that he may be worthy of his task candor compels the historian of aphasia to state without equivocation that Charcot did more to prevent the true interpretation of aphasia as it is understood to-day than did any other writer who essayed to illumine the subject. The scientific position of this deservedly honored and renowned physician was such an exalted one and his utterances were so generally considered *ex cathedra*, not alone by his disciples but by his contemporaries, that statements from his pen and from his lips were accepted as truth before they had received the corroboration and substantiation of others, as the true scientific spirit demands. Far be it from me to detract from the claims to immortality of one of the greatest clinicians of the nine-

[1] Charcot: "Cécité verbale. Leçons sur les Maladies du Système Nerveux," t. iii., 1887, p. 152.—"Observation d'hémiplegie droite avec aphémie et avec integrité des lobes antérieurs et des circonvolutions frontales." Gaz. Hebd., 17 Juillet, 1863.—"Des variétés de l'aphasie. Cécité verbale." Progrès Médical, 1883, pp. 23, 27, 441, 521, 859.—"Des variétés de l'aphasie. Aphasie motrice." Journal de la Santé Publique, 1883.—"Cas rare de cécité verbale pris à la clinique de Charcot." Wiener medizinische Presse, 1883, p. 834.—"Cécité verbale." Gazette Médicale, Paris, 21 Juillet, 1883, No. 29, p. 339.—"De l'aphasie." Progrès Médical, 4 Février, 1888, p. 81.—"Leçons sur les localisations dans les maladies du cerveau recueillis par Bourneville." Progrès Médical, 1876.

Charcot and Dutil: "Agraphie motrice suivie d'autopsie." Société de Biologie, Juillet, 1893, p. 129.

[2] Bernard: "De l'aphasie et ses diverses formes." Thèse Doctorat, Paris, 1885.

teenth century, but his excursion afield into the subject of aphasia has been a perpetual obstacle to the dissemination of the view of aphasia that passes muster to-day, and that has been endeavoring to get a foothold since Wernicke and Bastian first began their interpretations of aphasic speech disturbances.

Charcot framed a hypothesis for the explanation of speech, using the word bell, the object bell, the sound bell, the touch bell, to illustrate the various avenues by which sensory impressions arise in the consciousness and to show that the memories of such impressions were stored up in individual centres. The speech centres he taught were in pairs, two for reception of information, the auditory centre and the visual centre, and two for emission of impulses representing ideas, the articulate speech centre and the graphic-motor centre. These centres Charcot asserted, with great assiduity and aplomb, were possessed of striking functional autonomy which they had phylogenetically and as the result of education. A person inherited, or was born with, a tendency to form concepts mainly by arousing them through the visual, the auditory, or the articulatory images, and was destined therefore to be in thought and speech a *visuel*, an *auditif*, a *moteur*, and this predisposition was greatly advanced by education. Charcot adopted early the conclusions of Exner concerning the existence of a separate graphic-motor centre, although the evidence which the latter adduced in support of his claims was about as unscientific as anything could be. Nevertheless Charcot immediately proceeded to speak of the graphic-motor centre as if its existence and individuality were beyond a doubt.

The interdependence of the four centres, and the necessity for their harmonious co-operation, in order that intellectual expression be complete, was recognized, but the autonomy of each centre was contended for unswervingly. Charcot claimed that from the standpoint of development the centres were independent and separate, and that methods of education as well as natural proclivities tended to the development of one of the centres out of proportion to the others. In this way, those who receive the wealth of their sensory impressions through the hearing apparatus, are preponderatingly *auditives;* those whose education is most aided by their organs of vision, *visuels;* and so on. It was taught, however, that by means of education all of these centres might be developed more harmoniously than they are usually. The conception of the school of Charcot is a simpler one than that of the Germans, and for this reason it has been of service; but to the latter must be conceded an ability to unravel the intricacies of the complicated forms of aphasia in which the former fails.

While Charcot's views were being adopted the reign of diagrams began, an epoch which seems to us to-day to have been attended principally by the construction of schemata by which could be explained theoretically the different forms of aphasia that might occur from interruption in the conductive, receptive, and emissive parts of the various speech centres. To an unprejudiced observer at the time of the present writing, the activity of this period seems to have been directed toward constructing a diagram that would fit all possible forms of aphasia, the builders of the diagrams leaving it for a later task, and for others, to

find cases that would fit the theories. The names that are particularly associated with this period are Wernicke, Lichtheim, Kahler, and Pick.

Lichtheim,[1] for enample, drew a simple *triangle*, the angles of which represented the three speech centres, auditory, visual, and kinæsthetic; and the lines connecting them, the avenues over which stimuli must travel in order to produce perfect speech. In this way he showed that no less than fourteen different areas might be the seat of a lesion producing aphasia. Depending upon the primary seat of destruction and the segment of the speech pathway that is interrupted, the aphasia that results will have individual and diagnostic characteristics. Construction of such diagrams and speculation as to the form of aphasia that might result soon led to the necessity of positing a central arrangement for the interpretation of the various stimuli received through the different avenues of information. To this centre the name ideational centre was given. The existence of some such place of central reception, interpretation, and dissemination was absolutely necessary when attempts were made to subject the speech defects constituting aphasia to psychological analysis, and although no anatomical proof of the existence of such a centre could be furnished, nor from the conception of the very nature of the centre could it be allocated to any definite area in the brain, yet the reality of such an association area is universally conceded.

Because of this recognition, and as a result of processes of reasoning analogous to that which led to the establish-

[1] Lichtheim : "Aphasia." Brain, January, 1885.—"Ueber Aphasie." Deutsches Archiv für klinische Medicin, Leipzig, 1884–85, pp. 204–268.

ment of an ideational centre, grew the conception of a propositionizing centre on the motor side of the brain and of a naming centre on the sensory side, by Broadbent.[1] The latter has contended unswervingly for the reality and the existence of these centres, but the only convert to his views whose opinion is of great importance is Mills.[2]

This subject is referred to here only casually, as it will be taken up in detail and criticised in another chapter.

In 1881 appeared an essay, "Word Blindness and Word Deafness," from the pen of Mlle. Nadine Skwortzoff, which included an analysis of many cases of sensory aphasia, and which portrayed particularly the teachings of Magnan. Three years later this same side of the subject, sensory aphasia, was handled in a fashion very creditable to the author, Seppilli.[3] In 1886 Ballet[4] contributed a very thoughtful and serviceable article on "Internal Language and the Various Forms of Aphasia," and in the following year Ross published a series of papers in the *Medical Chronicle* on "Aphasia" which are to-day among the most lucid and trustworthy expositions of the subject

[1] Broadbent: "Cerebral Mechanism of Speech and Thought." Med.-Chirurg. Transactions, vol. iv., 1872.—"Case of Amnesia with Post-Mortem Examination." Lancet, March 2d, 1878, p. 312.

[2] Mills: "On the Localization of the Auditory Centre." Brain, 1891. —"Disorder of Pantomime occurring among Aphasics." Philadelphia Hospital Reports, 1893, vol. ii., p. 142.

Mills and McDonnell: "The Naming-Centre." Journal of Nervous and Mental Disease, vol. xx., 1895.

[3] Seppilli: La sordità verbale od afasia sensoriale." Riv. Sperim. di Frenatr., p. 94, 1884.

[4] Ballet: "Le langage intérieur et les diverses formes de l'aphasie." Th. Agrég., Paris, 1886.

in the English language. In these articles Ross strove to show, and succeeded I believe, that the contentions of Broadbent concerning a propositionizing centre on the motor side of language and a naming centre on the sensory side were erroneous.

After the claims for the separate localization of graphic-motor images had become more universally urged, diagrams framed to explain the symptoms of aphasia took on the shape of a parallelogram, each angle representing the seat of one of the allocated functions, the auditory, visual, motor, and graphic-motor, but which had the merit of distinguishing between the kinæsthetic and the emissive-motor sides of speech. The names of some of those who made special study of agraphia are Charcot, Pitres,[1] Wernicke, Lichtheim, and Grasset.[2]

All this time there had been accumulating evidence in the shape of apparently anomalous cases to show that the conception of aphasia as taught by Charcot and his school was largely erroneous, and the labors of Dejerine and of his pupils, of Serieux, and of others to be hereinafter mentioned, led to the overthrow of the reign of autonomous speech centres.

[1] Pitres: "L'aphasie chez les polyglottes." Revue de Médecine, November, 1895.—Congrès Méd. Interne, Lyon, 1894; Des Aphasies.—"Agraphie motrice pure." Revue de Médecine, Paris, 1884, pp. 855–873.—"Recherches sur les lésions du centre ovale."· Thèse de Paris, 1877.—"Localisations cérébrales." Gaz. Méd., 1876, p. 474.—"Aphasie et hémiplegie." Société Anatomique, 1875, p. 783.—"Rapport sur les aphasies." Journal de Médecine de Bordeaux, 1894, xxiv., 469–481.—Gazette Hebdomadaire de Médecine, Paris, 1894, v., 1,365–1,370.

[2] Grasset: "Localisation dans mal cérébral." Montpellier Méd., t. xl. et xli., 1878.—"Cécité et surdité verbales." Montpellier Méd., 1884, pp. 29–50.—"Des diverses variétes cliniques d'aphasie." Nouveau Montpellier Méd., 15th, 22d, 29th February, 1896.

The contributions of Dejerine[1] bearing on the subject of aphasia deserve special mention. As early as 1879 he made an autopsy on a patient who during life had such symptoms that the diagnosis of subcortical motor aphasia was made, and the autopsy fully corroborated it. This contribution was one of the first to show the necessity of differentiating cortical and subcortical aphasia, although perhaps to Pitres more than to any other individual writer is due our knowledge of subcortical motor aphasia, to which he turned his attention as early as 1877. The really important contributions of Dejerine to the subject of aphasia are embodied in a few short communications to the Société

[1] Dejerine : "Aphasie et cécité des mots." Progrès Médical, p. 629 ; Bull. de la Société Biologie, Juin, 1880.—"Aphasie et hémiplegie droite. Disparition de l'aphasie au bout de neuf mois. Lésion du faisceau pédiculo-frontal inférieur gauche, du moyau lenticulaire et de la partie antérieure de la capsule interne." Société Anatomique, 1879, p. 16.—"De l'aphasie et de ses differentes formes ; études de semiotique et de physiologie pathologique." Semaine Médicale, Paris 1884, second series, iv., 421–449.—"Contribution à l'étude anatomo-pathologique et clinique des differentes variétés de cécité verbale." Mémoires de la Société Biologie, 1892, p. 61.—"Sur un cas d'aphasie sensorielle (cécité et surdité verbales), suivi d'autopsie." Société Biologie, 1891, pp. 167–173.—"Sur un cas de cécité verbale avec agraphie." Autopsie. Société Biologie, 1891, pp. 197–201.—"De l'agraphie." Leçon clinique. Annales de Médecine, 1891.—"Contribution à l'étude des troubles de l'écriture chez les aphasiques." Société Biologie Mémoires, 1891, p. 97–113.—"Aphasie motrice sous-corticale et localisation cérébrale des centres laryngés (muscles phonateurs)." Société Biologie, 1891, pp. 155–162.—"Étude sur l'aphasie dans les lésions de l'insula de Reil." Rev. Méd., 1885, pp. 174–191.—"L'aphasie et ses formes." Semaine Méd., 1884, Nos. 44 et 47.—"Remarques à propos de la communication de MM. Charcot et Dutil." Société Biologie, 1893, p. 200.—"Aphasie sensorielle." Bulletin Médical, March 20th, 1895.—"Remarques à propos de la communication de M. Mirallié." Société Biologie, March 30th, 1895.

Dejerine and Mirallié : "La lecture mentale chez les aphasiques moteurs corticaux." Société Biologie, July 6th, 1895.

Dejerine et Vialet : "Contribution à l'étude de la localisation anatomique de la cécité verbale pure." Société Biologie, 29 Juillet, 1893, p. 793.—"Autopsie de cécité corticale." Société Biologie, 9 Decembre, 1893.

de Biologie in the years 1891–95. In these he established the dependence of sensory agraphia upon loss of visual memories in the angular gyrus, which causes alteration of internal language due to loss of visual images, associated with word blindness. He combated successfully the existence of a separate centre for the registration and storage of graphic images. Moreover, without directly polemicizing against the teachings of Charcot concerning the autonomy of the various centres in which are stored the different memory images, his contributions were of the greatest worth in tending to offset these views.

Dejerine also did much to establish clearly the distinction between word-blindness due to loss of visual memories in the angular gyrus (which is always associated with disturbance of internal language, agraphia, and paraphasia, and never with hemianopsia if the lesion is strictly confined) and word blindness due to lesion between the higher visual centres and the primary or true visual area in the occipital lobes. His contentions were in accord with the pathological findings of Henschen and the anatomico-pathological and clinical claims of Seguin, and were of such unique and convincing nature that there has been little hesitation in accepting them. Dejerine had in a pupil, Vialet, one fully worthy of his master. The contribution of this brilliant young physician, entitled "The Cerebral Centres of Vision and the Intercerebral Visual Nervous Apparatus," published in 1893, will serve to perpetuate his name.

The findings and interpretations of Dejerine were corroborated by the cases carefully observed clinically

and fully reported anatomically by Serieux[1] and by Berkhan.[2]

In 1893 and 1894 Wyllie's comprehensive lectures on the disturbances of speech appeared. In these a review of the status of speech disturbances was given, and in the chapters on aphasia particular attention devoted to the separation of the forms of motor aphasia and to an attempt at localizing psycho-motor images and the area in which such are stored. Wyllie's treatise[3] compels admiration because of the evidence of industry and perspicaciousness on every page. But all in all, the chapters on aphasia are not quite so critical as the period and the accessible material would seem to justify. Reference to one chapter may suffice to show this. Although he is duly appreciative of the value of Dejerine's contributions, and apparently upholds his contentions, he nevertheless proceeds, in a half-hearted way, to indite a chapter on graphic-motor aphasia as an individual condition. But even the believer in such a centre must be disappointed in reading it, for the author does not make so strong a presentation as could be made from the material at hand. In the following year, Freund,[4] of Breslau, published a short monograph, the thesis of which was that the conception of so-called subcortical sensory aphasia is entirely too narrow and limited. He furnishes

[1] Serieux: "Aphasie; cécité verbale, agraphie, hémiplegie gauche." Société Anatomique, 1891, p. 258.—"Sur un cas d'agraphie sensorielle avec autopsie." Mémoires de la Société Biologie, 1891, p. 195.—"Cas de cécité verbale avec autopsie." Société Biologie, 16 Janvier, 1892.—"Cas de surdité verbale pure." Rev. Méd., 1893, xiii., pp. 733–750.

[2] Berkhan: "Ein Fall von subcorticaler Alexie." Archiv für Psychiatrie, Bd. xxiii., 1892, H. 2, S. 92.

[3] Wyllie: "Disorders of Speech," Edinburgh, 1894.

[4] Freund: "Labyrinthtaubheit und Sprachtaubheit," Wiesbaden, 1895.

clinical observations of his own and analyzes others from the literature (one of which is strangely enough a case that Wernicke utilized originally to substantiate the occurrence of this form of aphasia) to show that the symptom complex as originally described by Wernicke must be enlarged to include labyrinthine disease, and perhaps other forms of affections of the peripheral auditory apparatus. He supports his contentions by citations from Freud, from Pick, from Adler, from Bleuler, and from other writers.

In the same year Redlich[1] gave a most detailed report, clinical and pathological, of a case of pure word-blindness, which is a model of its kind and which deserves to be mentioned as an example to be followed in making communications on this subject. In 1896 Mirallié,[2] a pupil of Dejerine, reviewed his teacher's work, made an analysis of the cases of sensory aphasia which have been recorded with autopsy findings, published several new observations of sensory aphasia, and gave a *résumé* of the status as represented by the school of Dejerine to-day.

In the following year appeared an apologist for the contentions of Brissaud, echoing the teachings of Charcot. Lantzenberg[3] aims, apparently, in his brochure on "Motor Aphasia" to do for his teacher what Mirallié did for Dejerine. This opuscle, which contains nothing new or original, as well as three important English contributions, appeared after the completion of the writer's mono-

[1] Redlich: "Ueber die sogenannte subcorticale Alexie." Jahrbuch für Psychiatrie und Neurologie, vol. xiii., 1895.

[2] Mirallié: "L'aphasie sensorielle," Paris, 1896.

[3] Lantzenberg: "L'aphasie motrice," Paris, 1897.

graph, and reference to them is here interpolated to show that interest in the phenomena of the dissolution of speech is not on the wane. Of these contributions in the English language, Bastian's[1] was most eagerly awaited. One of the pioneers in the subject, one whose early utterances on aphasia have been shown by time to be astoundingly unerring, has had opportunity to look over the entire field, to review the subject, and to modify, to reiterate, and to recant. In another part of this work I shall approach some of his claims with the deference and criticalness to which they are entitled. Another English writer, Bramwell,[2] of Edinburgh, one of our most trustworthy expounders of the intricacies of neurology, reviewed in the same year a number of extremely interesting cases, and illuminated in discussing them some of the most obscure and debatable points on the subject. On the whole, his conclusions are very much in accord with those of Dejerine, Mirallié, and the present writer. The last of the three above-mentioned contributions is that of Elder,[3] who has made a painstaking and careful study. His conclusions reflect the teachings of Wyllie and evidence his discipleship.

Thus even a general view of the history of aphasia suffices to show that this symptom has attracted the attention of many men and minds during the past third of the nineteenth century. It shows also that aphasia, the

[1] Bastian: "The Lumleian Lectures on Some Problems in Connection with Aphasia." Lancet, April 3d, 10th, 24th; May 1st, 1897.

[2] Bramwell: "Illustrative Cases of Aphasia." Lancet, March 20th and 27th, 1897.

[3] Elder: "Aphasia and the Cerebral Speech Mechanism," London, 1897.

explanation of which seemed so clear and so little liable to variation, after the publication of Broca's cases, is in reality one of the most complex problems to which the physician can direct his attention. The brief survey that I have taken gives inadequate consideration to the writings of some whose work has been of the most important kind, and leaves unmentioned the names of many who have contributed materially to the understanding of aphasia and the interpretation of its intricacies, and without mention of which the history of aphasia should not be written. Such names include those of Pick,[1] Hammond,[2] Ferrier,[3] Eskridge,[4] Wilbrand,[5] Berlin,[6] Grashey,[7] Soury,[8] Sachs[9] (Breslau), Starr,[10] Henschen,[11] Thomas and Roux,[12]

[1] Pick: Archiv für Psychiatrie, t. xxiii., p. 896. "Beiträge zur Lehre von den Störungen der Sprache," 1892.—"Zur Lehre von der Dyslexie." Neurologisches Centralblatt, 1891, pp. 130–132.—"Ein Fall von transcorticaler, sensorischer Aphasie." Neurologisches Centralblatt, 1890, pp. 646–651.—"Neue Beiträge zur Pathologie der Sprache." Archiv für Psychiatrie, vol. xxviii., 1896.

[2] Hammond: "Treatise on Diseases of the Nervous System."

[3] Ferrier: "Lectures on Localization in the Brain." Brain, May, 1898. —Croonian Lectures, London, 1890.

[4] Eskridge: "Symptoms of Speech Disturbance as Aids in Cerebral Localization." University Medical Magazine, January, 1897.

[5] Wilbrand: "Die Seelenblindheit als Hirnerscheinung," Wiesbaden, 1887.

[6] Berlin: "Weitere Beobachtungen über Dyslexie mit Sectionsbefund." Archiv für Psychiatrie, 1887, pp. 289–292.

[7] Grashey: "Ueber Aphasie und ihre Beziehungen zur Wahrnehmung." Archiv für Psychiatrie, vol. xvi., 1885.

[8] Soury: "Les fonctions du cerveau," Paris, 1892.

[9] Sachs: "Ueber Bau und Thätigkeit des Grosshirns und die Lehre von der Aphasie und Seelenblindheit," Breslau, 1893.

[10] Starr: "Sensory Aphasia." Brain, 1889,.pp. 82–101.

[11] Henschen: "Klinische und anatomische Beiträge zur Pathologie des Gehirns," Upsala, 1890–96.

[12] Thomas and Roux: "Aphasiques moteurs corticaux," etc. Société Biologie, 1895, pp. 531, 731, 733; and 1896, p. 210.

Stricker,[1] and many other writers in the three great languages of science. They in their own ways and in their own times have added facts that prevent their names from being forgotten.

[1] Stricker, S.: "Zur Lehre von der Aphasie." Wiener medizinische Blätter, 1881, iv., 1,477–1,509, 1,565.

CHAPTER III.

An Analysis of the Genesis and Function of Speech.

1. General Expressive Reactions; Mimic Reactions. 2. Articulate Speech. 3. Writing. 4. The Genesis of Percepts. 5. Remarks on Visual Sensation; Acoustic Sensation, and the Mechanism of Articulation.

An introductory analysis of the so-called faculty of speech, from the psychological point of view, must consider: (1) the function of vocal articulation and of writing as phenomena of bodily movement; and (2) the sensational, ideational, and other antecedents or stimuli provocative of these exhibitions of complex motorial co-ordination; and (3) the development of the function of speech. I shall limit the inquiries at the outset to vocal speech, considering the faculty of writing as subsidiary and as following upon the analysis that I shall now proceed to make of the relationship of speech to other forms of expression, and of its mental and physical antecedents.

Vocal speech must be regarded as one, and that the most important, mode of expression. In addition to words, articulate and written, the human being expresses his thoughts and feelings by gesture, by pantomime, of which the conventional signs of the manual language

taught to deafmutes is a variety, or by means of inarticulate cries. A wave of the hand, a contraction of the musculature of the face, the expanding or contracting aperture of the eye, the cry of pain, the start of fright, are all manifestations or expressions of bodily condition. The most highly developed and most exact form of expression is found in words; but the individual who says, "I am angry," has given expression, it is true, to a state of mind in set phrases which require for their exclamation a much more far-reaching examination of nervous and mental processes than does the note of anguish; but nevertheless the two present analogies, and we may safely consider the former as a development and outgrowth from the latter simpler mode of mental expression.

It must be remembered that these externalizations or expressions, whatever they may be, are manifestations of the reaction of the individual organism to its environment. So far as concerns our present analysis the environment acts upon the individual through his sense organs. In this preliminary treatment, I shall consider only the group of sensations that are known as the visual, the auditory, and the kinæsthetic.

A stimulus acting upon sense organs tends to the production of bodily changes. These bodily changes to a certain extent depend in character upon the nature of the stimulus. The organism reacts with different bodily movements to a strong and to a weak light. It makes differential responses to the color red and to the color blue. The organism of the child will react differently to the objects of its environment than will that of the man. In the life of the individual, the reacting organism is there-

fore modified so that certain differences in the resultant responses to stimuli must be considered as dependent upon its stage of ontogenesis. Again, the reacting organism of the human being responds in a different manner to the same stimuli of the environment that act upon it than does that of a dog or a pigeon, a lizard or an amœba; in other words, the character of the reacting organism is dependent upon its stage of phylogenesis. Biology is the science to which we must have recourse for an explanation of these variations of type. Again, the sight of the bottle produces upon the hungry child different responses from those brought out by the same object acting upon a child whose appetite is satisfied. Thus the organism at every stage of phylogenesis and ontogenesis is subject to characteristic modifications which manifest themselves in the differences of reaction. In substance, therefore, the expressive movements of the organism made in response to external excitation are subject to variations that are referable in part to differences in the stimuli, the stage of organism development, and to temporary changes in its condition. The nervous system is the centre for the production of these reactions; certain parts of it seem to be of greater importance than others. In the human being the cerebral cortex is the portion of the nervous system which is chiefly modified by successive ontogenetic variations. It is that portion, consequently, which is most educated in the lifetime of an individual, and in an adult its destruction in whole or in part is fraught with the most serious consequences to his mental life. The cerebral cortex enregisters, perhaps as changes of its substance, effects of past stimuli. These

have been called by the name of memory residua. Without such memory residua and the association or grouping of past experiences with present experiences, modification of the organism would be impossible. Therefore, if a visual stimulus acts upon the eye, and produces some bodily change, the result of that stimulus is to produce some mark or trait which is left upon the cells of the cerebral cortex. If after innumerable stimulations the organism of the child reacts to the face of its mother in a manner different from that which it did in the first few days or months of its existence, it does so because that same visual stimulus now acts upon a brain which has been modified to such an extent that past visual stimuli of a similar nature are evoked simultaneously, and together with them are auditory and kinæsthetic impressions and recollections of bodily well-being, such as being fed, and so on. Although all parts of the organism respond more or less intensely to every excitation, still certain portions of the body and, more narrowly, certain regions of the cerebral cortex seem to be foci of the bodily disturbance evoked by a stimulus, and loci for those modifications that we have called memory residua. Thus, there are centres for visual impression locally differentiated from those of auditory impression, and these two again locally differentiated from those of muscular impression.

The simplest mental consequence of the stimulation of a sense organ by an external object may be assumed to be the arousal of a sensation. A more complex resultant is the arousal of a perception of an external object. The flower *violet* may call up a simple sensation of color, or it

may arouse in the mind the perception of an object of certain form, color, odor, taste, and even of certain habitat. The arousal of a perception requires the conjoint action or simultaneous association of disparate sensation groups. To perceive the purple violet requires the conjoint action of kinæsthetic, visual, tactile, and olfactory sensory groups. To perceive *bell* requires most of these, with the addition of the auditory group. If we, without the stimulation of a sense organ, call up in mind the flower *violet*, we evoke through central excitation the same sensory group. To ideate requires the successive and simultaneous grouping of these after-images of perception, the so-called memory images. No one can carry on a train of thought for any length of time without being aware of the sensory origin and basis of the contents of most of his ideas. In some persons one group predominates in initiating in the zone of language the process that results in internal or external speech. If the auditory group predominates, such a person may be called an *auditif*, it being understood that the auditory images are relatively and not absolutely predominant. For instance, if I am an *auditif*, and in my mind I fail to observe the visual and articulatory elements, it is not because these are not acting at all, but because they are not acting with sufficient intensity to arouse consciousness or to stimulate the frontal lobes to activity, while the auditory images are acting with sufficient intensity and stimulatability. In a similar way, one may be a *visuel*, an *articulomoteur*, etc. The latter individual is fairly well represented by the person who has to think aloud, and who, as he thinks, has to articulate that upon which he cogitates. Ability to reason, to

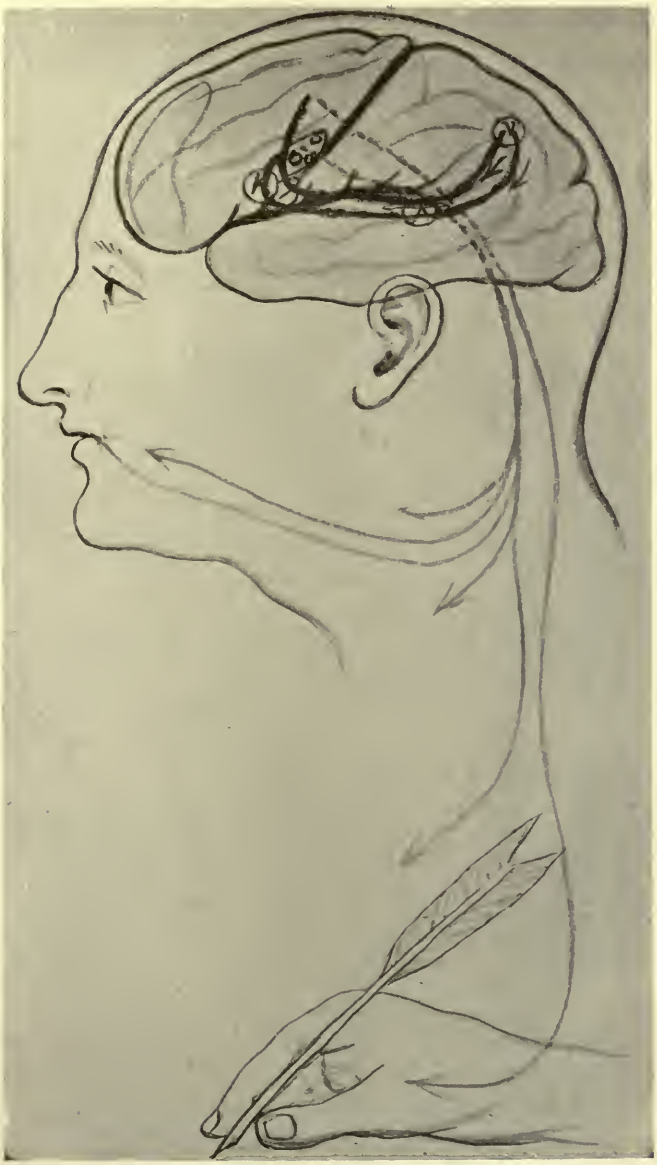

FIG. 1.—Diagram of Speech Centres and their Interconnection.

carry on a train of abstract thought, to recognize, and to remember, are dependent for their full preservation and for their development upon the integrity of these several sensory systems. It is probable, however, that the subtle and complex co-ordinations requisite for the processes of abstract thought are dependent upon ganglion cells having a different location, whose activity is superadded to that of those which are functional in the less complicated processes of sensory perception.

There are many facts, biological, physiological, anatomical, and pathological, which point to the frontal lobes as the seat of those neural processes whose function subserves the purposes of a physical basis for the higher or more complex processes of the mind; and although it is believed by the writer that there is no special centre of attention, or of thought, or of apperception, or of morality, in the frontal lobes, it can be, nevertheless, truthfully maintained that the integrity of the frontal lobes is essential to the full exercise of the human being's powers of complex thought.

If we designate (Fig. 1) by *V* the centre for visual memories, by *A* the centre for auditory memories, and by *K* the centre for kinæsthetic memories, we may connect these three centres by a line which will indicate, first, that for full and complete perception the integrity of these three centres is essential, so that, even if a stimulus reaches the brain through the eye, it will not evoke perception unless it succeeds in passing through appropriate channels from the visual to the auditory and to the kinæsthetic centres. Let us further represent by *C* a hypothetical centre or area for conception; we shall then draw

FIG. 2.—Diagram of Speech Centres and Conception Area.

a line from *C* to *V*, another from *C* to *A*, and another from *C* to *K* (Fig. 2). In ideation it is probable that all of these centres, *C*, *V*, *A*, and *K*, are co-ordinated in action, and that the various halves must be unbroken. In a case of visual conception, that is, if full recognition of an object that is seen is to be had, it is most important that the tract from *V* to *C* be kept open; and for an auditory recognition, that the tract from *A* to *C* be kept open. It will be observed that we make no sharp distinction between the centre for ideas and the centre for visual impression. They are not in reality separated, as represented here for purposes of demonstration, with fibres connecting them. The centres are rather to be looked upon as the end of a series, the abstract idea being at one end and the simple sensation at the other. I might illustrate it better by comparing it to the waves in a pond into which a stone has been cast. At the centre of the irradiating waves is the simple sensation; the waves farthest from the centre represents the concept or abstract ideas. For convenience' sake, and in nowise to be considered a definite centre, we let the letter *C* represent the area for the formation of concepts, and *V*, *A*, and *K* represent respectively the centres for visual, auditory, and kinæsthetic perceptions. We shall now add, to complete the figure, other points along the line of conduction to these several centres. Adjacent to *V* (Fig. 3) we place, joined thereto by a line, the circle marked *P V*. This circle represents the primary visual centre, or the centre of simple visual sensation. From this tract run two others, *v*, *v*, and from them we lead by two tracts *V R*, which we shall assume to represent the retinæ.

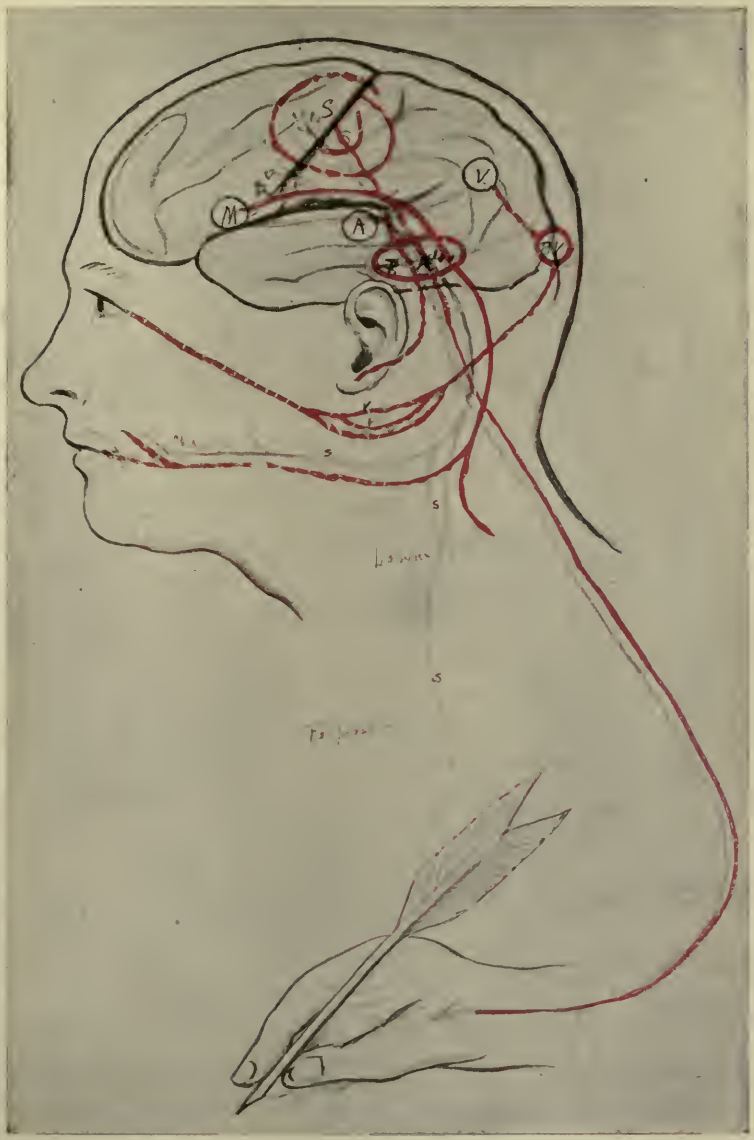

FIG. 3.—Diagram of Speech Centres and their Projection.

The intermediate *v* and *v* we assume to represent the basal ganglia. The construction is similar for the acoustic tracts and for the kinæsthetic tracts. If we now add to the figure a motor tract, the central portion represented by *S* and its termini by *s*, *s*, *s*, *s*, which are supposed to represent the several parts of the several nerves or neuraxons going to the musculature involved in articulatory speech, we have the figure complete. The muscles of speech are induced to contract by impulses that start from the lower end of the Rolandic area, to which impulses have been sent from the zone of language directly or indirectly from the higher intellectual areas, but always through the speech area. In other words, the frontal lobe cannot act on the part of the projection tract which functionates in externalizing speech, without first sending its impulses to the speech area. The impulses from the cortex go to the end of the primary motor neurons, that is, to the motor nuclei in the oblongata or in the basal ganglia.

I believe that the first cries of an infant are purely automatic, nearing reflex acts; the same, for instance, as respiration. Their occurrence is conditioned by peripheral stimuli which manifest their excitation in the pons, oblongata, and basal ganglia, in which lie the nuclei of origin of the cranial nerves innervating the musculature which must contract in order to produce crying-sounds. Such cries and sounds are produced as well by an anencephalic child as by a child with a cerebrum. In fact, at its birth a child is practically without a functioning cerebrum, and in this respect it is no better off for the time being than its anencephalic brother. Shortly after birth the brain

begins to mature, and it does so by the protons of the neurons developing myelin sheaths; in other words, by the medullation of fibres. The fibres passing to and from the brain develop myelin sheaths and functionability synchronously; in fact, the latter is entirely impossible without the former. The first paths to become medullated are those destined to carry sensory impulses. This is in fullest accord with psychological teachings that sensation conditions motion. Gradually the individual fibres of these sensory pathways become medullated one after another, the first being the olfactory and the last the auditory. The lateness of development of the auditory pathway has important ontogenetic significance and a direct bearing on the monitorship of auditory over other images, which is referred to in another chapter. If one studies the anatomy of the brain according to the developmental method, each sensory pathway can be traced distinctly to its termination, which is not diffuse, but limited to sharply circumscribed areas, such as witnesses the visual area, limited to the occipital cortex in the immediate vicinity of the calcarine fissure. These areas may be looked upon as internal sense organs, or as areas that mirror the activity of the peripheral sense organs. Destruction of them causes total destruction of the corresponding sense. Thus, destruction of the visual area produces blindness; destruction of the auditory area produces deafness; destruction of the somæsthetic area[1] produces loss of the bodily feeling. Each one of these strictly delimited areas is in relation with a higher area, which one may call an association area if he is satisfied with Flech-

[1] Flechsig's Körpergefühl-Area.

sig's nomenclature. In a portion, at least, of such areas are stored the memories of previous experiences, of previous functionings, and the materies of storage is called memory images. Before such memory images can be stored or enregistered they must be produced, and before they are produced the pathways that conduct the impulses must have been finished.

When the newborn child utters a scream or a shriek, it does so reflexly; the sound is neither an emotional nor a cognitive expression, and no anatomical structure above the brain ganglia is necessary. As it grows older and the brain ripens by the development of sensory pathways and of terminal areas that have already been mentioned, impulses begin to be conducted along these pathways. When the child screams after these pathways are developed, a kinæsthetic impulse going from the muscles that externalize the scream (which by the way are in part the muscles that externalize speech: the muscles of expiration and of laryngeal action) is sent along this pathway to the Rolandic-area cortex, and memories of it are stored up in an adjacent sensory area, the foot of the third frontal convolution. Later, when the auditory conducting fibres ripen and the auditory area becomes a reality, these impulses, likewise, are conducted to its area, in which they are enregistered as sounds, but not as differentiated sounds; for not until the child begins to develop intellectually does its auditory word area develop. Nevertheless, these autogenetic and slightly differentiated sounds, as well as heterogenetic sounds, are making their impress upon the general auditory field and are the outrunners to prepare it for the reception of articulate sounds, which

the child makes later in response to auditory and to visual stimuli.

It is not alone kinæsthetic memories that such inarticulate sounds store up in the speech area. The inco-ordinate contraction of muscles causes some change in the motor cells of the Rolandic cortex (to which the great central sensory pathway goes) which may have a decided bearing on the readiness with which these same peripheral muscles contract later in life in externalizing emotional and cognitive states when the child becomes possessed of consciousness and intellect.

Every contraction of the musculature of speech sends impulses along the tract *KK* (Fig. 3), which are carried to the sensory and percept centre and are there joined in simultaneous association or co-ordination with sensations or percepts that were aroused by the original stimulus passing up the tract *VV* or *AA*. At a later period, after these associations have become well fixed, we find inarticulate cries giving place to persistent articulated ones. We find, further, the beginning of speech imitation, which is probably due, in the first instance, to a stimulus that, passing from the tract *AA*, gives rise to a motor impulse running down the tract *SS*, which in turn causes such articulate co-ordination that the resultant vocal response is more or less an approximation to the sound that causes it. This vocal response is, of course, the source of two ingoing impulses, one up the tract *KK*, the other up the tract *AA*, both of them giving rise to appropriate sensations and perceptions which in the consciousness of the child supply the material for a comparison, and under the renewed stimulus of imitation lead to a

constantly nearer approximation to the original sound. It is not necessary for us to regard the centres that in this diagram are represented as being located within the cerebral cortex as taking part in the production of this responsive adaptation. The action is probably more or less reflex; it is a condition of echolalia, and the cross-connection between the sensory and motor tracts is probably made very low down, certainly not above the basal ganglia. At the same period of the development of the child we find evidence of visual imitation as the child copies the expressions, gestures, and bodily positions of those about him: it points as they point, just as it enunciates the words which it hears them enunciate. This is accomplished through the reflex co-ordination of the visual with the appropriate mental tracts. Thus we have complex adaptive movements, gesture and speech, provoked through the instinct of imitation, due to the constitution of the reactive organism. They require no volitional or even ideational antecedents, and yet they gradually build up and develop perception, memory, and the association of these into what will be the framework of a system of conceptual ideation.

As a result of these processes, there are stored up in the kinæsthetic centre for articulation motor images that are the result of reflexly or automatically excited contractions in the musculature of articulation, which serve as memory images and which are the immediate mental antecedents of the words that are to be enunciated. The central motor speech mechanism consists therefore of a purely sensory centre for motor memory images, a centre that is built up as the result of receptive processes, and an emissive centre

motor in function but which has, so far as we know, no conscious accompaniment of its activity; which is, in short, a part of the general motor area of the brain, the part from which proceed motor impulses externalizing speech, which externalization is the product of definite muscular contraction. Next we have formed at the end of the acoustic tracts a general auditory area for the reception of all forms of acoustic phenomena. A definite part of this area is destined for the reception of sounds having precise and limited significance. These sounds are deposited there partly as the result of the words of others and partly as the result of the words of the individual himself, and constitute acoustic verbal images. As the reflex centres, so the cortical centres are probably closely bound together, and these connections are known as association tracts. At the same time there have been forming in the visual centre and in the general auditory centre visual perceptions and auditory perceptions related to the objects of the external world, and these again related to the motor, auditory, and articulatory verbal images that have been invariably associated with these visual and acoustic perceptions. Each one of these centres is in its turn connected with the area of conception in the frontal lobes, to which the zone of language (a designation used to indicate the part of the cortical surface which contains the speech centres or areas) always sends impulses with conscious accompaniment, and elements from each centre are inextricably interwoven with the processes of ideation.

The development of abstract thought is to a great degree dependent upon the development of language, internal

or external. The name of an abstract quality is frequently the only definite content of an idea. Many persons do their thinking in words. As I have mentioned before, it depends largely upon individual constitution whether it be in auditory verbal signs or in kinæsthetic verbal symbols.

We are now in a position to consider one or two points of special interest connected with the use of language. To see, recognize, and name an object, requires an intact visual-conduction tract extending from the retina through the external geniculate body, the anterior quadrigeminal body, and the pulvinar, to the cortex of the occipital lobes by means of the optic radiations, and thence to the left angular gyrus. In a general way, to see the object requires only that part of the visual mechanism extending to the cortex of the occipital lobe. To recognize and name it requires all or part of the remainder of the visual mechanism. The recognition and complete apprehension of the object requires, further, that the lower visual centre be in intact relationship with the centre of visual images, so that images of the object can be contrasted with past experiences. If there have been no such past experiences, then the object is seen but not recognized or named. In order that the use, function, and wider relations of the object may be recognized, the centre of visual images must be in intact relationship with the concept centre. To name the object apprehended, the concept centre must either be in unbroken connection with the kinæsthetic motor centre or with the auditory verbal centre, which last must then be in connection with the kinæsthetic verbal centre, or else the connection must be made through the

visual centre to the auditory and kinæsthetic, or to the kinæsthetic direct. It is highly probable that the first is nearest to the real condition. These connections being intact, with the connection between the visual and concept centre unbroken, we shall have the perception of the object, followed by the naming of it. Similar conditions obtain in naming objects which impress themselves upon the sensorium through the ear. It is true also of the understanding of spoken language. To hear and answer a question intelligently requires that the acoustic conducting tract to the auditory area and to the centre of auditory verbal images be intact, and that the connection with the concept centre be undestroyed. It requires next that the outgoing tract from the area of conception to the kinæsthetic verbal centre, or to the acoustic and thence to the kinæsthetic centre, be intact. This leads to one remark concerning spontaneous speech, which is understood to be speech induced by a train of ideas—an oration, for example. In delivering an oration we ordinarily find involved only the concept centre and its connection with the kinæsthetic and articulatory centre. But, nevertheless, in every instance the area of cognition calls up the complete formula of speech product. The train of ideas, however, is the vivid content of the mind, in virtue of which the auditory verbal portion functionates below the level of consciousness.

The Genesis of Percepts.

In considering the development of the functions of speech from the psychological point of view it is necessary to speak briefly of the constitution of percepts and of ideas, of their relation to sensation upon the one hand and to

memory images on the other, and, lastly, to make inquiry as to the significance of the verbal symbols for our ideas and percepts, and of the mode of their acquirement.

The percept of an object is formed of the collocation in our minds of different sensory impressions that have been aroused in consciousness through the action of the object upon the special organs of sense. Sensations are therefore the material out of which a percept is formed. All the sensations that are capable of being aroused by an object need not be present in consciousness at one time. In becoming acquainted with some object of the environment (let us suppose a bell) the object has appealed at different times to eye, ear, and touch; from these experiences of various senses we have formed our idea of the nature of a bell, its appearance, the sound that it will emit on being struck, and its hardness or resistance. If our past sensory experiences with the object *bell* have been well preserved in memory, impingement of one single quality of a bell on the consciousness, through one sense organ only, will be sufficient to arouse in our minds the total percept of *bell*, which will really be an aggregate of the present sensation and of the memories of the sensory impressions of the past, and to revivify every quality of a bell and all it stands for.

The sensations of hearing and seeing are the most important sources of perception, and are those upon which we depend principally to obtain our knowledge of the world about us and what goes on in it. The other sensations are used more for corroborative or supplementary information. The formation of a definite composite percept of an object is generally followed by giving to it a

name. The name usually indicates some quality of the object, and in the beginning the child calls the stove "hot," the dog "bow-wow," or it expresses a generality of occurrences that a single conventional expression does not even suggest, such as "baba," meaning not only sleep, but the paraphernalia with which mothers of modern civilization surround the transition from consciousness to unconsciousness in their children—the nurse, the rocking of the cradle, the lullaby, and all the other things that the child has been accustomed to at this time. In the very beginning it is probable that the names applied to things are entirely imitative and the association between the name and the thing comes only after the child's attention has been repeatedly called to it. The more frequently and persistently his attention is directed to it, the sooner does the child learn to associate the sound with the thing. This is well illustrated by the early acquisition and understanding of, that is, the application of, the words "papa" and "mama." These sounds are uttered by the mother, and, as they are easily imitatively produced, and as the mother repeats them persistently, the child very early learns to say them, and moreover early learns to use them but not discriminatingly, for in the beginning every man is "papa." In a very short time the sound of the word "papa" calls up in the child's mind the visual memory of its father, and this is accompanied by numerous expressions of joy. Thus we have association of ideas as the basis of intellectual process and the originator of purposive speech.

Every one has had opportunity to observe young children, busy with their toys, repeating over and over sounds

or words to which they attach no significance, and which, if addressed to them directly, call up no memory pictures in their minds of the objects of which they are the names. This condition of echolalia indicates the existence of an automatic speech mechanism in the production of these words. The word is produced because some sensory impression has been caught up and is influencing the emissive centres to activity. Each time that the child repeats the word, memories of a sensory and motor nature are being stored up and held in readiness to be associated with their proper percepts. Thus speech is the product of the revival of memory in one or more sensory and motor areas simultaneously, the two memories blending into a single consciousness. As I have said previously, before the proper name has been associated with the object, as, for example, the word dog with a barking animal, the child for a long time may associate some prominent sense impression which the dog makes on its auditory centre, namely, its bark, with the sight of the dog. In this condition it recalls the memory of dog and refers to it as "bow-wow." It is thus seen that the giving of a specific name to an object is a more complicated mental process, and therefore one more easily destroyed, than is the giving of expressive or suggestive designation to a sensory impression.

The name of an object, as well as the names of qualities of the object, is stored up in definite parts of the brain, and its retention by the brain constitutes a word memory or name memory of the object, which can be drawn upon for purposes of perception or thought or speech, as are other memories. The sound of a church

bell, which to an aborigine would call to mind no image of belfry or church building, no thought of the material from which it was cast, nor of its size, color, and shape, nor of the purpose of its ringing, and thus would bring to his mind no memory picture of all the environmental associates of the bell, starts a sequence of associations in the mind of one who has learned of a bell, which may be of religious worship, of death, of marriage, of public calamity or rejoicing, and along with them it calls up the name which he has heard others give to the object which is the immediate cause of the sensation.

If a normally developed adult person were to have that part of the auditory cortical areas destroyed in which are stored up the memory images of all percepts obtained through the sense of hearing, he would be unable to recognize the sound of the bell, as a bell, although the sensations which the ringing of it produced might be the same as before, and heard quite as acutely. His auditory centres have been reduced to the simple untutored state of the aborigine. An exactly analogous condition maintains for percepts that are formed through other sense organs, as, for example, through the eye, as in learning to read; and for the expression of language in motor visual form, for example, in writing.

An individual who has learned to speak transforms memory images of words and ideas into spoken or written or other forms of conventional language. To accomplish this there must have been stored up in the brain cortex, in the centres of vision, of audition, and of kinæsthetic perception, a large number of sense impressions which are the symbolic representatives of percepts. These come

from without and are first presented through the sense of hearing.

Although speech is not absolutely essential to thought, still it is probable that most persons find it necessary to use words internally in order to think. For some persons, at least, thinking is always accompanied by and dependent upon internal speech. A knowledge of the psychology and physiology of internal speech is very helpful to the understanding of some aspects of aphasia. Internal speech is dependent upon a revival of auditory, visual, and articulatory memories; its integrity depends upon the united action of these three centres, but that one which is most highly cultivated is revived most vividly. This special one in some cases reaches such a stage of development that it seems to overtop the others; for instance, some persons find it necessary in speaking to call up the visual images of the words, others depend largely upon the auditory, and still others upon the articulatory kinæsthetic images. Nevertheless, internal speech is imperfect when the speech centre considered of least importance in any given individual is in any way deranged. The first is the most common and the second least. As I have said before, Charcot and his followers refer to those who in speech depend upon visual memories as *visuels*, to those who depend upon auditory memories as *auditifs*, and to those who depend upon motor memories as *moteurs*. This division is fanciful and has but slight practical application as has before been suggested. I shall have occasion to revert to this point in the chapter on "Conception of Aphasia."

All of the centres entering into word production are

intimately connected with one another, and a variety of combinations is possible. With these combinations I shall not concern myself here.

Clinical experience teaches that occasionally visual images of letters and words are lost, while those for ciphers and other graphic symbols are preserved. It is necessary, therefore, to say a word here concerning the acquisition of such symbols. Ciphers and other forms of notation are, like words, symbols devised by mankind to represent certain definite values, which are used to facilitate human intercourse. As symbols they are very much less differentiated in their application than letters are, and unlike letters, which vary in their value with every varying combination and change of position, ciphers have given to them a very definite, limited, and unvarying value which remains unchanged, it matters not their combination. Moreover, they are not complex as words are, but may be looked upon as simple, unvarying substitutes for words. This graphicness of registration on the visual areas is shown most convincingly by a study of arithmetical prodigies. Such persons, as a rule, have well-defined intellectual limitations in a number of directions. In fact, many of them belong to the class of *idiots savans.* Such persons are enabled to perform the most stupendous arithmetical problems usually calling only for primitive methods of computation. When the method by which they accomplish this is analyzed, it is seen to be very simple and in keeping with the graphicness of representation of ciphers in the visual sphere. Mathematical prodigies are endowed with the capacity of enregistering ciphers so vividly that they can see them as distinctly in

their "mind's eye" as if the characters were written on paper before them, and they not infrequently explain their methods by saying they read off the figures as they see them in their mind.

It is not at all likely that there is a separate centre in which such symbols are stored; all the evidence bearing on this point tends to show that, like letters, memories of them are stored in the angular gyrus but that the registration is a simpler one. If this be so, it accounts for their preservation after letters are lost and for the greater readiness with which they are evoked.

Writing.

The faculty of writing is developed, like that of articulate speech, under stress of the impulse of imitation, the co-ordination being one between the visual centre and that part of the motor cortex from which impulses start to move the member that holds the pen, whether it be the hand or any other mobile part of the body which has acquired or is acquiring facility by practice in making the co-ordinated movements of writing. These movements of writing are all accompanied by the reception of kinæsthetic impulses that go to the somæsthetic area of the brain, and the renewal of these memories makes writing seem in those who have had long practice almost like an automatic act, but in every instance, it matters not how facile or apparently divorced from consciousness the act of writing may be, the physiological procedure consists of impulses passing through the same series of operations as it did in the beginning. It must be remembered that the motor tracts for writing and for speech are tracts for ex-

pression and that they are influenced by any conditions that influence the reacting organism.

The motor act of wielding a brush in painting or in portraying visual sensory images, whether imaginary or real, is done by a cortical area quite as specialized as that for writing. When the child learns to write, it does so, as has just been said, under the stress of imitation. It is first made to copy or to trace letters or words that have been put before it in orthodox script. Formerly the child was taught to trace or copy individual letters. Even now this method is by no means obsolete, which it richly deserves to be. It is gratifying to hear that very few of even the most "old-fashioned" schoolteachers require their charges to "print" words, *i.e.*, to copy them from type, a procedure that was very common in bygone days. Such inculcation was a striving for the antithesis of the correct method. Most modern pedagogues teach their young pupils to trace or copy syllables and words in the same way as they teach them to learn syllables and words instead of letters. Pedagogical experts are also maintaining as a matter of sound principle that writing with a pen should be taught relatively late in a child's educational career and should always be preceded by writing upon the blackboard with free arm movements from the shoulder, because the former movements being the result of more specialized centres, should be developed after and from the latter.

In the beginning, tracing and copying words and letters are a slow and tedious process, principally, however, on account of the awkwardness in holding and guiding the pen. If the child is left-handed the awkwardness is more evi-

dent, because schoolteachers, almost without exception, unless oriented to the contrary, insist upon having the child write with the right hand, notwithstanding the fact that the organism is left-handed. This the child soon does with the same facility as though he had been allowed to write with the left hand.

If the child copies or traces without understanding the letter or the word, the neural process is a very simple one, and all that is required is that the cortex of the brain shall mirror the letters and then the transcription of these visual images by the arm-hand motor area. If, however, the child is being taught to recognize the letters at the same time, which is usually the case, there are stored up in its higher visual centre memories which form part of the general concept of the letter or the word. After the child traces or copies letters for a time he acquires a visual memory image of them; he may and probably does at the same time acquire their names and significance. After this he no longer requires a copy before him to produce the letters; he copies the letter in fact out of his visual storehouse. In the beginning, before the images have been firmly embedded, it requires strict attention to evocate these imperfectly enregistered images, and the whole process is done slowly and laboriously. In fact, oftentimes some of the images cannot be evoked at all; then they are said to be forgotten. But any one who has studied a child who is learning to write knows that it has not wholly lost the memory of the word, and by putting before him a part of the recalcitrant letter he may succeed in revivifying the letter or the word of which it forms a part. For instance, a child who is asked to write the name

"Frank" may say, "I don't know how it begins," meaning that it cannot jut the first letter of the word with sufficient vividness into the zone of language, and thus to the concept centre. If, however, the perpendicular limb of the letter and the middle short horizontal stroke is made for it, the child may go on and complete the letter *F* and the remainder of the word. In this case the visual images are not thoroughly incorporated and need other than volitional stimulation to invoke them. When writing becomes an habitual act, the letters and words which one writes form an integral part of internal language, and all the images of words are evoked simultaneously. In such voluntary writing an impulse starting from *C* (Fig. 2) calls up the images in *V*, and these proceed to an area in the motor cortex at *S*, but in every instance there is reason to believe that this pathway is through *A* and *K*. Color is lent to this view by a study of writing in children and in those who learn to write late in life. The fact that the articulatory image of the word is called up is shown by the experience that a word is always uttered either articulately or to themselves immediately preceding its production. As writing becomes more automatic, *i.e.*, as one becomes more adept in writing, this phenomenon ceases entirely, particularly in those who have been instructed "to keep what they are doing to themselves" (a common admonition in schools, at least in New England country schools); in others the phenomenon of articulating without vocalizing the word is retained throughout life.

If one examines himself carefully and repeatedly while writing, he is cognizant of a peculiar feeling or sensation in the ears and throat. These correspond to the auditory

and articulatory evocation of the letters and words that he is about to write down. It is, I believe, quite the same in typewriters. I am sure this phenomenon occurs in my own case when I operate the writing-machine, which I do with rapidity and facility. If this is the case when one externalizes his own thoughts by means of the writing-machine, it is legitimate to inquire if the same process or phenomenon is gone through when one writes from dictation. Numerous interrogations of my stenographer, a very intelligent man, always bring the reply that in writing from dictation the words or the letters composing them are uttered audibly immediately preceding the striking of the keys, or this is preceded by a feeling of movement and tension in the ears and throat, which are the congeners of such outward word production. I would not be understood to say that this takes place in every instance. Indeed, it is highly probable that long practice does away with the necessity of conscious activity of the articulatory centres before the externalization of the letters and words by the tapping of the keys.

Thus we have a series of functions developing in succession: first, general expressive reactions; second, mimic reactions; third, articulatory speech; fourth, writing. In cases of disorder we should expect the last to be the least stable and the first to be the most stable, and our experience as physicians teaches us that this is what really occurs.

The following areas and centres have been considered: (1) The visual area, and in close relation to it the visual word centre. (2) The auditory area, and in close relation to it the auditory word centre. (3) The kinæsthetic

area, and in close relation to it the word-image centre, the motor-articulatory centre.

The position of these centres and areas and the constitution of the zone of language by the former, as well as its environmental relationships to the different parts of the brain, are pointed out in the chapter on "Conception of Aphasia." The relation of the primary visual centre, the visual centre for verbal images, the general auditory centre, and the auditory centre for verbal memories, and of the kinæsthetic articulatory centre to the conception centre are highly important. The centre in which are stored visual images is adjacent to the primary visual area. The centre in which are stored auditory word memories is in the upper part of the general auditory area, while the images of articulation are allocated to the third frontal convolution, immediately adjacent to the somæsthetic area, and to the motor speech area.

The destruction of any one of these centres or of the connecting links is liable to produce the disorder of speech known as aphasia. A consideration of the most frequent types of aphasia with reference to this analytical scheme of speech will show that some of the centres and their pathways are frequently the seat of lesion and others less frequently. It should be remembered (1) that the location of a lesion that will cause aphasia is largely influenced, as far as frequency of occurrence goes, by the blood supply of that region; and (2) that the amount of speech disturbance that a lesion in the same locality in different brains will produce will vary only slightly with the sensory basis of the ideation of the individual to whom the brain belongs.

To complete this analysis it will be necessary for us to take up briefly the various elements in visual perception, to consider (1) color sensations, (2) form, (3) dimensions, (4) distance, (5) relation to the environment. It will be necessary in the case of audition to distinguish between the hearing of noises, musical tones, and the hearing of articulated words; and to consider (1) rhythm, (2) pitch, (3) tone quality, (4) intensity, (5) sound sequence. On the motor side similar complications will be met with. In articulatory speech we can distinguish (1) the lips, (2) the tongue, (3) the soft palate, (4) the nose, (5) the larynx, (6) the lungs, (7) the diaphragm.

Visual Sensation.

In attempting to analyze the various elements in visual perception, I shall have to consider in the briefest possible manner the typical physiological and psychological factors of vision and the relation of physical and psychological stimuli to psycho-physiological conditions.

I have previously spoken of the component parts of the visual mechanism, and here I shall confine myself to an enumeration of the elements of the peripheral visual apparatus. They consist, 1, (*a*) of a neuro-epithelial percipient apparatus, and (*b*) a ganglionic and nerve-fibre conducting apparatus; 2, of a refractive apparatus; and, 3, of a muscular mechanism arranged to move the eyeballs in any direction which may best contribute to the placing of optical stimuli on the point of most acute perceptive power.

Next in importance to the neuro-epithelial percipient

apparatus is the muscular mechanism which not only moves the eyeballs but also changes the shape of the refractive media that project the rays of light on the retina. These muscular actions, some of which are continually being made during states of consciousness, and, to a limited extent during states of unconsciousness, produce afferent stimuli, the memories of which are stored up probably in the supramarginal gyrus, adjacent to or in the somæsthetic area to which they are directly sent. The evocation of kinæsthetic ocular memories plays a part in the proper perception of visual impulses and contributes to the interpretation of visual percepts.

The customary physical stimulant of vision is light, which is a wave motion of the ether. These wave motions occur with a rapidity varying from 400,000,000,000 to 900,000,000,000 per second. Other forms of stimuli than light may excite visual sensation. Such are mechanical and electrical stimuli. If one places the electrode carrying a galvanic current on each temple, the make and break of the current are accompanied by a flash of light.

The physical stimulus, luminous vibration, or wave motions of ether are said to act by decomposing the visual purple of the retina, which procedure indirectly sets up a commotion in the optic-nerve filaments, which are in connection with the cells over which the photo-chemical substance is spread, and the filaments conduct the commotion to the occipital cortex, where they are registered. This, at least, is the theory of physiologists and psychologists. No real proof can be offered of the existence of such a process. It may be said, indeed, that the part

played by the visual purple has been allotted too great importance, as vision continues after it is exhausted and is not lacking in animals devoid of it. Therefore I prefer to say that luminous waves excite the elements of the percipient apparatus, probably by photo-chemical means, which excitation is carried by the conducting apparatus along the optic nerves and their continuation to the occipital cortex. The registration in the cortex is not distributed in a haphazard manner. Certain parts receive impulses coming from different parts of the retina, so that visual impulses coming from one quadrant of the retina are registered in a definite and correspondent portion of the occipital visual area.

The three different substances assumed to be affected in a photo-chemical way by luminous waves are supposed to lie in different percipient elements, so that one of the primary colors excites only those percipient elements which contain the substance corresponding to that color. Mixed colors excite two or three sets of percipient elements in different degree.

Psychologically, the different qualities of visual sensation are known as color. Different color sensations are produced by the different colors of the spectrum, and these color sensations differ according to wave lengths, the longest waves being those at the red end of the spectrum and the shortest waves those at the blue. The luminous intensity of a color varies with the amplitude of vibration, but with an equal amplitude of vibration some colors are inherently more luminous than others. As the number of acoustic vibrations constitute the pitch, so the wave length or number of vibrations to a second consti-

tutes the color. The relationship existing between the intensity of color sensation and auditory phenomena is a close one, and constitutes the basis of color audition. Persons in whom this phenomenon takes place apparently get auditory excitation through the perception of certain colors. This is not a physiological interaction, however, but a psychological one, and is often met with in psychopathic states.

Each percipient element is not trained to vibrations of certain wave lengths, and therefore not to the reception of individual colors. The areas of the retina in which the cones and ganglion cells are few are relatively less sensitive to color perception, requiring for perception and excitation greater intensity or a stimulus covering a greater area.

Before a child acquires perception of space and form, it must acquire perception of color. It has been accepted on the teachings of Preyer and others that in so doing children follow such an unvariable formula that it may be looked upon as a law. First they learn the recognition of yellow, then of red, later of green, and very much later of blue; in fact, Preyer contends that the color blue may fail to be recognized until very late. Binet's and Baldwin's studies seem to disprove this temporal acquisition of color perception. The latter's experiments showed that the order of acquiring color perception was blue, white, red, green, brown. Much further experimentation and observation are needed on this point before it can be considered settled. The acquisition of color perception is supposed to be proportionate with the maturation of the photo-chemical substance, whatever that may be, in the per-

cipient membrane. As a matter of fact, the acquisition of visual perception of any kind is dependent upon the ripening of the optical neuron. The manner in which children acquire perception of color can, however, legitimately be explained by the fact that prismatic colors have different luminosities, yellow being the brightest, green and red next, and blue and violet the dullest; and it is not at all unlikely that children get perception of colors in the above order on account of these different luminosities.

Visual memories for form are the result of muscular co-ordinations made in the movements of the various components of the eye which have been utilized in getting an outline percept of the object, with the color sensations coming from the object contrasted with the latter by the conceptual sphere. The original motive of all ocular motions is the desire to perceive objects clearly, and to accomplish this we must of necessity have the perception of light and color, a conception of form and space, and the faculty of seeing things in the places where they are, which may be called the projection of perception. When a person is in possession of these three factors, he may be said to be in possession of the means for fullest visual orientation. It is this orientation visually of objects, animate or inanimate, symbolic or concrete, that enters as an element of the mind and as a most important factor in the evolution of thought communication.

Acoustic Sensations.

Acoustic sensations are produced by wave motions of elastic substances. Although the rapidity of these wave motions may vary within wide limits, a certain number

must take place within a second of time in order to produce the sensation of sound. Ordinary sounds, or clangs, are a series of component tones which, irrespective of their intensity, can be distinguished from one another by their tone quality.

The peripheral auditory apparatus consists essentially of three parts: (1) of a neuro-epithelial percipient apparatus, and a ganglionic and nerve-fibre and conducting apparatus—the organ of Corti, which has its origin in the cochlea; (2) of the tympanum and ossicles; and (3) of the meatus and the external ear. There is another part of the auditory nerve than the cochlear one, which takes its origin from the ampullæ of the semicircular canal, and which for this reason is called the vestibular nerve. It does not go to the auditory area; it has nothing to do with audition; it goes to the cerebellum and in all probability it is of paramount importance in contributing to the maintenance of equilibrium.

The meatus and the membrana tympani serve as protectors to the terminals of the cochlear nerve. They act to prevent all stimuli, except those adequate and requisite to contribute auditory information, from acting on these terminals, and particularly from acting deleteriously on them. The tympanum through its tensor muscle is excited reflexly to contraction to meet these sounds and to prepare for them. The auditory nerves proper, that is, the cochlear divisions, have their eventual distribution in the superior temporal lobes on both sides of the brain in what is called the general auditory area. It is highly probable, although it cannot be absolutely so stated, that these nerves undergo complete decussation, the left nerve

going to the right auditory area, and the right nerve to the left auditory area.

The organ of Corti, a neure-epithelial structure, lies on an expanded membrane composed of fibres of different lengths. This membrane may be compared to a harp, each constituent of which is attuned to the various primitive sounds. These different lengths are connected with the elements of the organ of Corti, and it is probable that more than one are connected with the same element of the organ of Corti.

The periodic vibrations that the ear takes cognizance of may be considered grossly as of two kinds: (1) musical sounds or tones, and (2) noises. Musical sounds are periodic vibrations, while noises are irregular. The human voice, which may produce both noises and musical tones, may be taken as an example of these two forms. Musical sounds or clangs are very complex in their constitution, but they can be separated into simple acoustic elements or tones. When musical sounds reach the human ear they are analyzed into component tones by the membrane covering the organ of Corti, and this communicates the commotion to the individual organ of Corti, which is represented in the auditory area in the temporo-sphenoidal lobes.

The characteristics of auditory perceptions that must be considered are the intensity, the pitch, and the timbre. The intensity of a sound depends upon the amplitude of the vibration. The pitch or tone quality depends upon the number of vibrations; the timbre depends upon the number and relative dominance of the constituent tones.

Individuals vary greatly in their discrimination of pitch,

and on this discrimination depends the musical capacity of the individual. When a person cannot distinguish tones separated by an interval corresponding to a semitone he is tone deaf. We shall see later on that this condition may be caused by lesion of the auditory area, and probably as well by lesion of the right temporo-sphenoidal lobe as by lesion of the left.

Each nerve end of the cochlear nerve can be excited by only one pitch, or at least by a very limited number of pitches. The same stimulus of sound does not act at the same time upon many nerve ends, but upon one or a few neighboring terminations, and each nerve fibre of the organ of Corti becomes accustomed and especially sensitive to a certain pitch. This necessitates, for the fullest effect of sounds, the condition known as sound sequence, as there is no special sequence in the arrangement of several tones heard simultaneously. This, in connection with the fact that the sensations of sound are not projected into space with any exactness or with any differentiation, although some assume, without furnishing tangible proof, that there exists an auditory field that can in a way be compared to the visual field, necessitates a mechanism of great selective or differentiating capacity. The only accessory means of determining the locality from which sounds proceed is by turning the head, as the human ear lacks a muscular apparatus especially contrived to put the trumpet external ear in different positions to aid the localization. Thus no kinæsthetic sensations of any import, except those connected with the tensor tympani muscles, enters into the auditory memories, nor are any sent to the general somæsthetic area.

Articulation.

In the analysis of the mechanism of articulatory speech, I must discuss, first, the part played by respiration, or, to be more specific, by expiration; the parts concerned in phonation; and the parts concerned in articulation, using the word in its limited sense. The expiratory current is the absolute *sine qua non* of phonation, and the following characters of expiration have to be considered, viz., the rapidity, the rhythm, and the force. Expiration, as well as the entire respiratory act, is a reflex act which is often described as an automatic act. It is not automatic in the strict sense of the term. Its integrity is vouched for by a centre consisting of two parts, an inspiratory and an expiratory, situated in the oblongata, and the functional activity of this centre stands in very close relationship to the state of the blood. This is shown by the overactivity of the centre in cases of anæmia and by the cessation of activity in cases of superoxidation.

The respiratory function is, nevertheless, under the direct influence of the will, and can, within certain limits, be subject to considerable change, and particularly it can be influenced in its rapidity, rhythm, and force. Consideration of this fact leads one to believe that there is a distinct representation for the thoracic and laryngeal respiratory movements in the motor cortex, and that this area can be stimulated to activity by impulses coming from the higher intellectual area, either directly by force of the will, or indirectly, as by fear and joyful emotions. There is some experimental evidence to show the location of

this respiratory centre to be in the upper part of the lower third of the ascending frontal convolution. That is, it is adjacent to the centres for movement of the lips, tongue, and larynx, in the lower part of the Rolandic region.

The expiratory current is necessary for all phonic phenomena and for articulation, and in fact they are dependent upon it. This can be easily demonstrated if one attempts to articulate or phonate at the end of a full inspiration. Not a tone, not an articulate sound can be produced until the lungs have taken some air in that can go out in expiration. The degree of perfection of articulate speech depends upon the rapidity, rhythm, and force of expiration, as witness the phonation and articulation of a patient suffering with dyspnœa or with marked asthenia, such as that accompanying Asiatic cholera. In the latter the patient is as toneless and as inarticulate as if he had a tracheotomy tube in the windpipe. Yet such patients are in full possession of their mental faculties, including that of internal language, if the statements of those who recover can be relied upon.

The expiratory current is the motor power that sets the vocal cords into vibration. The vocal cords may be considered simple bands of fibro-elastic tissue, having a marvellously arranged muscular mechanism which is capable of putting them in different states of tension and of relaxation. The expiratory current setting the cords in motion originates all the different series of air waves of which the voice is composed. After these series of air waves have been started by the cords they are modified by the resonance cavities. These resonance cavities are controlled by

the muscles surrounding them, the principal ones of which are those of the pharynx, mouth, and nose. They reinforce the air waves set up by the vocal cords and thus give character to the tone. The tone as it leaves the cords is a mere squawk, and would always remain so were it not for the resonance cavities. Moreover, without resonance cavities we could not articulate.

The vocal cords originate several different tones, probably eight or ten, all the time, and it is the blending of these different tones that constitutes the "timbre" of the voice.

The number and relative strength of these different partial tones determine the quality of the tone, and here it may be said that the different vowel sounds are simply changes in quality of the tone. This is brought about by changing the size and shape of the resonance cavities, so that they reinforce some of the tones started by the cord and suppress others. For example, the vowel *a*, as in *father*, in a good voice may have ten partial tones, with the lowest pitch strongest and the tones gradually diminishing in strength as they rise in pitch. There must be a certain position of the tongue, lips, and soft palate to produce this result. These positions of the lips, tongue, and palate in the same individual would always give the same combination of partial tones, and this combination of partial tones would always give the vowel *a*. To articulate the vowel *e* there must be a change in position of these parts. In the vowel *a* the tongue is in a low position in the mouth, giving a large resonant space there. In the vowel *e* the tongue is raised so that it fills the mouth cavity, thus destroying it as a resonance chamber. The

effect is to suppress all the tones except the first four, and of these the second is strongly reinforced. For the long *a* we have another position of the articulating muscles, and hence another combination of partial tones, and so on through the whole list of vowels. The consonants, on the other hand, are merely interruptions of the vowel sounds produced by the use of the tongue and the lips. Without the expiratory currents we could not produce the sound, and without the resonance cavities we could not articulate, as they are the only means by which we change the character of the sound, and, after all, that is what articulation is in reality.

The degree of perfection of articulate speech depends largely on the use made of the resonance cavities; in other words, the use of the muscles that change their size and shape. The force of the expiratory current affects the intensity of the tone, and the intensity of the tone is determined by the height of the air waves of which the tone is composed. The altitude of the air waves depends, first, upon the extent of motion of the vocal cords. It is not thought necessary here to enumerate the factors that affect the pitch of the tone, or to describe the variations that such causative factors may undergo by the contraction of the muscles which control the activities of the cord. Sufficient, it is believed, has been said to show that a number of composite factors enter into the production of articulate speech as a mode of externalizing thought. It will be seen that the essential element in them all is one conditioned by muscular action: expiration by action of the diaphragm and other expiratory muscles, tension of the vocal cords by the intrinsic musculature of the larynx,

change in the shape and size of the resonance cavities by the muscles which enter largely into the constitution of surrounding parts. It will be seen, moreover, that three different sets or groups of muscles, or possibly four, partake of this composite act: the respiratory, the laryngeal, the lingual, and the buccal. Each set or group of these muscles has a definite representation in the Rolandic area of the cortex, specifically in the inferior part of the ascending frontal convolution, immediately adjacent to the third frontal convolution. From experimental and pathological evidence it is believed that the representation for each of these muscles is very strictly defined.

All the phenomena constituting articulation are under the direct control of the will. When consciousness is externalized in words of different phonic and articulatory characteristics the words are framed in the zone of language, and appropriate stimuli are sent to the cortical motor area in which are represented the parts concerned in phonation and articulation. The conscious impulse that is responsible for their being starts also impulses that modify them and give them the qualities that have just been enumerated. There exists between the cortical speech mechanism and the peripheral speech mechanism an intervening mechanism whose function is to regulate and co-ordinate the impulses coming from above. This may be considered a subsidiary station, and it is situated at the level where the peripheral axones of those neurons of the cortico-motor tract whose function it is to carry motor and articulatory impulses undergo end arborization, *i.e.*, in the pons-oblongata. Whether this subsidiary

station has all to do in co-ordinating and regulating the different elements of articulatory impulses cannot at the present time be said. It is thought, however, that it is very largely concerned in this function.

The child in learning articulate speech brings into play a very composite motor mechanism. In the beginning, before the impulses starting from the cortex are properly co-ordinated, and before the child has learned how to use its external speech apparatus, articulation may be defective in many ways. As soon, however, as the co-ordinating centre for these cortical impulses performs its function in the intended manner the externalization of speech becomes perfect.

Although I have no intention of discussing in this chapter the dissolution of speech, but propose to confine myself entirely to the genesis of speech, it may be well to call attention to the completeness with which externalization of speech can be lost by a destruction of that part of the cortex in which are located the motor centres for the respiration, the larynx, and the resonance mechanism. Such a destruction may constitute, as we shall see later, the lesion of subcortical motor aphasia, a lesion that produces as complete aphemia, without in any way interfering with internal speech, as if the patient were devoid of an external speech mechanism. It must also be seen, I think, that a lesion that severs all the projection fibres coming from these areas, it matters not at what level—as far, in fact, as the termination of the primary motor neuron—would produce exactly the same result. As a matter of experience, we have learned that a subcortical lesion, to cause complete aphasia, must be situated very close to the

cortex, and the reason for this is not far to seek. As far as possible, nature attempts to make up for its apparent shortcomings by substitution, and, as it is a necessity to send all the motor projection tracts through an extremely small space, the internal capsule at the base of the brain (Fig. 4), it is probable that it does not send the projection fibres coming from the various articulatory parts of the cortex in which are situated the representation of respiration, laryngeal, buccal, and labial movements in a direct way from the centre to the subsidiary station through the same part of the capsule. Therefore a lesion, unless it is sufficiently great to cut across the internal capsule, would not at the level of the latter produce *aphemia*, but would, rather, produce a dysarthric disturbance of speech. As lesions so extensive as to cut across the internal capsule are rarely consistent with life, we do not meet with this form of subcortical aphasia.

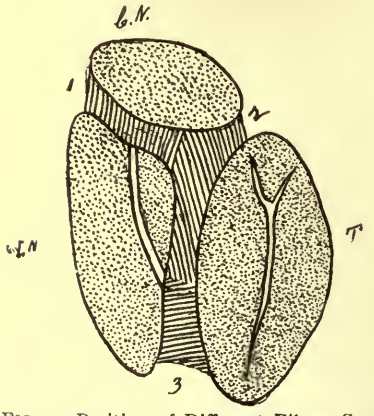

FIG. 4.—Position of Different Fibres Constituting the Internal Capsule, after Van Gehuchten. 1, Cortico-thalamic; 2, mixed motor fibres from the cortico-protuberantial fibres; 3, sensory fibres; *CN*, caudate nucleus; *T*, thalamus; *LN*, lenticular nucleus.

A few sentences devoted to the word as a phonetic phenomenon and we must take leave of this part of the analysis. A word varies phonetically according to the language that it is a constituent of. In the languages of civilized people it is an acoustic phenomenon, phoneti-

cally differentiated, to which such peoples have given a definite value as an expressive sign. In the language of some uncivilized peoples, such as the Chinese, the value of each sound or word varies with the intonation, and a study of aphasia in a Chinese subject would be of great service in illuminating the part played by expiration as a psycho-motor act, for this conditions intonation.

CHAPTER IV.

Conception of Aphasia.

1. *Remarks on the Anatomy of the Brain.* 2. *Zone of Language. Site of Revival of Words in Silent Thought.* 3. *Evidence in Favor of and against the Existence of a Special Graphic Motor Centre.*

Before outlining the conception of aphasia which I venture to believe is the one most in accord with anatomical, physiological, psychological, and pathological evidence, and before discussing the theories or ideas of aphasia that have held sway in the past, as well as those taught to-day, I shall outline as briefly as possible the parts of the brain concerned in the faculty of speech. To do this understandingly it is necessary to take a survey of the architecture and anatomy of the brain in general, and particularly to describe explicitly the connections of the different parts or centres that are known as speech centres.

No one will dispute that the cerebral hemispheres are necessary for the maintenance of the faculty of speech. No one will be likely to contend, having in mind the studies of Goltz, that the cerebral hemispheres are necessary for the maintenance of life in the higher animals. It is well known that this investigator was able to keep a dog alive for a considerable time after he had completely extirpated both cerebral hemispheres. The dog was

devoid of memory and judgment, and incapable of finding out for himself the things necessary for the satisfaction of his needs; nevertheless, he could stand and run, and he responded to direct stimuli, such as a blow, light, noise, etc. When he was deprived of nutrition the whole body was agitated, and when he had eaten abundantly he became content and showed evident signs of satisfaction. That absence of the cerebral cortex is consistent with life is shown by many cases of anencephalic monsters whose histories have been recorded. Moreover, it has been shown by Flechsig that the infant at birth, and for a short time after, is practically in the same condition as a child without cerebral hemispheres. The child is unable to make any conscious acts, all its movements and responses are reflex, and not until the ripening of the cortex begins by the development of myelin sheaths for each nerve fibre has the cortex any functioning ability. Flechsig has demonstrated beyond any question of doubt, and many other scientists have corroborated the demonstration, that the nerve fibres of the hemispheres develop their myelin sheaths at different periods of the infant's life, that those destined to function first are the first to develop myelin sheaths, while those destined to function later develop them at a different time. By means of this method of investigation the anatomist of Leipzig has shown that the fibres serving different functions can be followed with almost mathematical precision to the parts of the cortex in which they terminate, or from the parts of the cortex whence they go toward the periphery. This method of investigation has put him in possession of facts of such indisputability that it becomes necessary to re-

model, if not entirely to reconstruct, some previous teachings of the anatomy of the brain, and particularly of theories built in consonance with such teachings. Of such theories, that of aphasia is not one of the least important.

Flechsig's teachings of what may be called the physiological structure of the brain do not, however, clash materially with the present topographical teachings, although they point to the uselessness of dividing the cerebral hemispheres into a number of lobes and lobules for any other purpose than that of simple topographical orientation. Bearing this in mind, it may be said that the cortex of the brain is divided naturally by primary, principal, or complete fissures (using the term complete fissures in the sense of His) into the frontal, temporal, occipital, parietal, and insular lobes. The fissures by which such division is made are the fissure of Sylvius, the central fissure, and the parieto-occipital fissure. The fissure of Sylvius is the one that particularly concerns us now, for about it the so-called zone of language is built. The fissure of Sylvius is one of the first markings to show itself in the embryonic state. It appears at about the fourth week of intra-uterine life as a large depression on the external surface of the cerebral vesicle, or that part of it destined to be the forebrain. The forebrain grows and expands toward the front and backward, in fact in every direction except outward; consequently there is left a depression at the lateral surface which corresponds to the position of the corpus callosum internally. This depression constitutes the fossa of Sylvius (Fig. 5). As the brain continues to develop and to rotate on its axis, and the various parts of the hemispheres approximate one another, this

space becomes narrowed; the portion of the cortex lying at the bottom of it is covered over, and is known later as the island of Reil, while the prolongations of the space become very much narrowed and constitute the limbs of the fissure of Sylvius. This elongation of the fossa of Sylvius takes place at about the fourth month. On ac-

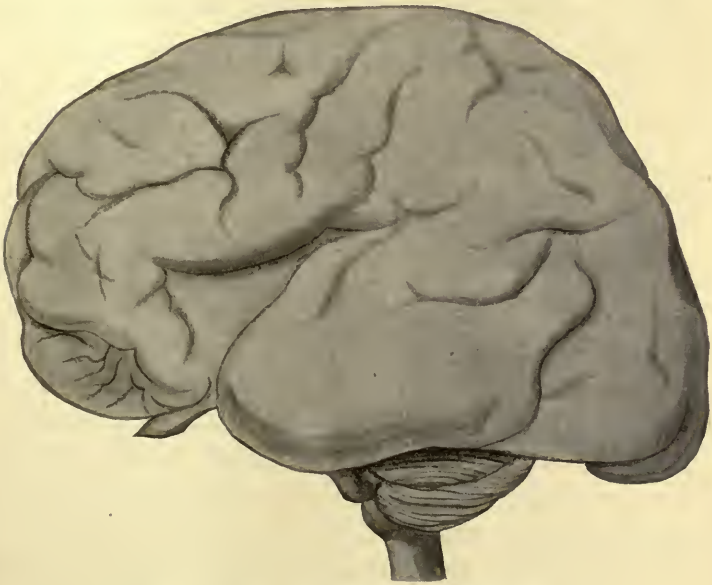

FIG. 5.—Triangular Fossa Sylvii (fœtal brain).

count of the disproportionate development of the occipital lobe, this elongation is directed obliquely upward and backward. The various parts bordering the fossa of Sylvius, developing, extend into the fossa as well as in other directions, and these interfossal projections form lids or opercula, which are designated frontal, parietal, and temporal, according to the lobe of which they are a part. Thus, when the fissure of Sylvius is completed, the parts of the

cortex that extend into the fossa of Sylvius, which it is to be remembered was originally a large triangular space, are known as opercula—the inferior operculum being formed by the anterior part of the temporal lobe, the superior operculum formed in part by the frontal and in part by the parietal, the anterior operculum, the shortest, constituted by the frontal lobe. When the operculum alone is mentioned, the superior operculum is always meant, for it is in reality the important one.

The fissure of Sylvius in the fully developed brain is a most striking feature. It starts from the fossa of Sylvius, which is just lateral to the anterior perforated space, passes forward and upward to the lateral surface of the brain, where it divides into a short anterior horizontal branch, and a posterior ascending branch which continues backward obliquely across the external surface of the cerebrum and terminates in a bifurcation near the middle of the lateral surface, this point being usually in the inferior parietal lobule. The posterior limb gives off immediately after the beginning of its oblique course a short anterior vertical branch, which juts into the inferior frontal convolution and cuts off a portion of the convolution which is known as the "foot." The Sylvian fissure separates the frontal and the parietal lobes above from the temporal lobe below. At its bottom is the most important annectant convolution of the hemisphere, the island of Reil.

The frontal convolutions are named superior, middle, and inferior, the latter alone being of interest to us now on account of the physiological importance attributed to it in the production of speech, or, to be more specific, I should say, attributed to the left inferior frontal convolu-

tion when the organism is right-handed, and to the right inferior frontal convolution when the organism is left-handed. This convolution is usually known as the convolution of Broca. It borders the fissure of Sylvius below, and posteriorly it is continuous with the ascending frontal convolution. The anterior horizontal limb of the fissure, and the much more insignificant branch of the fissure of Sylvius, the anterior vertical branch, jut into the convolution of Broca.

These branches of the Sylvian fissure divide the inferior frontal convolution into three parts:

1. The opercular part, or foot of the convolution of Broca, situated in front of the lower extremity of the anterior central convolution and comprised between the precentral fissure and the anterior vertical branch of the fissure of Sylvius.

2. The triangular part, sometimes called the cap of Broca's convolution, which is comprised between the anterior horizontal branch and the anterior vertical branch of the fissure of Sylvius.

3. The orbital part, which is situated below the anterior horizontal branch and which is continuous with the orbital convolution on the inferior surface of the hemisphere.

Of these three portions, the first alone is considered now to have any concern in the production of speech, and when the unmodified term, Broca's convolution, is used, this is the part that is referred to. It is the part of the brain in which are stored the sensory memories (articulatory-kinæsthetic) of a motor act—that of articulate speech.

The temporal convolutions, of which there are also three—superior, middle, and inferior—constitute the lateral

part of the hemisphere below the posterior limb of the fissure of Sylvius. The convolution immediately bordering the fissure of Sylvius, the superior temporal convolution, is the one to which is allocated the general auditory area. The auditory area is not spread over the entire external surface of the superior or first temporal convolution; a large part of it is hidden in the wall of the fossa of Sylvius, appearing on the external surface of the hemisphere only in the middle and posterior thirds of the convolution. There is some pathological evidence to indicate that musical memories have separate allotment in the left superior temporal convolution in the anterior part of the auditory area.[1]

The convolutions bordering on the fissure of Sylvius above are, after the inferior frontal convolution, of which we have already spoken, the central convolutions. These have nothing to do with the genesis of speech, but all to do with its execution. As we shall see later, they constitute almost exclusively the somæsthetic area and the cortical motor area. Next beyond these, bordering the Sylvian fissure, comes the inferior parietal convolution. It is believed that the higher visual centre is limited to the posterior portion of the inferior parietal lobule, which is known as the angular gyrus, and that the anterior portion, or supramarginal gyrus, has little or no function of storing up visual images. These gyri, the inferior frontal, the

[1] The only discordant testimony bearing on localization of the general auditory area is that of Schafer and Sanger Brown, whose experiments on monkeys did not lead them to substantiate Ferrier's statement that the function was allotted to the superior temporal convolution. This negative evidence of Schafer and Brown should not be given the slightest consideration, however, in view of the incontrovertible pathological testimony at our disposal, corroborative of this allocation.

superior temporal, and the inferior parietal, represent the confines of the zone of language, and it only remains necessary to speak of the relationship of the primary visual areas in the cunei, the tip of the occipital lobe, and especially on its internal surface immediately around the calcarine fissure. To this the radiations of the optic nerve can be directly traced. In the chapter on "Analysis of speech," I have said that the optic nerve develops its medullary sheath in the tenth month; that before this time it can be distinctly traced as a bundle of non-medullated fibres, passing from the eyeballs by means of the optic tract to the external geniculate body and from there to the anterior quadrigeminal body, while another bundle passes directly into the optic thalamus, and fibres go from the external geniculate body to the thalamus (see Fig. 6). The course of the optic nerve from these ganglia backward is directly to the cortex of the occipital lobes in the immediate vicinity of the calcarine fissure, the connection being effected by means of the so-called radiations of Gratiolet. In their course to the cuneus the optic radiations pass immediately subjacent to the angular gyrus and a lesion in the latter location, if it is not very superficial, is apt to interrupt some of these fibres. The pathways leading from both cunei to the left angular gyrus, the seat of visual memories, cannot be traced with the same exactitude as the tract to the primary visual areas can be.

The convolutions that have thus been described form the anatomical basis of the zone of language (see Fig. 7), and this definite part of the brain carries on the faculty of speech; or, expressed in another way, the centres neces-

sary for the reception and interpretation of impulses coming to the brain bearing on speech, and for the emission

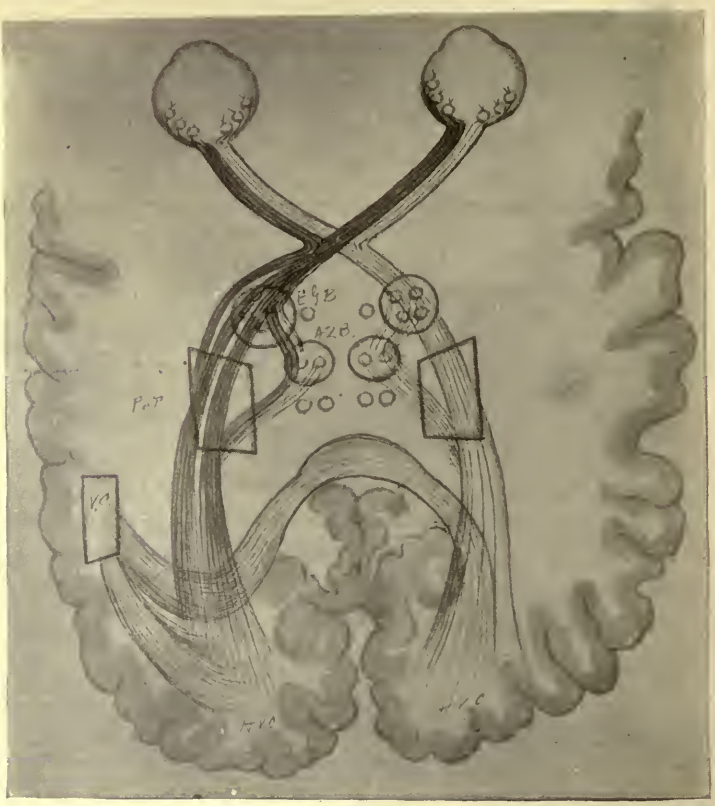

FIG. 6.—Course of the Optic Fibres. *EGB*, External geniculate body; *AQB*, anterior quadrigemineal body; *P of T*, pulvinar; *VC*, visual centre; *HVC*, half-vision centre.

of impulses to the peripheral speech mechanism, are situated in a definite area of the brain cortex. To this area, which is as much entitled to strict localization as the motor area, the name zone of language is given. Every one knows that the faculty of speech is maintained by

the left hemisphere of the brain in those who are right-handed, and by the right hemisphere in those who are left-handed. And this for the same genetic reason that the organism is right-handed in the majority of people and left-handed in the few; when the organism is left-handed, then the right hemisphere contains the zone of language. It would seem that this is one of the facts of physiology which is indisputable; nevertheless, every now and then some one brings forward evidence purporting to deny it. One of the most recent purveyors of such evidence is Moltschanow,[1] of Moscow, who in relating a case says that his observations were confirmed by Rossolimo. He cites the following instance in support of the claim that the speech area is not always on the left side of the brain when the organism is right-handed: A man fifty-six years of age, of alcoholic habits, had a stroke of apoplexy, followed by disturbance of speech two years before his entrance to the hospital, when the author discovered sharply defined "amnesic aphasia" with word deafness and total left-side hemiplegia. Recognizing that such an unusual combination as left-side hemiplegia and aphasia probably bespoke a left-handed man, the author questioned the patient's wife on many occasions to learn if the patient used the left hand by preference, but she insisted that he was right-handed. At the autopsy there were found two areas of softening in the right hemisphere, one in the posterior segment of the first temporal gyrus, the second in the posterior portion of the third frontal gyrus. Nothing abnormal was found in the left hemisphere. The

[1] Moltschanow: "Zur Frage von der Localisation des Sprachcentrums." Neurologisches Centralblatt, 1893, p. 673.

writer says that this case speaks unequivocally against what he is pleased to call Broca's dictum concerning the location of the speech area. He says further that other authors have probably had similar cases, for in looking over the literature of aphasia he had noted that it is oftentimes impossible to determine from the reported cases whether the patient was right-handed or left. This point the writer need have no difficulty, I believe, in determining in the future. If the patient whose history he reads has a left hemiplegia with aphasia, that patient is a right-handed man, and *vice versa.* It is almost incredible that physicians will attempt to convince themselves that evidence of this kind can have the slightest effect in overthrowing such an invariable rule as the one relating to the location of the speech area, just cited. An attempt to discredit a law so firmly established as this by the citation of testimony of a woman concerning the right-handedness or left-handedness of her husband is like trying to trip up Atlas by putting a microbe to obstruct his path. How is any one to know that I as an organism am left-handed? I write with the right hand; with it I throw, I use a knife to whittle; I have used it in the past to operate; in fact, every ordinary and extraordinary act calling for dexterity is performed *secundum artem* with the right hand. Yet I am left-handed in the sense of the term that originally the organism was intended to be served particularly by that hand, and were it not for the care taken in the nursery the left hand would be the dextrous member to-day. Therefore, it seems to me that unless those who come in the most intimate contact with me are informed to the contrary, I

should be considered a right-handed individual. And yet, no one, I think, would deny that, if such a person were to have a lesion on the right side of his brain involving the areas to which are allocated the functions of speech, he would have with it aphasia.

The determining causes of right-handedness is a problem in biology that I can refer to only very briefly. It is an extremely important one, because no one now doubts that dextrality is conditioned by the same factors that determine the unilaterality of executive-speech representation, and that the former is an evolutionary differentiation of the latter. It has been considered to be dependent upon tribal and social customs originally, and as an inherited characteristic. The most fanciful theories as well as the most careful scientific experiments have been undertaken to explain it. Of the former the one that crops up most irrepressibly in popular and in scientific literature is that right-handedness has to do with the way in which the child is held in its waking hours and the side on which it lies while it sleeps. This can be refuted so easily that it is scarcely worth the doing. If a child becomes right-handed because the nurse carries it so that the left arm is hampered from free movement, then left-handed nurses (who naturally carry their charges on the left arm and therefore keep the child's right arm close to them) could always be depended upon to bring up left-handed children; but as a matter of fact they cannot be. Among the serious theories that have been propounded for its explanation a few deserve specific mention. Ogle suggested that it was conditioned by peculiarities in the mode of origin and distribution of the left internal carotid artery, and Bastian

pointed out that right-handedness, or the conditions that determine it, has a definite relationship to the increased specific weight of the cortical gray matter of the left hemisphere and to greater convolutional complexity. Both of these conditions may be the consequence and not the cause of dextrality. Recently Baldwin has propounded a theory, based on careful and intelligent observation of his own child, that right-handedness is a manifestation of brain variation utilized for expansion which develops further into speech; that it is due to differences in the two half-brains reached at an early stage in life and that the promise of it is inherited.

It has been proven beyond cavil that destruction of the foot of the third frontal convolution causes inability to articulate words; that destruction of the superior temporal gyrus causes inability to understand spoken words, and a lesion in the inferior parietal gyrus causes inability to interpret words which can be seen. These are the three components that enter into the constitution of speech, and they are the three factors that are absolutely essential to the production of perfect speech. The convolutions of the brain in which they are situated are immediately adjacent to each other. They are intimately connected by means of annectant convolutions, the most important of which are the insular gyri, and by association tracts to be named presently. These convolutions border on the Sylvian fissure, and they are all supplied by branches of one blood-vessel, the middle cerebral or Sylvian artery, the direct continuation of the internal carotid artery.

The area thus mapped out is in shape somewhat like a gondola; the bottom being formed by the superior tem-

poral gyrus, the upturned hind end by the angular and supramarginal gyri, the front end, which does not rise so high as the other end, by the frontal operculum, the inferior end or foot of the third frontal convolution (Fig. 7). This area is not entirely occupied by the speech centres, by which is meant portions of the cortex in which are stored the visual, auditory, and kinæsthetic articula-

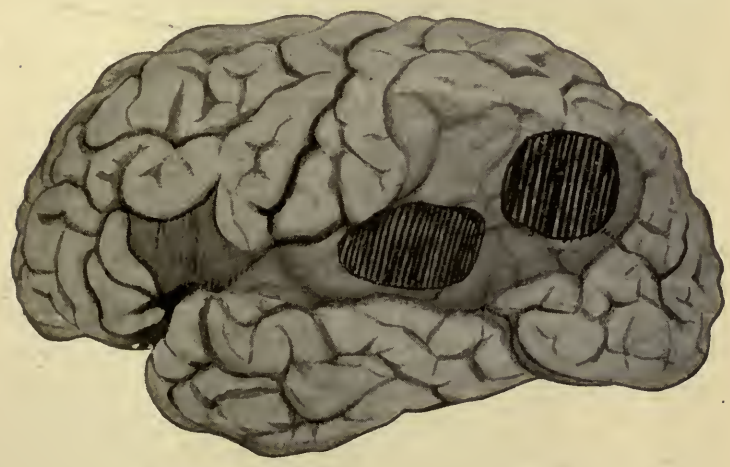

FIG. 7.—Zone of Language; Position of Centres.

tory memories. These centres, the storehouses of such memories, are narrowly confined (though not sharply delimited, in all probability), and the remainder of the zone of language, that is, the area between the centres, is constituted by association tracts or pathways connecting these centres.

The association or connecting pathways of the brain must now be discussed. It is well known that the nerve fibres of the brain are divided into three groups:

1. Commissural fibres, which unite symmetrical points

in the two cerebral hemispheres. These constitute the corpus callosum and the anterior white commissure or psalterium. They need not further concern us here.

2. The fibres of association; long and short fibres which unite different points in the same cerebral hemisphere.

3. Projection fibres; fibres that unite the cortical gray matter of a hemisphere with the gray substance of the brain stem and cerebro-spinal axis.

The association fibres of the brain are divided into the long and short fibres. The short fibres unite points in the cerebral hemisphere that are more or less adjacent, and they are found immediately beneath the cortical gray matter. The long fibres, on the other hand, unite distant parts of a hemisphere, and they are situated at some depth from the cortex. They are described as the (*a*) superior longitudinal fascicle, which unites the gray matter of the frontal lobe with the gray matter of the occipital and the temporal lobes; (*b*) the inferior longitudinal fascicle, made up of fibres that unite the occipital lobe and the tip of the temporal lobe; (*c*) the arciform fascicle or cingulum, which is made up of fibres going in an antero-posterior direction from the frontal to the temporal lobe (the fascicle gets its name from the fact that it bends to conform to the corpus callosum); (*d*) the uncinate fascicle, which connects the inferior frontal convolution with the tip of the temporal lobe immediately beneath the fissure of Sylvius. These association fibres, which are formed by the axones of the pyramidal cells in the cerebral cortex or their collateral branches, are shown graphically by the following diagram (Fig. 8), which, if contrasted with the diagram showing the zone of language, will represent graphically

the avenues of connection between the three speech centres.

All text-books on the anatomy of the brain, with the one exception of Van Gehuchten's, describe under the name of the cortico-protuberantial fascicle a bundle of nerve fibres which unites the cerebral cortex of the frontal lobe and of the occipital lobe with the gray masses constituting the nuclei of the pons. But the recent researches of Dejerine and of Flechsig have proven that the cortico-protu-

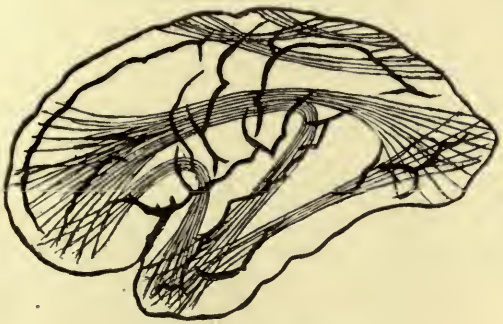

FIG. 8.—Association Fibres.

berantial fibres, instead of arising from the cortical substance of the frontal and of the occipito-temporal lobes, arise entirely from the gray masses which constitute the central convolutions, and the latter contends that no fibres from other parts of the cortex than those of the central lobe enter into the constitution of the pyramidal tracts. The extremely important bearing of this on our conception of aphasia will be seen later on. A second fact of not much less importance than the first, but one that is less generally accepted, is that heretofore it has been held by many authors that every region of the cere-

bral cortex in man was in connection with the inferior gray masses by fascicles of nerve fibres that were designated projection fibres.

Even so recent a writer on the subject of aphasia as Mirallié has described the inferior pediculo-frontal fascicle, which is considered by Pitres, by Raymond, by Brissaud, and by others to be of such paramount importance in explaining the symptomatology of motor aphasia. But to-day, in the light of the evidence furnished by Flechsig and upheld by such a brilliant and trustworthy anatomist as Van Gehuchten, the inferior pediculo-frontal fascicle must go, and with it the idea that the projection tracts arise from all or indefinite parts of the brain. It remained for Flechsig to prove, as he has done to the satisfaction of some neuropathologists, although not to that of others (Dejerine), that the projection fibres exist only for a third of the entire cerebral cortex, and that two-thirds of the cortex of the human brain has no connection whatsoever with the inferior nerve centres.

Flechsig has shown in his study of the embryonal and the postnatal cerebrum that the sensory paths are the ones which first become medullated. And of these sensory paths the first to ripen is that concerned in the sense of smell, and the last that which carries auditory impulses. By following these sensory paths to the cortex he has shown that the latter may be divided into four areas or spheres, to be enumerated presently. I shall make a somewhat detailed review of the recent statements of Flechsig regarding the localization of function in the cortical area. This author has divided the cortex, first of all, into two very distinct zones, the zone of the centres

of projection, or the sensory spheres, which take up about one-third of the whole cortical area; and the remainder of the cortex, which is devoid of fibres of projection, but which is united to the other sensory spheres by innumerable fibres of association, and to which are given the names of zones or centres of association.

FIG. 9.—Sensorial and Association Spheres, according to Flechsig. Dotted areas, sensorial; lines, association areas. External surface. *A*, Somæsthetic area; *B*, parietal association centre; *C*, visual area; *D*, occipito-temporal association centre; *E*, auditory area; *F*, island of Reil; *G*, frontal association centre.

The zones or centres of projection comprise, according to Flechsig, four sensorial spheres (Figs. 9 and 10):

1. *The tactile sphere, or somæsthetic area.* It occupies the entire region between the fossa Sylvii up to the corpus callosum, and includes the central convolutions, the paracentral lobule and the posterior part of the three frontal convolutions, and the midde third of the gyrus fornicatus.

This zone represents the cortical field in which terminate on either side the fibres of the median fillet which do not stop at the basal ganglia. These fibres carry into the cortex the impulses that are concerned in sensations of touch, pain, temperature, muscle and tendon sense, thirst, sexual sensations, and in short, all the sensations that

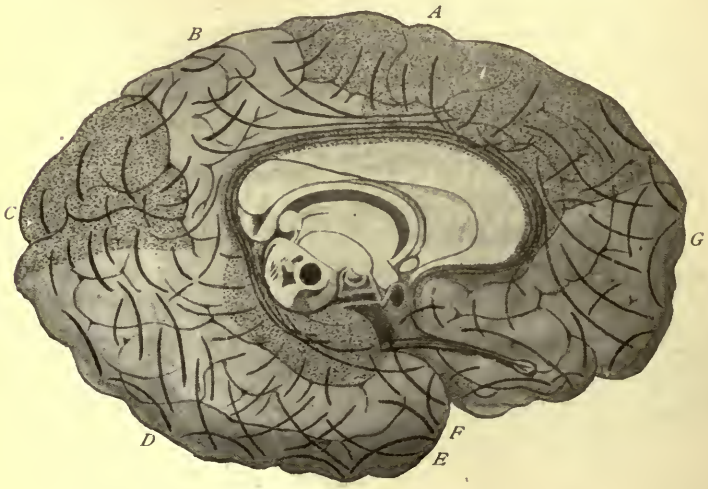

FIG. 10.—Internal Surface of Brain, after Flechsig. *A*, Somæsthetic area; *B*, parietal association centre; *C*, visual area; *D*, occipito-temporal association centre; *E*, gyrus hippocampus; *F*, olfactory area; *G*, frontal association centre.

arise from within the body and inform us as to the condition of the body. Therefore the name, somæsthetic area. This is the area in which are mirrored bodily sensations. In addition to being a sensory field, this area is also the great motor region from which nearly all the movements serving for the voluntary satisfaction of the bodily feelings and instincts seem to start. It is the area from which the impulse starts when a person voluntarily

breathes, speaks, swallows, chews, or does any purposive movement. The cells of origin of the fibres conducting these impulses are analogous to the giant pyramidal cells of the ventral horns of the spinal cord; the axones of these cells constitute the frontal cortico-protuberantial fascicle of Flechsig.

2. *The olfactory sphere.* This sphere is limited to the olfactory trigone and the adjacent part of the convolution of the corpus callosum, the anterior perforated space, the uncinate gyrus, which is in contact with the island of Reil, and the adjacent parts of the hippocampus.

3. *The visual sphere.* This is situated on the internal surface of each cerebral hemisphere, in the immediate neighborhood of the calcarine fissure, and fibres run from this tract into those adjacent areas of the occipital cortex which show the macroscopic stripe of Vicq d'Azyr. The centripetal fibres of the visual sphere come from the optic thalamus, the external geniculate body, and the anterior quadrigeminal body. Among these centripetal fibres, those that come from the external geniculate body terminate exclusively in the borders of the calcarine fissure, while those coming from the optic thalamus and the quadrigeminal tubercles terminate around this fissure. This distinction, according to Flechsig, is important, because the external geniculate bodies receive exclusively the fibres coming from the macula lutea.

4. *The auditory sphere.* The fibres communicating auditory impulses to the cerebral cortex form the lateral fillet in the pons and are connected largely with the median geniculate body. They terminate in the transverse temporal gyri, especially in the anterior one. This sphere

is united by centripetal fibres to the inferior quadrigeminal body and to the internal geniculate body, and by centrifugal fibres (the temporal cortico-protuberantial fascicle) with the nucleus of the pons. Flechsig considers these descending fibres as a motor pathway which has for its function the transmission of impulses arriving in the auditory sphere from the muscles that move the ear.

In addition to these projection fibres uniting the sensory spheres with the inferior gray masses of the brain axis, there exists in each centre of projection a number of fascicles, composed of centripetal and centrifugal fibres, uniting the centre with the corresponding part of the thalamus.

These centres of projection are in connection with all the peripheral organs by a double set of nerve fibres, one the ascending or centripetal fibres, which are sensory, the other a descending centrifugal set of fibres, which are motor. These two sets of fibres end in the same cortical region. According to this there is no exclusively motor cortex, no exclusively sensory. The sensory spheres are thus in reality the sensori-motor regions of the cerebral cortex. All the impressions received by the terminals of the peripheral nerves and their sensory fibres are conducted to the tactile sphere of the cerebral cortex by a series of centripetal neurons. These impressions are transmitted to the cells of origin of the descending or motor fibres of this same region, which then descend by a series of centrifugal neurons as far as the peripheral muscles. It is the same for the olfactory impressions, the visual, and the acoustic. They are transmitted by the

centripetal pathways to the corresponding sensory spheres. The centres of projection, considered by themselves, are completely separated from the centres of association which surround them, and constitute thus a sort of nervous centre for reflexes of cortical origin.

When these centres have become ripe, that is, when the fibres going to them and coming from them are medullated, only about one-third of the whole area of the cortex has been disposed of. Two-thirds of the cortex appears to have nothing to do with the periphery, but is reserved for other and apparently higher work. These areas are the so-called association centres of Flechsig. Before considering them it is well to draw attention to two statements of Flechsig regarding the projection areas.

The somæsthetic area is much more extensive than are all the other areas combined. This disproportion should not be a matter of surprise. In it are represented all the forms of tactile excitation that acquaint us with the outer world, and from it start all impulses by which are externalized thought, feelings, desires. The olfactory sphere is very little developed, because the sense of smell is so rudimentary. The auditory sphere and the visual sphere reproduce in the brain, the one the sensitive surface of the organ of Corti of the internal ear, and the other the retina. These last three spheres inform us of the external world and are dependent for their activity on sensation coming from without. The second point made by Flechsig is that the sensory spheres are localized around the primary fissures, the visual sphere around the calcarine fissure, the tactile sphere around the central fissure, the olfactory

sphere and the auditory sphere around the fissure of Sylvius.

The zone of the association centres is formed of three distinct centres: first, the posterior large association centre, which comprises a part of the lingual convolution, the fusiform convolution, all of the parietal convolutions, the inferior temporal convolution, and the anterior part of the external surface of the occipital lobe; second, the median association centre, which corresponds to the island of Reil; third, the anterior association centre, which is constituted by a portion of the superior frontal convolution and a large part of the median and third frontal convolutions. Flechsig believes that these association centres represent arrangements which unite the activities of the central internal sense organs and build them up to higher limits. Sensory impressions of different qualities, visual, auditory, tactile, olfactory, and gustatory, are united, or, at any rate, the anatomical mechanism is afforded for their union. These association centres are completely independent of the inferior gray masses, and are the portions of the cerebral cortex which above all others are concerned in the higher intellectual manifestations, judgment, memory, etc.

The middle association centre is represented by the cortex of the island of Reil. This is the centre which unites all the convolutional areas bordering the fossa of Sylvius, the integrity of which is indispensable for the preservation of language. It belongs in part to the somæsthetic area (especially to the region for the speech organs), partly to the auditory area, and partly to the olfactory area. The large posterior centre of association is

the part of the cortex which puts us *en rapport* with the external world. It is that which unites the superior centres for all the sensations, tactile, visual, and acoustic, that come to us from without.

Flechsig points out that the association areas, the convolutions which have no direct communication with the crus, central ganglia, or the corpus callosum, are those which are latest in the order of development, and on this ground alone might be supposed to be concerned in more strictly mental faculties, which are latest in their manifestations. These areas constitute a striking difference between the human cerebrum and that of lower animals. The centres of projection exist in the lower animals to a very much more highly developed extent than in man. But in man, on the other hand, the centres of association are immeasurably greater, while as we descend the scale of animal life the centres of association become fewer and finally disappear. It has been noted that the centres of association are often interpolated between the centres of projection, so as to separate these last centres completely one from another. It would seem to be in accord with the general plan of construction of the nervous system and with what we know of mental operations, that these convolutions which are withdrawn, so to speak, from direct relation with the outer world should be the seat of the more purely intellectual operations.

From a consideration of the morbid anatomy of some cerebral diseases, especially general paresis, Flechsig has come to the conclusion that the anterior association centre is the part that conditions consciousness of personality

(Persönlichkeitsbewusstsein). Lesion of this area causes, in a word, loss of the faculty of abstraction.

This extensive reference to the view of Flechsig has seemed to me fitting not only because it is a most important advance in the interpretation of the physiology of the brain, but also because it is of direct and signal service in the conception of aphasia as here outlined, although it is not in entire harmony with it. Moreover, it has a direct bearing on many problems in the morbid physiology of the brain that confront the physician to-day. In a way, Flechsig's contentions are not at all new. Views very similar to them were expounded by Broadbent nearly twenty-five years ago, when he pointed out that the centres which Flechsig calls "association centres" were parts of the cortex that were neither in direct relation with peduncular fibres nor with those of the corpus callosum. These association areas are also in reality what Bastian has described as "annexes of the perceptive centres."

Some of Flechsig's views, it seems to me, will be very slowly accepted, particularly those referable to the projection systems of fibres. Already Monaköw and Kölliker have taken serious exception to a number of his contentions and very recently Dejerine contests them.

Zone of Language — Site of the Revival of Words in Silent Thought.

The zone of language is a part of the brain in which are carried on the processes essential to the facultas synatrix. It is that part of the brain whose function is the

necessary material substrate of conception and of comprehension and expression.

By way of introduction, it may be said that when the expression "zone of language" is used I do not mean an area which can be mapped out on the surface of the brain by unvarying mathematical lines. Rather than have such interpretation put upon the use of the term, I should prefer to be understood to use it merely as an expression of convenience.

This speech area or zone of language is not, in all probability, strictly delimited. It varies in individual cases, and at different periods of life in the same individual, *i.e.*, it is subject to phylogenetic variation and to ontogenetic variation as well; the latter depending somewhat on the speech acquisition of the individual, and on the range and number of avenues by which he receives or has schooled himself to receive information of objects. This area is a receptive and an emissive centre for all forms of stimuli or excitations that reach it, and which its individual developmental metamorphosis has accustomed it to accept, to give tenancy to, and to elaborate into new forms of stimulation. It is receptive chiefly to auditory and visual stimuli, which it emits to other centres, and also to kinæsthetic, olfactory, and gustatory stimuli. It is emissive to the frontal lobes and to the cortex of the Rolandic region, from which start the motor projection tracts and by which all thought externalization is mediated.

The speech area or zone of language is an area made up of neurons, some of which send their axones into the Rolandic region and into the frontal regions of the brain, while others confine their distribution to the speech area

itself, and, as they do not pass outside of this area, they may be looked upon as intercentral neurons.

The zone of language has no projection fibres going directly into the motor projection tract; it sends no impulses directly to the projection tract which carries down neural impulses to be externalized as speech. On the contrary, the zone of language sends impulses composed, in the illiterate, of auditory and articulatory memories of the word, and, in the educated, of auditory, visual, and articulatory memories, to the Rolandic cortex and to particular areas of this region, depending on the manner in which the idea is to be externalized; that is, whether by spoken or written word or symbol, or by some form of mimetic or purposive action, which the judgment of the individual decides to be most serviceable in conveying the thought. If the idea is to be expressed by articulate speech, the impulses are sent to that area of the Rolandic region in which there is separate allocation for the movements of respiration, vocalization, lingual and labial action. This area is in the foot of the ascending central convolution, adjacent to the area in which are stored sensory memories of articulatory movements, Broca's area. From here the real motor impulses start. They go down through the motor projection tract, the axones of which *en masse* form the pyramidal tract, and the central motor projections of the cranial nerves, to the various muscles whose contraction produces articulate speech. There is reason to believe that these outgoing impulses are coordinated, given rhythm, force, and association, not in the cortex of the brain, but in stations situated in the brain ganglia, the cerebellum, and the pons-ob-

longata, the centres composing these stations acting automatically.

When the idea is externalized in writing, in complex movements such as mimetic movements, or by a simple nod of negation or affirmation, a simple movement of beckoning, the genesis of the symbol or the pantomime is exactly analogous to that of articulate speech. They are all the result of internal language, and the person who writes an impassioned editorial on some subject that fires his patriotic spirit is as cognizant of the words ringing within him as the orator who enunciates them from the platform. The preparation of the language and the words is in both cases the same. They both require the absolute integrity of the zone of language, the only difference being that in the second instance the finished product is sent to the cortical area which is the centre of the articulo-vocal musculature, and in the other to the Rolandic allocation of a much less complex motor mechanism, namely, to the cortical centre of the member that holds the pen, whether that member be the hand or other mobile part of the body. In the case of the speaker, the primary revival is in the auditory centre, and this influences to activity both of the other speech centres—the articulatory-kinæsthetic centre strongly, the visual centre slightly—and the perfected word is externalized by the articulatory area of the Rolandic cortex.[1] In the case of a writer, it is probable that the visual centre is the seat of primary revival of the word, but it may be quite as le-

[1] Some students of language believe that the primary revival of word images in the case of the speaker is in the kinæsthetic articulatory centre, but I hold to the view as stated above.

gitimately explained by considering that this takes place in the auditory area. The visual centre being then thrown into a state of vigorous activity, the articulatory-kinæsthetic influenced very mildly, the finished product is sent to and executed by the arm area of the Rolandic cortex. The direct association that may take place between the primary revival of auditory word images and the production of written symbols is seen in those rare cases of children born blind, who afterwards learn to write. As a rule, the visual centre conditions writing, both in its development and in its production.

The action may be compared to that of a corporation which is accustomed to send simple commissions to one broker for execution, while other more complicated commissions, requiring the concerted action of a number of people, are sent to a firm having facilities for the execution of such orders, who have accustomed themselves to such responsibilities. The first broker may execute offerings in a way very similar to the second, but his transaction is done in a different place and requires little or no concerted action, while the transaction of the latter is so complex that it requires the associate action of a number of individuals in different locations, perhaps in different cities.

It is the same way in pantomime. Every one has felt the intensity with which some simple command, such as "Come back!" "Get out!" and the like, has been formed in internal speech on occasions when necessity or circumstance compelled communication by signs. In such an instance the words are fully formed in the zone of language; we are cognizant of internal language, but our judgment informs us that it is wiser to attempt to sum-

mon to us a person who is beyond the reach of the voice by motioning of the hands or arms than by shouting. In such a case internal speech, though fully formed by the activity of centres in the zone of language, is externalized by a wave of the hand, the motor impulse of which starts from that portion of the cortex known as the arm-hand centre, and which is externalized by simple muscular movements.

All thought or ideas are revealed through words, acts, and deeds, all of which are the immediate result of muscular action. This action is conditioned by influences operative on the Rolandic cortical area, for it is there and there alone that movements having differentiated functions have representation. It should be said here that this is in apparent contradiction to the claims of some experimentalists who teach that by excitation of the superior temporal convolution there results contraction of the musculature of the ear, and by excitation of the occipital cortex there follows movement of the eyeballs. It is by no means so on closer examination, and the theory here suggested explains more logically than does any other the slight muscular activity that results from such excitation. All the special senses are provided with a highly developed muscular mechanism whose function is to facilitate the action of the special sense and to contribute to its perfection. The most typical example of this is the complex musculature of the eyeball, which is of inestimable importance in contributing to all of the welfare and pleasure that result to one through vision. The degree of complexity and the amount of musculature devoted to any one sense are conditioned by its needs, which always

stand in definite relations to the evolutionary stage or period of its possessor. In the human, the most advanced product of evolution, the musculature of the external ear is rudimentary, though not yet vestigial, because in man the sense of hearing has reached such a rare degree of perfection, such an advanced stage of development (it conditions speech), that he no longer requires a trumpet-like apparatus to turn reflexly in the direction from which sounds emanate, as do lower animals. It is the same with the other special senses, smell, taste, and touch. The sniffing movements of smelling, the smacking movements of tasting, the delicate movements that facilitate the tactile sense and which reach such a degree of acuteness in the blind, are all in evidence to show how important a part movement plays in contributing to the exquisite function of a special sense.

Every time that the eyeballs move to look at an object, every time the ears are moved to contribute to the readiness and completeness with which the auditory excitation is obtained in the lower animals, there is stored up a memory not alone of the object seen or the sound heard, in their respective situations in the inferior parietal and superior temporal lobules, but a memory register is made of these movements, kinæsthetic memories, and it is highly probable that where the one is registered the other is registered also. It will then follow that excitation of such an area might produce movements similar to those the memories of which are there enregistered. Such movements would result on excitation of these areas by sending the impulses of excitation to the Rolandic area of the cortex; thus the movement would be in reality an

indirect one along the route exactly analogous to that travelled by impulses from the zone of language to the Rolandic cortical area that result in articulate speech.

I have said that all thought is externalized by words, by acts, or by previous acts called deeds. Articulate speech is the customary manifestation of thought, but the word speech is often used generically to include other forms of thought externalization, such as writing, painting, instrumentation, pantomime, etc., which are specifically called acts; and I use it in such generic sense here.

What is said for aphasia might as truthfully be said for amusia and for agraphia if it were wise to consider music and writing independent forms of expression, but it is not. Writing and other forms of symbolic notation are but media to which mankind attach a certain conventional significance in order to expedite and harmonize intercourse. The possession and utilization of such notation requires the integrity of the zone of language—the zone of symbols and the production of internal speech. In order to use them, the memory images, visual, auditory, and articulatory, must be revived by stimuli coming from without, or spontaneously by the intelligence. In either case they are jutted into consciousness or not, as the case may be, after the completion of internal language, and they are externalized by motion in some form. I have used the phrase, "they are jutted into consciousness or not, as the case may be," advisedly, for articulated and written language may be produced without imperfection during unconsciousness. Normal sleep is the typification of unconsciousness. The time occupied by it is taken out of the cognizant existence of the individual as com-

pletely as if he were dead; consciousness of the slightest degree is antipodal to normal sleep. Yet it is within the experience of every physician to have witnessed examples of pavor nocturnus, somnambulism, etc., in which articulate speech, even to a considerable extent, was produced. In such cases the speech centres act harmoniously to an unknown excitant. The auditory images of the word are in some way revivified, and they in turn invoke the articulatory images which send their impulses to the foot of the ascending frontal convolution, from which they proceed to cause the movements of vocalization and of articulation. It seems very probable that, when speech impulses from the zone of language are deposited in the cortical motor area of articulation, the latter has no discretion or decision about what it shall do with them; it must execute them at once. In the normal conscious state, however, the faculty of inhibition plays the part of monitor to this motor area of articulation and decides whether or not impulses sent there from the zone of language shall be externalized. If the judgment decides that it is wiser, more politic, more humanizing, not to externalize the messages sent up from the speech area, it will annul them, but they are none the less vivid in internal language. It is not necessary, it appears to me, to dwell on this, for every reader must be in possession of numberless experiences conveying the truth of it. It is at the basis of so many social amenities, the retort polite, the true psychological explanation of turning one cheek when the other has been smitten instead of making the reply that instantly arises in the zone of language, that I need dwell no further on this conscious inhibition of execution of all in-

ternal language. I wish only to emphasize that in states of unconsciousness the zone of language may be incited to activity and send its finished product to the Rolandic area cortex, which executes it, and executes it as it is sent there, without inhibition, addition, or deviation by the mandates of consciousness. In similar manner may be explained the aphorism "*In vino veritas.*" Alcoholic intoxication brings about various degrees of intellectual abnegation, extending up to profoundest unconsciousness. There is a degree of this unconsciousness characterized by a cessation of the inhibitory influences of consciousness over the amount of internal speech that shall be externalized, and when this stage is reached the *veritas vini* is evident.

The motion that externalizes ideas may be of the fingers in executing a musical fantasy; it may be of any complex action which "speaks louder than words." The more intricately co-ordinate the action by which ideas are externalized, the more specialized is its representation in the Rolandic area, and the more liable is the localized lesion to cause disturbance of it. Articulate speech requires the co-ordinate action of a number of highly specialized peripheral parts. And it has been shown beyond parley that the larynx, tongue, palate, and lips have special Rolandic representation, while those for the execution of movements of writing have no other specialization in the Rolandic cortex than that of the mobile part which holds the pen. Persistent repetition of the act of writing may bring about such great facility that the act is performed almost automatically; but practice, though contributory to perfection, is not contributory to the development of a special centre; and the scrivener does not possess a special centre in

which are located the graphic motor memories any more than does the telegrapher a telegraphic centre, or the typewriter a typewriting-centre, or the pianist a special centre in which are represented the complex movements of the fingers. There is a special and very definite cortical allocation for movements of the hand and for movements of the fingers, probably for each individual finger; at least it would seem to be definitely proven that there is such representation for the index finger. The acquisition of great dexterity from long practice in writing facilitates the readiness with which such specialized motor centres functionate, the same as practice facilitates the execution of all acts, voluntary and involuntary. And this facilitation may become so great that the muscular movements required are made quite unconsciously, apparently automatically, as witness the movements of the telegrapher's fingers when they touch the keyboard, or of the violinist as he fingers the strings. Nevertheless, notwithstanding this facilitation, which may be dependent anatomically upon new association tracts, the essential constitution of the act is the same as it was in the beginning, and no short cuts have been formed nor has a new centre been developed.

I shall return to the discussion of a special writing-centre later on, but before doing so it seems to me desirable to cite some of the evidence that may be offered in support of separate localization in the Rolandic regions for the movements of the larynx, palate, tongue, lips, and respiratory movements before taking up a discussion of the constitution of the speech area and the closer relation of the centres there situate to other parts of the brain.

Because of the importance that I attribute to clinico-pathological observation in comparison with the experimental, although fully cognizant of the great value of the latter, I shall cite first, as briefly as possible, a few cases that have been studied clinically and anatomically which seem to give unequivocal support to such localization. And before doing this, I shall call the reader's attention to the necessity of bearing in mind that in this monograph the term Broca's convolution is not used in the sense which many writers of the last generation would give to it—to include the foot of the third frontal convolution, the lower part of the ascending frontal convolution, and the lower part of the ascending parietal convolution, and thus not only the entire frontal operculum but a part of the parietal as well. In this treatise the term Broca's convolution is used synonymously with "foot of the third frontal convolution," and its physiological synonym may be said to be "centre for articulatory kinæsthetic images."

A case reported by Elder[1] is one of much importance. The essentials of this case are as follows: A man sixty years old, who had previously been healthy, suddenly developed difficulty in speaking. Speech became indistinct and blurred, and saliva trickled from the mouth. On the following day he was so weak that he demanded admission to the hospital, and it was then found that there was paresis of the right side of the face, more marked in the lower part and at the angle of the mouth and not involving the orbicularis palpebrarum. The patient was perfectly conscious, understood everything that was said to him, had no hemiplegia, and his only trouble

[1] Elder: Edinburgh Hospital Reports, vol. iii., 1895.

apparently was incapacity to enunciate words, due to difficulty in moving the tongue, lips, and other muscles of articulation as readily as was necessary. There was no aphasia. The voice was unimpaired. He could protrude the tongue, although it came out rather slowly, and there was difficulty in swallowing liquids. The patient grew weak very rapidly and died five days after the occurrence of the dysarthria, the immediate cause of death being hypostatic pneumonia, coma, but no hemiplegia having preceded death. On examination of the brain, there was found in the lower part of the ascending frontal convolution, about half an inch from the Sylvian fissure, a blood clot which had pushed its way through the cortex and which could be seen before cutting into the hemisphere, so completely had it destroyed the cortical substance. On slitting the brain horizontally, the blood clot was found to contain about two drachms of blood, situated at the level of the lower part of the ascending frontal and the ascending parietal convolutions. It had destroyed almost entirely the cortical substance of the lower end of these convolutions. It did not involve quite the whole of the lower end of the ascending frontal, as there remained intact a strip of cortex adjoining the foot of the third frontal. The foot of the third frontal was quite intact. The accompanying diagram shows the extent of the lesion, both as it appeared from a view of the uncut hemisphere and on horizontal section (Fig. 11, *A*). Internally, the hemorrhage extended inward and forward immediately about the level of the lenticular nucleus in a very thin layer for about two-thirds of the distance between the cortical surface and the internal surface of the hemisphere. Its far-

thest point internally was close to the anterior limb of the internal capsule and quite in front of the motor tract. The situation and extent of the lesion is shown in Fig. 11, *B*.

This observation is almost equal to a mathematical demonstration, so convincing is it that a localized lesion of parts of the cortex which are strictly motor, namely, the Rolandic-area cortex, may when diseased be attended by inability to make the peripheral associations or movements necessary for speech. It is not the only one of its kind on record, but it is the most convincing one on account of the fact that there were no complicating lesion and, of course, no other symptoms. Then the strictness with which the lesion was confined to the lower part of the Rolandic cortex, its sharp delimitation from the third frontal convolution, and the rapidity with which death followed the occurrence of the lesion, all tend to make it an ideal case in proof of the claim of separate localization for the peripheral speech mechanism.

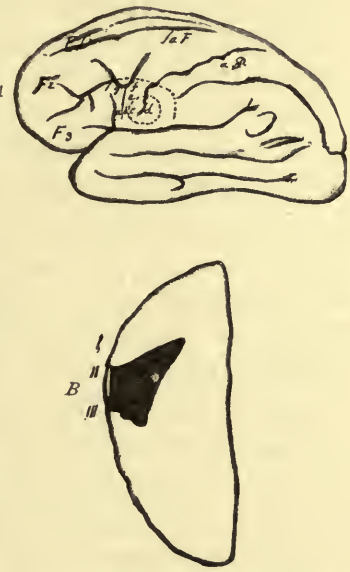

Fig. 11.—*A*, Subdivision of Broca's convolution, according to Elder. *a*, Centre of psycho-motor speech images; *b*, centre for adduction of vocal cords; *c* and *d*, articulo-motor centre; *e f g*, centre for lower and upper face, respiratory movements of larynx, respectively; dotted circle indicates seat of lesion. *B*, Lesion on cross-section.

Cases in which there has been subcortical lesion of the motor tracts that come from somewhat indefinite parts of the Rolandic area are recorded to a considerable number; but, no matter how carefully they have been observed or the pathological lesion has been described or depicted, they never can be offered as absolute proof, because the fibres which are interrupted by such a subcortical focus of disease can never be said positively to come from a definite localized part of the cortex. Moreover, it is extremely rare for a subcortical lesion to be so limited that it will sever the fibres going from one small area of the cortex and leave intact all the others, for it will be remembered that the projection fibres continually approximate one another from the time they leave the cortical cells until they are brought into the internal capsule, where they are crowded together in a space that is little more than visible to the naked eye.

Dejerine[1] has described two cases of subcortical lesion in which a small area of softening existed just internal to the anterior part of the foot of the ascending frontal convolution. These patients had the symptom complex of subcortical motor aphasia and were not hemiplegic, which shows that the lesion was not of sufficient extent to involve any of the projection fibres coming from the arm or the leg areas. The only discordant symptom in these cases of Dejerine was that in both cases there existed a degree of paralysis of the right vocal cord; in one case the paralysis was complete, and in the other partial. This is in contradiction to the findings of experimentalists, who believe that

[1] Dejerine: "Aphasie motrice sous-corticale et localisation cérébrale des centres laryngés." Société Biologie, 1891, p. 97.

the vocal cords have a bilateral and double representation; that is, stimulation of the vocal-cord centre on one side of the brain will cause adduction of both vocal cords, and therefore lesion of one side should not cause complete paralysis of either vocal cord. This apparent contradiction, however, should not stand in the way of utilizing the cases to prove that a lesion subcortically situated in the brain, that cuts off the projection fibres coming from the lower end of the ascending frontal convolution, will be followed by a degree of dysarthria which may be so complete that the patient is devoid of articulate speech, and therefore of proving that the movements necessary for articulate speech have a cortical representation all in one definite area of the Rolandic zone.

Bernheim[1] has recorded a case which indicates with a good deal of directness the cortical localization of lingual movements. Clinically, there was pronounced deviation of the tongue to the right; no other paralysis, and no aphasia. After death there was found a sarcomatous blood cyst, five to six millimetres long, located near the inferior border of the left ascending frontal convolution, about six millimetres behind the sulcus which separates it from the third frontal.

An observation of Garel[2] is strikingly corroborative of Dejerine's contention that the cortical laryngeal centre is situated in the lower end of the ascending frontal convolution. In this case, the principal symptom was paralysis of the vocal cords. The autopsy showed a localized me-

[1] Bernheim: "Contribution à l'étude des aphasies." Rev. Méd., pp. 372–388, 1891.—"Des aphasies." Congrès Français de Méd. Interne, Lyon, 1894.

[2] Garel: Ann. des Mal. de l'Oreille, du Larynx, etc., April, 1890.

ningo-encephalitis at the lower end of the right ascending frontal convolution.

The cortical representation of the laryngeal muscles has been carefully studied by Krause, by Masini, Beevor, and by Horsley and Semon. Their conclusions regarding the centre for the laryngeal movements are very much in accord. They show that in the ape the centre for the larynx is situated in the anterior part of the foot of the ascending frontal convolution, that the representation is bilateral, but that the preponderant representation for the vocal cords of one side is in the opposite hemisphere, and that each hemisphere governs the movements of both vocal cords. Wallenberg has published a case in which there was laryngeal paralysis of the left side, and the projection fibres from the portion of the cortex in which the laryngeal centre is situated were entirely destroyed. The experimental physiologists furthermore have proven that in the anthropoid the centres for the movements of the lip and the tongue are situated in the lower part of the ascending frontal and the ascending parietal convolutions, and that these movements are not represented in any way in the third frontal convolution. They have shown, furthermore, that the centres for the movements of the muscles of the tongue, throat, and mouth are situated adjacent to one another in the central convolutions on either side of the fissure of Rolando, and that the centres for the angle of the mouth and the upper part of the face are placed higher up, toward what is generally known as the face area.

The part of the cortex to which the centre for respiratory movements is allocated is yet unknown. Respiration is essential to speech, and, although we may produce sep-

arately the three distinct mechanisms of respiration, vocalization, and articulation, it is by the simultaneous activity and co-ordination of these three that perfect speech is produced. As respiration, then, seems to be one of this important triad, and, as it is positively proven that the other two have a cortical representation in the Rolandic region, it seems reasonable to assume that there is a cortical centre or area for respiration. This representation is a difficult one to prove, because respiration is more completely under the control of the reflex centre situated in the oblongata than is any other function. The only experimental evidence that can be utilized in favor of the localization of the movements of respiration is that of Horsley and Semon, who found that stimulation of the frontal convolution immediately adjacent to the face area in the ascending frontal caused an increase in the rate and in the force of respiratory movements. In the dog and in the cat, Franck has shown that by stimulation of the motor areas of the cortex decided acceleration or slowing, according to the duration and the intensity of the stimulation, was produced.

After this brief account of the separate localization of parts concerned in the externalization of articulate speech, I may, before discussing the relation of speech to consciousness, intelligence, or, in general, to the frontal lobes, revert for a moment to the relative positions of the centres of speech in the area of language. As I have previously said, these centres are three in number: the centre for auditory memories, the centre for visual memories, and the centre for articulatory kinæsthetic memories. The latter, unfortunately, is often called the centre for motor memo-

ries. These three centres have a very definite localization, and their position is of great ontogenetic importance. The centre in which are stored the images of articulation is situated in the third frontal convolution immediately adjacent to that portion of the Rolandic cortex the cells of which give origin to the projection fibres going to the tongue, the lips, and the larynx; that is, to the parts which supply the peripheral mechanism of articulate speech. Where else should such a centre be, if its fullest integrity were desired, than immediately adjacent to the part whose commissions it executes? The centre in which are stored the visual images is situated in a definite part of the inferior parietal lobule, in that portion of the lobule known as the angular gyrus, and if we have in mind the central projections of the optic tract after it leaves the external geniculate body, the anterior quadrigeminal body, and the pulvinar of the thalamus, until they reach the lingual and fusiform lobules bordering the calcarine fissure, we shall appreciate that the angular gyrus is the most direct, the most adjacent, and the most elective place in which the visual images could be stored. In fact, its relationship to the primary visual centre and to the fibres that convey visual impulses, the radiations of Gratiolet, is analogous to the environmental relationship between the centre for articulatory memories and the Rolandic cortex that externalizes speech, but there must always be a revivification of the articulatory kinæsthetic memories before speech can be produced. The third centre, the auditory centre, the most important of all speech centres, the one by whose functioning speech is developed, the one that conditions speech more than do both the others combined, the one in which

words are primarily revived in the vast majority of peoples, the one that is least tolerant of disorder without manifesting itself by imperfections of speech, is situated between these two centres in the zone of language, and occupies the first temporal convolution and particularly that portion of the cortex which surrounds the temporo-parietal sulcus. It occupies such a position in relation to the distribution of the auditory nerves and the mechanism of hearing that experimentalists as well as clinicians have been led to posit it as a general auditory area. The centre for the storage of auditory memories is not placed anywhere in the general auditory area: it is placed in the posterior part of the first temporal immediately adjacent to the gyrus in which are stored visual memories. Thus it will be seen that the auditory and visual memories which are contributory to the development of speech and education in general are not widely separated. They are not so far removed from each other that a great distance must be travelled before the products of one can be compared with those of another; they are, on the contrary, immediately adjacent. On the other hand, they are not adjacent to the centre in which are stored the memories of articulation, and to which the products of the activity of the former two are sent before they go to the Rolandic-area cortex to be executed. Furthermore, the location of the visual and auditory centres on the one hand, and of the centre for articulatory memories on the other, would seem to me to have a very definite suggestiveness in relation to the frontal lobes, which physiologists, psychologists, and pathologists believe to be the most essential parts of the brain for the production of the higher mental processes—

judgment, will, inhibition, and emotions — and whose integrity must be maintained if the individual is to develop or persist in habits of attention, concentration of thought, balance of feeling, sound judgment, and moral conduct. In a previous portion of this chapter we have referred to the important part played by consciousness or intelligence in the faculty of speech. The relation which the zone of language bears to the frontal convolution, topographically and morphologically, is not without its significance. A glance at Fig. 7 which represents the zone of language will show that it is on the lateral surface of the brain; that behind, it is contiguous with the visual area; below, with the auditory area; above, with the motor executive area, and in front with the intellectual area. This speech zone, this zone of language, receives information; it is apperceptive of impulses coming into it, mainly from below and behind; it submits them to the area in front, and they are externalized by the Rolandic area. It is, then, one great association sphere of the brain, in which are located for reasons of expediency certain centres for the reception of impulses. But the reception of impulses alone could not give rise to intelligent speech. Even granting them a certain autonomy, they could give rise only to an echo of the impulse, and this is often beautifully illustrated in certain forms of sensory aphasia in which one of the centres is destroyed and the circuit for internal speech broken; the patient, though still capable of receiving in his zone of language some speech impulses, has not the means to co-ordinate them and submit them to the intelligence, and he can only echo sounds heard, a condition called echolalia.

Pathological observation and clinical experience are in fullest accord with this conception of language, and if clinical experience has proven one fact more conclusively than another to the satisfaction of trustworthy observers, it is that no centre of this zone of language can become disorganized without there appearing a perversion of function, not alone of the centre that is destroyed, but of all the others; in short, that in every form of genuine aphasia, whether it be so-called motor, auditory, or visual, there is some perversion of idea externalization and of language. It is to Dejerine and his pupils, Mirallié, Thomas and Roux, that we owe the proof of this statement more than to any others, and to them we cheerfully accord deserved praise for maintaining their contention in the face of very nearly universal opposition.

Perversion of internal language is manifest most discernibly by disorder of internal reading. During the past few years I have examined all my cases of aphasia, whether motor or sensory, with the object in view of determining whether or not there was defect of internal or mental reading, and thus of corroborating the statement of Dejerine. I have found that in every case, without exception, examined with sufficient care and patience and in the right way, there was detectable loss of the ability to read mentally. Naturally, it is in the cases of cortical motor aphasia that the results are of great importance, for in genuine sensory aphasia, visual and auditory, there necessarily exists disturbance of mental reading. As I shall say in the chapter on articulatory or true motor aphasia, disturbance of reading has been noted by nearly every observer from the time of Trous-

seau, and various explanations have been put upon its occurrence, the usual one being that it was the result of either a slight dementia or a mixed aphasia. According to the conception of aphasia which I have attempted to outline, defect of mental reading is just as likely to occur in a patient with cortical motor aphasia, although not so profoundly, as in a patient with auditory aphasia.

The degree of dyslexia that occurs when the convolution of Broca is diseased is discussed in another chapter (Chapter V.), but it needs to be mentioned here that one reason for its less common recognition, and therefore for the unwillingness on the part of certain writers to admit its occurrence, is the fact that after it has existed for a certain length of time it is only slight and very often latent. These patients essay to read and often busy themselves for hours with books and papers, from which they apparently get the customary enjoyment and diversion. On the other hand, after the alexia has lasted for a time, the patient may learn anew to read, and the fact that he learns anew in a manner which is entirely the reverse of that which he employed when a child has been used by modern pedagogues as an argument in favor of the word-and-syllable method of learning to read. The patient with cortical motor aphasia who must re-learn to read does so by first getting the perspective of the word, the outline, then the syllables that constitute the word, and, last of all, the letters and their association that enter into the formation of the word. Thus they learn to read in a way similar to that at present advanced by many educationalists, and the reverse of the way in which most of us learned to read.

It will be seen at a glance how materially this conception of aphasia differs from that which is taught by a majority of writers on the subject of aphasia to-day. Even the most recent writers on the subject, those who essay to contribute to our knowledge of aphasia, of whom we may mention Wyllie, Elder, and others, as well as those who have no such object, who merely restate in their own way that which passes as current knowledge, describe different forms of aphasia and the different centres diseased to produce these forms, as if the faculty of language were made up of a different set of entities and each of these entities were localized to a different part of the brain. The conception of aphasia that passes muster to-day with the majority of writers is one suggested by Marcé, in 1856, and which was adopted by Charcot; and the wide dissemination and acceptance that have been given to it are largely due to the assiduous and lucid teachings of the latter. I shall now concern myself particularly in showing that these views are no longer tenable. At the very outset, I wish to say that it is not the logic of the arguments of Charcot and his followers with which I am concerned, but it is with the original contention, which is, I believe, entirely unwarranted.

Charcot and his school, as well as many other writers on aphasia who are entirely opposed to Charcot's conception of it, such as Bastian, believe that there are four speech centres. I have already stated that the Charcot school teaches the autonomous action of these centres, although admitting that they have a certain functional interrelationship. These centres on the emissive side of language are the articulatory motor and the graphic motor,

and on the receptive side the centres for auditory images and for visual images. These centres have a definite autogenetic individuality, which is augmented decidedly by education. Depending upon this congenital or inherited tendency for the predominance of one centre over another, and contributed to by education, the individual acquires a knowledge of things, their properties, their names, their usage, principally by the employment of one of these centres. In speech, words are revived predominantly by the activity of one of these centres, and in speaking he orients himself through the activity of this individual centre. Depending upon the centre that plays such an important part in the primary revival of words, the individual is said to be a *visuel*, a *moteur*, an *auditif*, a *graphic moteur*, or an *indifferent*, both in the acquisition of knowledge by language and in the use of knowledge by language. Leaving aside for the time being the fact that psychology, physiology, and pathology are in accord in denying the autonomous existence of any one of these centres, this theory of Charcot and of his followers must stand or fall with the existence of the four centres, and I venture to believe that we are in possession of sufficient evidence to deny the existence of a graphic motor centre.

I have striven to show that the zone of language contains three centres, each of them the seat of memory images of a sensory nature and none of them of absolute autonomous activity. That they are relatively interdependent, and that one cannot be deranged without causing some disturbance in the totality of function of these three centres, has already been shown. Indeed, in the early history of aphasia, before Broca made his

memorable observations, Naugere taught that disturbance in the production of one component of speech manifested itself by disorder of all the others. These teachings were followed by Gairdner and by the Nestor of English neurologists, Hughlings Jackson. According to the theory of Charcot, the emissive centres of speech, the articulatory motor centre and the graphic motor centres, are *loci* in which are stored the motor memories of articulation and the motor memories that guide the hand in writing. Indeed, it is not entirely apparent that this savant was not of the opinion that these centres are the places in which originate the motorial impulses that are externalized as speech and as writing. The localization of these two autonomous centres Charcot believed to be absolute, and he fixed the one for graphic motor images in the foot of the second frontal convolution, and that for articulatory motor images in the foot of the third frontal. The frequent association of motor agraphia and motor aphasia he explained by the juxtaposition of the centres, and he averred, apparently in the face of conclusive contradictory evidence, that a lesion that destroys the third frontal does not entail agraphia. The coexistence of motor agraphia with motor aphasia he claimed was not necessary, and it might be said that since then his pupils, Pitres, Grasset, Marie, Brissaud, and others have claimed the same and contend that there are cases of pure motor agraphia without aphasia, but the cases they cite to substantiate their position do not stand the test of analytic scrutiny.

It will readily be seen how opposed to our conception of speech and of the function of the zone of language is this conception of Charcot. In one word, according

to his teaching, the centre is all-important; according to ours, the centres are important, but only as one individual in a community. I have no intention of dwelling further on this distinction, except to show the untenability of the position of the school of Charcot by discussing the existence of a graphic motor centre.

Is there a Graphic Motor Centre for Registration and Regulation of Writing-Movements?

The most important evidence that the claimants for the existence of a graphic motor centre, in which are stored the motor images of writing (which by this school are looked upon as a constituent part of the idea of the word), can offer is that which was furnished by Exner,[1] of Vienna, in 1881. It is not hazarding the truth to say that the evidence offered by Exner in support of the existence of such a centre would not be admitted as proof in any other department of science than medicine. Nevertheless, it has been admitted to the latter and it must, therefore, be discussed. It was the necessity for the existence of such a centre apparently that led Exner to seek it, and Charcot,[2] seeing in it a contribution to his

[1] Exner: "Untersuchungen über die Localisation der Functionen in der Grosshirnrinde des Menschen."

[2] It is astonishing how universally accepted is the existence of a particular or undivided graphic motor centre. Writer after writer accepts the findings of Exner in a way that leaves the impression that no doubt can possibly exist concerning the reality of the centre. Even Bastian, who is not a believer in the complete topographical distinctness of the several sensory centres in the cerebral hemispheres, and who has been one of the most unswerving opponents to the conception of aphasia taught by the school of Charcot, says, in the Lumleian Lectures, the first of which appeared after this monograph was practically finished, that "the grapho-motor centre can be localized with nearly as much confidence as the articulatory

theoretical exposition of the faculty of language, accepted it.

Exner made an artificial division of the external, internal, and inferior surfaces of the cerebral cortex. There was no method in his division, no rule, no preconception. So far as possible the principal fissures were the lines of separation of these artificially outlined areas, but this was merely for the sake of convenience and ease of orientation. The areas had different shapes and sizes, so that when the division was made the result might be compared to a territorial map of the United States. If one will look at such a map, he will see that oftentimes natural divisions, rivers and mountains, separate the States. These are the principal fissures of Exner, but in most cases the division is arbitrary. He then searched the literature of local brain disease and made notes of three hundred and sixty-seven cases, to find how many times any one area was the seat of lesion in the cases observed and how often each area was affected when individual symptoms had been observed in relation to the disease.

I do not deem it necessary to give the details of his analysis but may say that the majority of his conclusions do not correspond with the present teachings as to cerebral localization. The correspondences that do exist might laudably be explained by chance. The fact that

kinæsthetic centre." There is such a dubiousness about his further statement of its localization that I shall quote his exact words. "The tendency for some years has been to follow Exner, who believes the graphic motor centre to be situated in the posterior part of the second frontal gyrus, though we shall see later the evidence in favor of this localization is at present extremely scanty. All that can be said on this point, therefore, is that we know approximately where to look for it." This, it would seem to me, scarcely warrants the language of the first quotation.

every brain varies in fissuration, in convolutional area, and in formation, and the fact that no one surface marking can be taken to fit another brain, make it difficult to see how any definite inferences could be drawn from such a method of investigation. This method would seem to me to be paralleled if a sociologist who had become convinced of a definite locus for homicides should set to work to substantiate his contention by getting statistics of the number of men slain in the United States, let us say in ten years, and then statistics to show how many homicides were to be found living in any State, and how many times the victims were found in any given State. The State that had the greatest number of homicides and the greatest number of victims would be a homicidal centre. It would depend upon the sociologist whether or not that State would be considered the only homicidal centre. In seventy-three of Exner's cases there were lesions that gave rise to paralysis of the face, and of these about one-half were grouped in the median part of the ascending central convolution, while four of them showed lesion of the third frontal; fourteen, lesion of the second frontal; eleven, lesion of the angular gyrus; eight, lesion of the first temporal, etc. It must be granted that such contributions as these are not of much service in helping to give brain localization scientific exactitude. Let us take, for instance, another example. The lesion in one hundred and thirty-one cases was one that caused paralysis of the upper extremity, and in eight of these cases the third frontal was the seat of lesion, in thirteen the second frontal, in eleven the first frontal, in twenty-three the ascending frontal, in eighteen the ascending parietal, in fourteen the superior parietal,

in thirty-three the angular gyrus, and so on. In other words, the area of the cortex for the representation of the arm is distributed all throughout the brain cortex. Let us contrast this for a moment with the facts as they really exist. All clinicians and anatomists are in accord in allocating to the cortex of the central lobes, particularly the ascending central, the representation for the upper extremities.

Exner's analysis of the cases taken from the literature, and studied by the men who had reported the cases, showed only four instances in which agraphia had been present. In all of these, with the exception of a case reported by Bar[1] in 1878, there were multiple lesions in the brain. In Bar's case, the symptoms had been motor aphasia, agraphia, and facial hemiplegia, and the lesion was localized in the foot of the second frontal convolution; therefore Exner posits this convolution as a graphic centre, and by dint of repetition by writers on aphasia since Exner's claim was first made this fallacy has taken an almost undislodgable place in medical teachings. It seems to have been forgotten that the patient had motor aphasia and only partial agraphia. He could not express his thoughts in writing, no more could he in speech. If the lesion was of the graphic motor centre and strictly confined to it, as we are told that it was, why was the agraphia not total and why did the patient have aphasia? The fact that he had aphasia is evidence enough that there was lesion of the zone of language (a subcortical lesion being excluded), and the fact that he had motor

[1] Bar: "Aphasie et hémiplégie faciale passagère." La France Médicale, 1876, p. 609.

aphasia is sufficient explanation of the partial agraphia. The former entailed the latter. This case, therefore, instead of lending itself to Exner's claim, to Charcot's theory, or to Ziehen's argument (not to mention the scores of other writers on aphasia who have quoted it with the reverence accorded to Scripture), is unequivocally opposed to them.

The ideal case to prove the existence of a centre whose function is to store graphic images, and whose destruction would cause agraphia, would be one in which there was inability to write sponanteously and from dictation, and in which no disturbance of motility, particularly no disturbance of the right upper extremity, no aphemia, no alexia (that is, no inability to read aloud), no verbal deafness, and no verbal blindness existed. It is remarkable, considering the zeal of physicians in observing and reporting cases of aphasia during a period which now covers nearly a half of a century, that no such case has been recorded. It is also remarkable that no instance has been reported in which there was strict limitation of the lesion to the second frontal convolution, with the exception of Bar's case, which has been already mentioned.

Although clinical cases should not be allowed to bear absolute testimony in favor of the existence of a separate centre for graphic motor images, when such are unaccompanied by autopsy, nevertheless, a number of clinical cases have been reported in which the evidence, on superficial examination, seems to point with great certainty to the existence of such a centre. Although all of these cases cannot be considered here, I shall cite a few of them to show that a different and satisfactory interpretation can be put

upon them, one that does not require the existence of a graphic motor centre. A case of this kind that has often been cited to prove the existence of such a centre, but which has been interpreted by Dejerine and others in an entirely different manner, is one reported by Pitres.[1] In this case, Pitres says, there was no disturbance of intelligence, nor was there paralysis of the right side. There was no trace of word deafness or of word blindness. Nevertheless, the patient was incapable of expressing a single thought by writing, and he was quite as incapable of writing from dictation. Examination of the patient nearly ten years after the history was first published showed that the conditions were practically unchanged; that is, that still the unique symptom was agraphia. It therefore seemed probable to Pitres that this one symptom, persisting unchanged for such a long time, was the result of lesion of a region of the brain which, does not serve mental vision, nor yet phonetic articulation, but which presides uniquely over the motor excitation of writing. It does not seem to me that such inference is at all warrantable. In Pitres' last reference to this case he has neglected to mention one or two very prominent symptoms, whose importance is, I believe, vital. In the first place, the patient had hemianopsia, and, in the second place, he was able to write with the left hand. These two facts alone would seem to me to stamp the case as one of sensory agraphia dependent upon a unilateral subcortical lesion that had interrupted the connection of the area in which is stored the visual memory of words, the angular gyrus, with the motor zone of the left hemisphere, leaving

[1] Pitres: "Agraphie motrice pure." Rev. Méd., 1884, p. 855.

the connections with the right hemisphere intact. In such event, the patient would be unable to project the visual images of letters and words which were properly conserved to the Rolandic-area cortex of the left zone, but there would be no impediment to the passage of such images to the right Rolandic area. Therefore the inability to write with the right hand would follow as a natural consequence, likewise the ability to write with the left hand after the latter had been trained to writing-movements. Pitres states that the patient, on being asked to write the word Bordeaux, said that he could call up in the mind, that is, visualize, most distinctly the word Bordeaux, but he could not write it, and the author offers this in evidence that the inability to write was not due to loss of visualization of words, but was due to destruction of a centre in which are stored graphic motor images. To quote the patient's own language, "I know very well," said he, "how to write the word Bordeaux, but when I wish to write it with the right hand I cannot make anything." This appears to me to be the keynote to the interpretation of the case. The centre of visual images was intact, but the pathway by which images travel to the hand centre in the Rolandic-area cortex, to be externalized, was interrupted. Therefore he could not write anything with the right hand. Pitres very correctly says that this is quite different from sensory agraphia, and so it is from sensory agraphia due to destruction of the angular gyrus, but it is not different from the cases of agraphia due to a subcortical lesion. If there be a centre in which are stored graphic images of words, it would seem to me that destruction of this centre must entail complete agraphia,

not only for one hand, but for the other, and for any other part of the body which may be trained to hold the pen. If this is not so, then persons who are capable of writing as well with the left hand as with the right, as I am, must have a separate graphic motor centre on each side of the brain. This does not seem to be at all in accord with what we know definitely of the cortical localization of speech functions.

A second case that has taken an important place in literature to prove the existence of cortical motor agraphia is one cited by Charcot.[1] The patient, a Russian general, who was able to speak with facility in addition to his mother language both German and French, suddenly lost the ability to speak in either one of these languages, although he was still able to understand them. After a time the capacity to speak French was regained, but he was never again able to use the German tongue. At no time was there any paralysis, nor was there any difficulty in understanding any of these languages in print or in writing. The interpretation put upon this case by Mirallié seems to me lucid and satisfactory. The patient was probably a motor aphasic for two of the three languages which he had spoken fluently, that is, there was loss of the articulatory images of the languages which he had last acquired, and apparently, they were lost, relatively to their temporal acquisition. The articulatory images of the mother tongue, which every one concedes are more indelibly fixed than the images of languages acquired later in life, were not concerned. The motor aphasia was, therefore, very slight, and a considerable degree of re-

[1] Charcot: "Leçons sur l'aphasie." Progrès Médical, 1883.

covery followed. Careful examination of the report of the case shows that the patient was not totally agraphic, but that he could write a few letters. When he was asked his address he said, "I reside at the Hôtel de Bad, Boulevard des Italiens," but when he was asked to put his answer in writing he was able to write voluntarily only "I re——" and then wrote the remainder of the word from dictation. It is interesting to note that the agraphia was most complete for the German language, less so for the French, and least so for Russian, for he was able to write Charcot's name in the latter language without much difficulty. If one admits that there was here a slight form of cortical motor aphasia that had undergone considerable amelioration, that is, loss of the articulatory images to a slight degree for the Russian, and to a very much greater degree for the French and German, he must admit that such loss of images would cause a disturbance of internal speech, and that this disturbance would be manifested by agraphia.

A very similar interpretation must be put upon the case recently reported by Prevost,[1] who cites a case of Jacksonian epilepsy accompanied by motor aphasia without agraphia to prove that the former may exist without the latter. The patient was a man sixty years old who had been neurasthenic for a number of years. A few days before he consulted Prevost he had remarked a considerable difficulty of speech, particularly in finding appropriate words. After this the nervousness increased; he complained of headache and of a disagreeable sensa-

[1] Prevost: "Aphasie motrice sans agraphie." Revue Médicale de la Suisse Romande, June 20th, 1895.

tion in the right arm. Shortly after this there occurred epileptiform attacks with partial loss of consciousness. During these attacks he made efforts to speak, and succeeded in uttering some unarticulated sounds while he grasped the chin with the left hand and the head was turned convulsively to the right by a series of clonic convulsions extending into the right facial region and associated with conjugate deviation of the eyes toward the same side. Immediately after the attack, there was very pronounced difficulty in speaking. During the next few days the patient had a number of attacks in which the convulsions extended to the right arm, but spared the right leg. On the eighth day after the beginning of the epileptiform symptoms he became, immediately after an attack, completely unable to speak, although it is stated that he understood perfectly spoken and written words, and that writing was in no way disturbed. Considering the fact that the patient was not tested for ability to write from dictation and from copy, it cannot be said positively that writing was not disturbed. Under vigorous antisyphilitic treatment, the fits ceased abruptly and the ability to speak returned. Eight months later the patient developed a right-sided hemiplegia. It is probable that in this case the lesion was a syphilitic one, as there was an inconclusive history of luetic infection and the symptoms yielded to antisyphilitic treatment, and that the first manifestations of the lesion were in the lower part of the Rolandic-area cortex; that the patient had a subcortical motor aphasia, and that the twitching in the right side of the face and later of the right arm indicated an accession of the lesion irritating the fibres conducting

motorial impulses from the cortex to these regions; that the syphilitic vascular lesion was not an extensive one and did not destroy the area in which it was seated. This explanation would readily account for the temporary loss of speech following the convulsive attack, and for the preservation of ability to write. The fact that the patient developed complete hemiplegia a few months later is of considerable weight in substantiating this interpretation as being one of slight cortical motor aphasia in the beginning, the lesion being one that interrupted the passage of motor articulatory images to that part of the Rolandic region that starts the impulses to externalize them.

Finally, a number of cases must be considered, to show that there are no real examples of cortical motor aphasia without agraphia. Several examples have been cited to prove the existence of such a condition, and of these the one by Banti[1] is the best-known. His case was that of a man thirty-six years old, sufficiently well educated to read and write correctly, right-handed, who was suddenly stricken in the street with apoplexy, followed by temporary loss of consciousness. During the following night the right-sided hemiplegia from which he suffered almost entirely disappeared, but the inability to speak which had come on simultaneously with the loss of consciousness still persisted. He was examined by Banti on the following day, who noted that there was scarcely any trace of paralysis. On being asked his name and other simple questions, the patient made futile efforts to speak, but was unable to articulate a syllable or a sound.

[1] Banti: "L'aphasie et ses formes." Lo Sperimentale, 1886, vol. lvii., p. 261.

He chafed under this inability and essayed to convey his thoughts by pantomime. On being given a pencil, he wrote his name and the answers to other questions, with great readiness. Moreover, on being requested to give an account of his illness, he wrote a very detailed description. Writing from dictation and from copy was also possible, and the patient assured Banti that he was able to understand everything that he wrote, everything that he saw written, and everything that was said to him. That is, there were no traces of word blindness or of word deafness; but the inability to articulate was complete, although Banti states that he wrote a number of simple words and endeavored to incite the patient to read them in a loud voice and to say them after him. Although the patient observed with great attention the movements of the physician's lips and made strenuous efforts to obey, he was never able to pronounce a single word. Three years later the patient had entirely recovered, the amelioration of the aphasia having been a gradual one. After the recovery was complete, it was still noticeable that occasionally he slightly mispronounced words. The patient died five years later of an aneurism of the aorta. In the brain there was found a patch of yellow softening situated in the posterior portion of the left third frontal convolution, comprising part of the cortex situated between the pre-Rolandic fissure and the anterior branch of the fissure of Sylvius. The author explicitly states that it did not involve the white substance. This case, which apparently on first sight proves the possibility of the occurrence of motor aphasia without concomitant agraphia, does not really do so. The fact that the patient recovered the

ability to speak shows that all the cells of Broca's convolution could not have been destroyed. Moreover, the fact that there were no word blindness and no auditory aphasia shows that the lesion of the zone of language must have been slight. If particular information had been given of the time and way in which the patient recovered the ability to speak, the case would have been very much more instructive, for it might be made to bear testimony in behalf of the assumption of speech function by the uneducated centre of the opposite side. Yet I am inclined to think that we must admit that neighboring cells are capable of taking up the functions of destroyed cells, not only of symmetrically located ones but of adjacent ones. Such information was not vouchsafed, and therefore the case must be looked upon as an inconclusive one.

Kostenitsch[1] has published an observation of a man fifty-six years old who had a right-side hemiplegia with complete motor aphasia of seventeen years' duration, but who wrote with great ease with the left hand. He avowed that he could understand everything that was said to him, he could sing melodies without the words, and he could read what he had written. At the autopsy very extensive lesions were found, particularly in the central ganglia, and the whole left hemisphere, more particularly the frontal lobes, was very much atrophied. The atrophy of Broca's convolution was especially well pronounced. This may be considered an illustrative example of the cases that have been cited to prove the existence of so-called motor

[1] Kostenitsch: "Ueber einen Fall von motorischer Aphasie," etc. Deutsche Zeitschrift für Nervenheilkunde, 1893, vol. iv.

aphasia without agraphia. It is an exact counterpart of the case cited on page 201 of this monograph, and it is susceptible of very similar interpretation. The case is, in reality, quite a typical one of subcortical motor aphasia, and the report of the autopsy is the most conclusive proof of this contention. That the original lesion was subcortical was shown by the complete destruction in the central masses, and to these the atrophy of the cortical substance, a pure inactivity atrophy, was due. The microscopical examination of the cortex, which showed stunted cells and cells lacking in axones, is decidedly in favor of this view.

In this country a case observed by Osler[1] has often been cited in support of a centre for graphic motor images. A *résumé* of this case is briefly as follows: An old man suddenly developed, without paralysis, inability to read the newspaper, right lateral homonymous hemianopsia, paraphasia, and complete word blindness. On being asked his name, he was able to pronounce it, but the alteration of spontaneous speech was very manifest when he attempted to tell his occupation, which was that of bookkeeper. As Osler noted it, the response to this question was, "Keep, keep, keep—oh, you say it for me." In referring to a wetting he said, "Deliberate attack of wet dress." It was difficult to get him to write and it was impossible for him to write from dictation. He was able to write his own name quite as well with the eyes closed as with them open. He wrote the word "Record" when told to do so,

[1] Osler: "A Case of Sensory Aphasia—Word Blindness with Hemianopsia." American Journal of the Medical Sciences, Philadelphia, pp. 219-224, 1891.

but he spelled it "Freedom." These symptoms persisted two months, during which time there was gradual loss of muscular strength and mental power, and finally, thirty-six hours before death, there was paralysis of the right arm and leg. The autopsy showed necrotic softening in the left hemisphere, especially of the posterior part of the first and second temporal convolutions and of the two annectant convolutions uniting the first temporal to the parietal lobe. There was complete transverse softening of the white matter between these convolutions externally and the lateral ventricle. *The gray and white matters of the occipital lobe were uninvolved.* The softening in the supramarginal gyrus was more superficial than in any other portion, and it seemed to involve only the gray matter. The gray matter of the angular gyrus looked wholly normal.

The concluding sentence, taken in connection with the clinical history, makes it evident that this was a case of subcortical sensory aphasia, the softening proceeding from the deeper parts toward the periphery. The lesion was a progressive one probably, but the patient died before the cortex of the angular gyrus became destroyed. This accounts for the writing-capacities of the patient, as stated by Osler.

Charcot and Dutil[1] put upon record the case of a woman, sixty-four years old, who had suffered during the last twenty years of her life four distinct attacks of apoplexy, after the first of which she was unable to write, although there was no speech disturbance. Following on

[1] Charcot and Dutil: "Agraphie motrice suivie d'autopsie." Mém. Société Biologie, 1893, p. 120.

a second attack, there were added difficulty of speech, left-sided hemiplegia, and eventually trouble in swallowing, all of which indicated a pseudo-bulbar palsy. Although the authors say that the agraphia had all the clinical characters of motor agraphia, it is to be noted that all forms of writing were interfered with. She copied defectively, she wrote from dictation very badly, and was incapable of writing spontaneously. At the autopsy there were found in the right hemisphere numerous small foci of softening and in the left hemisphere two such foci, one of which occupied the second frontal convolution and the other the inferior parietal convolution. In reality, this case was one in which the visual images were disordered, and the patient was not able to evocate them sufficiently in her internal language to give the complete idea of the word. Instead of its being a case of motor agraphia, the case was in reality one of sensory agraphia, as all the modalities of writing, spontaneous writing, writing from dictation, and writing from copy, were defective.

After taking all the evidence into consideration, of whatever nature, that can be cited in favor of the existence of a specialized graphic motor centre, and carefully weighing it, the conclusion is forced upon us that, so far, the advocates of the existence of such a centre have furnished us with no absolute proof. It seems to me entirely contrary to all that we know of speech, from a study of its genesis and its dissociation, to look for the existence of such a centre.

If claimants for the existence of a graphic centre are willing to admit that this centre coincides with the allocation for the hand and arm in the Rolandic area of the

cortex, and that writing is a hand motor function innervated from either hemisphere, as the case may be, I, for one, am inclined to agree with them. The product that writing externalizes, however, is the result of activity in a sensory area, the zone of language.

CHAPTER V.

Motor Aphasia.

1. Motor Aphasia; General Considerations. 2. Cortical Motor Aphasia; Articulatory Kinæsthetic Aphasia. 3. Subcortical Motor Aphasia.

There seems to be a unanimity of opinion among writers on speech disorders in using the term aphasia generically to designate the disturbances of speech that result from lesion of the zone of language, or of the pathways leading to and from the zone of language, whether these relate to its reception, its interpretation, or its emission; although, strictly speaking, aphasia occurs only when there is perversion of function of the zone of language. Aphasia may be classified, as I have pointed out in another connection, in a variety of ways. Disturbance of speech is manifested predominantly in its reception and emission, and as the reception of speech is dependent essentially on sensation, and only in a contributory way on motion (movements of eyes, ears, extremities—kinæsthetic sensation), the defect in speech that results from interference in the reception and interpretation of speech is called sensory aphasia. On the other hand, communication of thought by speech, and, in fact, communication of thought in any form, is mediated through movement. For the spoken word it is by the co-ordination of the respiratory movements, the movements of the vocal cords, the palate, tongue, and

lips; in writing, by the movements of the mobile part of the body holding the pen and acting under the conscious or subconscious direction of the visual centre; in the case of pantomime, by movement of the muscles of the face and of the extremities. A lesion that disables or militates against externalization of speech is termed, in a general way, and principally for convenience' sake, motor aphasia.

For a number of years after the universal recognition of aphasia which followed on Broca's description of his first cases it was supposed that the unique lesion of aphasia was in one very definite part of the brain. In the chapter on history we have seen how the labors of Wernicke and of others disproved this when they separated sensory aphasia. Then for a time, although it was fully recognized that aphasia was both motor and sensory, it was taught that motor aphasia occurred only when the gray substance forming the foot of the third frontal convolution, the seat of the articulatory kinæsthetic memories of words, was destroyed. Since that time, however, a new conception of aphasia has become dominant, and motor aphasia has lent itself to subdivision with a readiness equal to that of sensory aphasia. And to-day, thanks to the labors of Pitres, Dejerine, and other masters in the field of language, it is possible to differentiate cases of motor aphasia according to the seat of the lesion, whether it be in the articulatory kinæsthetic centre, in which are preserved the phonetic images, or whether it be at any level of the pyramidal projections extending from the Rolandic area, where the movements of respiration, phonation, and articulation are separately allocated, down to the

pons-oblongata, where the peripheral neurons of these pyramidal tracts begin.[1] Cortical and subcortical motor aphasia are dependent upon anatomical lesions which may be widely separated. Clinically, if they are studied with great care, they can be differentiated, although it must be said that the features which allow us to distinguish the one from the other are not quite so absolute and convincing as one might be led to infer from reading the authors who have contended for this differentiation in the past. Lichtheim[2] averred that subcortical motor aphasia was characterized by the loss of voluntary speech, by inability to repeat what was heard, by inability to read aloud, by the preservation of the comprehension of spoken and written language, and by the faculty of writing in all its forms—that is, voluntarily, from dictation, and from copy. Lichtheim suggested an apparently original test for subcortical motor aphasia to prove that patients preserve the memorial notion of the word; that is, that they have in their minds the name of the object which they are incapable of emitting. This test is not infrequently mentioned in literature as the Lichtheim test, but Pitres has pointed out that it was first suggested and utilized by Proust in 1872.[3] Nevertheless, Lichtheim's employment and advocacy of it caused its general utilization and acceptation. As the test is of great importance in differentiating cortical from subcortical motor aphasia, it

[1] I desire to repeat, even at the risk of tiresome repetition, that in the true sense of the term subcortical motor aphasia is in reality not aphasia at all.

[2] Lichtheim: "Ueber Aphasie." Deutsches Archiv für klinische Med., 1885, vol. xxxvi., p. 204.

[3] Proust: "De l'aphasie." Arch. Générales de Médecine, 1872.

is necessary to be explicit concerning it. The patient with cortical motor aphasia is devoid of articulatory kinæsthetic memory images. When he is shown the flower chrysanthemum, or when the word chrysanthemum is said to him, he sees the flower and he hears the spoken word; but when the impulses going from the visual centre and the auditory centre attempt to invoke the word chrysanthemum in its application to the flower seen or to the word heard it cannot evoke them in his internal language, because the area of the cortex in which articulatory kinæsthetic memories are stored is destroyed. This perversion of internal language is the distinguishing feature between cortical and subcortical motor aphasia. Internal language being disordered in cortical motor aphasia, there is always some imperfection in every modality of internal language, for it is a law that admits of no exception that when one of the physiological factors constituting or subserving speech is impaired all manifestations of speech will be disordered, just the same as when one portion of an intricate mechanical device is disarranged it interferes with the proper and harmonious working of the whole apparatus. This will be the case despite the fact that special conditions may determine the relatively greater prominence in any given consciousness of one element of internal speech over others. What these conditions may be it would be impracticable, were it not impossible, for us to say. Psycho-physiologists have given no clear account of the causes that determine the excitation of cortical nerve elements above the threshold of consciousness. To call the phenomenon a selective direction of attention to one of several possible mental elements, as does Bald-

win,[1] is only restating the problem in terms somewhat less direct and to me more difficult of comprehension.

To test this deficiency of internal language, Proust and Lichtheim showed that it sufficed to ask the patient who had heard some polysyllabic word, such as chrysanthemum, or had seen and recognized the object indicated by that word, to press the interlocutor's hand as many times as the word has syllables; for instance, in the case of the word chrysanthemum, to press it four times, and, if not by pressure of the hand, to indicate by some movement the number of syllables; then to indicate by similar pressings the number of letters in the word and the number of letters in the syllables. It will be readily seen that this test is merely the embodiment of the experience that every child has in learning to talk. When a child who is learning to talk and to read finds itself in the presence of a new word it splits it up into syllables and pronounces the syllables, if the child is moderately advanced in the acquisition of printed or written language; if it is not, it splits the word up into letters and pronounces each letter separately and aloud. If the articulation of these words aloud (or even not aloud—under breath, as it is called) calls up in the mind by the evocation of auditory memories similar words or objects or qualities of objects for which the word stands, the child comprehends. If it does not, the child has no comprehension of the word until after it has educated the articulatory kinæsthetic centre to associate the production of the word with that which the word stands for. The articulatory centre becomes cognizant of that word by learn-

[1] "Mental Development," The Macmillan Company, 1896.

ing the sounds of the letters and syllables of which it is composed, and when this area is destroyed this knowledge is lost; so that, when the patient is asked to indicate by sign or movement the number of letters or syllables in a word, he is quite unable to do so.

Subcortical motor aphasia spares the centre in which are seated the memories of articulation. The lesion interrupts the fibres that carry the speech impulses from the cortex, to which they have gone from Broca's centre, passing from the area for the movements of the lips, the tongue, the palate, the larynx, and the respiratory muscles, at some level of their course before they reach the crura. It has been contended by some writers, by Pitres,[1] by Brissaud,[2] by Juhel-Renoy, and Revilliod, that such lesions are not situated at some indefinite point, but that they are invariably situated at the level of the internal capsule, and that they are never in the so-called infero-pediculo-frontal fascicle, adjacent to the cortical gray substance; that a lesion of the latter location causes aphasia which does not differ from pure cortical aphasia. With this last statement I can in nowise agree. I have already pointed out in the chapter dealing with the genesis of speech why this symptom complex is usually associated with lesion very closely subjacent to the cortex. A lesion that cuts across the axones of the motor neurons constituting the pathway from the lip-tongue-laryngeal-respiratory cortex, no matter where this lesion may be, will produce the clinical picture of subcortical motor aphasia, even though it be of the very cell bodies of these neurons. On the other hand, unless the lesion be of the cells constitut-

[1] *Loc. cit.* [2] Article "Aphasie." "Traité de Médecine," vol. vi.

ing the articulatory kinæsthetic centre and their axones, which I believe to be entirely association tracts and not at all projection tracts, the features of the aphasia will not be those of cortical motor aphasia. Nevertheless, I frankly admit that it is very much more difficult to make a diagnosis of subcortical motor aphasia when the lesion is situated contiguous to Broca's centre than when it is situated at some distance. It must be kept in mind, however, that it is not easy in any instance to make during life a diagnosis differentiating between cortical and subcortical motor aphasia. The truth is that the Proust-Lichtheim test is not very readily applied, because many of the patients with whom we have to deal are quite incapable of comprehending what we are striving for when we endeavor to utilize it and what we wish them to do; but when it can be applied it is a test of great value.

The most striking picture of cortical motor aphasia is the loss of speech, articulate or non-articulate, which is manifest on attempting to make voluntary communications, on attempting to repeat what is heard, and on attempting to read aloud. If the lesion is closely restricted to Broca's convolution, these are the leading features of the aphasia. The patient understands all that is said to him; he gets the proper apperception of visual objects which leads to the formation of concepts; he receives trustworthy information from other sources on which he has been accustomed to rely for information, at first hand or for corroboration, and they in turn endeavor to invoke the images of articulate speech, which images normal man utilizes in intercourse with his fellows and which many persons, particularly the illiterate, use in thought. The

area of the cortex in which such images of articulative speech are stored is destroyed, and as the result of its destruction its function is abolished. The lesion that causes motor aphasia is in the great majority of the cases a vascular one, and as the same blood-vessel, the left middle cerebral, is the principal medium of arterial supply for the remainder of the speech area, it is only in exceptional instances that destruction of Broca's convolution is not accompanied by some anatomical perversion of other parts of the zone of language, although these are usually transitory. There is invariably some perversion of function of the other speech centres, because perfect speech demands the harmonious co-operation of all the speech centres, and one cannot be disordered without entailing derangement of all.

Cortical Motor Aphasia. Articulatory Kinæsthetic Aphasia.

By cortical motor aphasia I mean a disturbance of speech due to loss of the sensory images of articulation associated with loss of the sensory memories of co-ordinate movements entering into vocal expression; the latter is not essential, but it is an accompaniment in nearly every instance. Cortical motor aphasia is characterized particularly by a loss of spontaneous and repeated speech and by the preservation of the capacity to comprehend spoken speech. The peripheral speech mechanism—the tongue, lips, palate, and vocal cords—is in condition to functionate. The only justification for the use of the word motor in this form of aphasia is that the images of articulation are called into being by movement and are externalized by

movement. Therefore in true cortical motor aphasia there exists the same inability to call into being the sensory memories of articulation, and thus to make them a part of internal speech, as there is to externalize them in the shape of articulate words. Many of the cases of aphasia in the literature which are considered to belong in this category are not of this variety at all, but are examples of *pure* motor aphasia (of Dejerine), or subcortical motor aphasia; that is, disturbance of speech dependent upon interruption of the projection tracts which convey the articulatory impulses from the cortical area of the peripheral speech mechanism to the peripheral speech apparatus. In fact, not much more than one-half the reported cases have been studied with sufficient care to warrant including them in this category. And this is so for many reasons. In the first place, the centres of the speech area in which are stored definite memories are rarely involved alone. In the great majority of instances the lesion is of vascular origin, and the nutritive supply of one centre or the structure of the centre itself is not perverted without a material disturbance of function in some of the other centres. And in the second place, it is only within recent times that the possibility of, and the necessity for, differentiating this form of motor aphasia from the subcortical form has been generally understood. The motor area of the brain should be, as I have said before, limited to that part of the brain from which the motor projection system of fibres passes, and there is no unassailable evidence to show that the inferior frontal fascicle, which has been allotted as the system of projection fibres extending from Broca's convolution, has anything to do with this purpose. Until within

a very recent time, motor aphasia, which was described by Broca as true aphasia or aphemia, by Wernicke as motor aphasia, and by Kussmaul as ataxic aphasia, was supposed to be fully understood and most thoroughly established on an anatomical foundation. But the writings of Dejerine and his pupils, of Wyllie, of Elder, of Onuf, and of others, and the labors of the physiologists, particularly Horsley, Krause, Masini, and Semon, have shown the necessity of rewriting the chapter on motor aphasia and emancipating it from the dicta of the older writers.

As a case of cortical motor aphasia in which the symptoms indicated that the lesion was very closely limited to Broca's convolution, the seat of articulatory-kinæsthetic images, I may cite the following:

A young man, thirty-two years of age, was admitted to my wards in the City Hospital, suffering from an incomplete right-side hemiplegia. His history antecedent to the attack, which had occurred five months previously, is of no interest, except that he had had two attacks of inflammatory rheumatism. After the apoplectic stroke, which came suddenly in the night and which was followed by loss of consciousness, he was unable to talk, and this condition obtained when he entered the hospital. The apoplectic stroke was considered to be of embolic origin, as examination of the heart showed well-marked incompetence of the aortic valves. Investigation of the speech defect showed that he was unable to communicate his thoughts by spoken words, that he was unable to repeat from dictation; that he was unable to write, save his name, and that very slowly and laboriously with the left hand; and that he was unable to indicate the number of syllables in a word previously seen or heard, by pressing his interlocu-

tor's hand as many times as there were syllables. He interpreted quickly and easily everything that was said to him, but he could not write from dictation. There was no trace of word blindness or of letter blindness, and he copied letters and words correctly. On account of the paralysis of his right arm, he was not able to copy quickly, but if he was given sufficient time he showed that he was quite capable of doing this. When he was given a paragraph in a newspaper and asked to copy it, he copied it in writing. The patient was able to read and apparently to comprehend what he read, but he never took any interest in the newspapers, nor was he seen to read anything spontaneously during his stay in the hospital. That he comprehended written and printed requests, however, was shown by the readiness with which he obeyed requests when they were communicated to him in this way. The patient remained in the hospital but a short time, and only one opportunity was had to test his ability to write by means of individual letters. On this occasion a number of each of the letters of the alphabet were put before him on the table and he was requested to construct the sentence, "The President of the United States is inaugurated on the 4th day of March." This sentence was completed after the expenditure of a considerable time, and, save for two or three mistakes in spelling, it was correctly done.

This brief rehearsal of the symptoms in this case shows that the speech defect was, strikingly, loss of the sensory images of articulation. The patient, though in full possession of the avenues by which one receives stimuli, auditory, visual, and kinæsthetic, necessary to speech production, was unable to speak any words except "Yes" and "No," the former of which was enunciated most indis-

tinctly. That this loss of power of articulation was dependent upon lesion in the zone of language and not upon the projection tract of pyramidal fibres is shown conclusively by the fact that the patient could not call up the articulatory kinæsthetic memories of words. For in every instance when he was tested in this way he failed to indicate by pressing the hand or by holding up the fingers the number of syllables in the word desired.

From this it will be seen that the essential accompaniment of cortical motor aphasia is: loss of spontaneous speech, due to a destruction of the sensory images of articulation stored up in the foot of the third frontal convolution. Associated with this loss of spontaneous speech there is a loss of all forms of speech utterance for which an evocation of articulatory kinæsthetic memories is required. Therefore there are inability to repeat words and inability to read aloud, but the patient comprehends spoken words, oftentimes somewhat imperfectly. There is inability to express thoughts in writing, because in writing the motor word representations are always revived by the impulse which travels from the percipient centre (which is either in the visual area of the brain in spontaneous writing, or in the auditory speech area in writing from dictation) through the articulatory kinæsthetic centre to that part of the Rolandic region which guides the mobile part of the body holding the pen. On account of the ontogenetic intimacy existing between the receptive speech centres and the emissive, disturbance of the emissive speech centre almost invariably produces some disturbance which is manifest through the former. This is shown in cortical motor aphasia by difficulty in calling

up promptly and with readiness auditory word images to which articulatory kinæsthetic images are subservient, and in some degree by a disturbance of internal reading. In the great majority of people it is probable that reading to one's self is accomplished by evoking the images of articulation, and that as a matter of education, of expediency, a short cut is established between the area in which visual images are stored and the association tracts constituting the anatomical basis of comprehension.

In cortical motor aphasia there is sometimes very complete amimia. This is to be explained by the fact that studied pantomime is associated normally with arousal of the images of articulation.

If destruction of Broca's area is total, or nearly so, the capacity for articulate speech will be correspondingly complete; while if the convolution of Broca be only partially destroyed, and particularly if the lesion be a vascular one, such as plugging of the branch of the middle cerebral artery that suplies Broca's convolution, with subsequent exudation, a reparative process may set in. Then the degree of the completeness of the aphasia bears some relation, though just how much cannot be said, to the amount of repossession of articulated images. It seems to me that this offers quite as satisfactory explanation of the possession of certain words or the acquisition of a few words by patients who have a lesion of Broca's centre as does the view that attributes such partial recovery of speech to the vicarious assumption of function by the so-called uneducated centre of the opposite side, of which so much has been made by many writers in accounting for recoveries and for the utility of certain modes of treatment. I

hasten to say that I do not deny that the corresponding centre of the other side, although not ontogenetically intended for speech, may and does take up in a crude compensatory way the function of the speech area. This subject will be referred to again, in the chapter on "Treatment." Nevertheless, I believe that it never does so except by process of education; it does not seem to me that there is any spontaneous assumption of function. Naturally, I cannot offer any tangible or incontrovertible evidence in support of this belief, but here my position is not very different from that of those who hold to spontaneous assumption of speech function by the uneducated side. They cite in support of their contention cases which I believe to be typical examples of subcortical motor aphasia. Such a case has been reported by Wyllie:[1]

A young man developed a right-sided hemiplegia, probably of thrombotic origin. The attack came on gradually and in successive stages. At first he was completely speechless, but soon regained ability to say "Yes" and "No" and to *answer questions in writing.* "*He states that he never from the first had any difficulty in calling up in his mind the words that he wished to write*" (italics mine). This, it seems to me, stamps the case as one of aphasia due to lesion *outside* the zone of language. If there was no other evidence than this, it would be sufficient to convince me that he had likewise no difficulty in calling up the words that he wished to utter, for people of his education usually spell words articulately when writing them. But witness testimony to this effect from the patient: "With regard to my forming words in my mind, i could always form them in

[1] Wyllie: "The Disorders of Speech," Edinburgh, 1894, p 323.

my mind but tro ible was in getting them out. i could not get them out at all and after making all the signs i could think of to my friends if they could not understand me i would have to take paper and pencil and write it down for them."

This, I venture to believe, is conclusive evidence that the patient had no defect of internal language, such as invariably accompanies destruction of the articulatory kinæsthetic images in Broca's area. The patient recovered the use of the paralyzed side, and consequently it excites no astonishment in me that he recovered the power to externalize speech commensurably with this; nor can I for a moment consider it an example which goes to show how "the previously uneducated centre educates itself." Moreover, it does not seem to me that the case is remarkable, "inasmuch as it is one of that comparatively rare variety in which the patient, utterly aphasic as to spoken speech, is yet capable of expressing himself in writing." I shall hope to show in this chapter that subcortical aphasia is not rare, and that it is characterized by the retention of the capacity on the part of the patient to express himself in writing.

There are on record a number of cases which illustrate a partial destruction of Broca's area with preservation of the ability to write and repossession of the ability to speak. Of such cases the most remarkable is one recorded by Banti.[1]

This patient, a man thirty-six years of age, right-handed, capable of reading and writing, was stricken suddenly with an attack of apoplexy, while walking the street, fol-

[1] Banti: Sperimentale, Firenze, 1886.

lowed by loss of consciousness. Consciousness returned after a few minutes, but he was paralyzed in the right arm and right leg and was wholly unable to speak. The hemiplegia disappeared almost completely during the following night, but the inability to speak still continued. On the following day he was admitted to the hospital and there examined by Banti, who noted that the motility of the extremities was normal and that there was no trace of paralysis of the face or of the tongue. The patient made futile efforts to speak, but he was incapable of articulating a word or a syllable. He chafed exceedingly under this mutism and sought to convey his meaning by gestures. On being asked if he could write, he made a gesture of affirmation, and on being given a pencil immediately wrote his name. To numerous other questions he responded with accuracy in writing, and without hesitation wrote a detailed account of his disease when he was requested to do so. He wrote also the names of objects that were shown to him, and of objects whose qualities he heard. He answered questions that were asked in writing with perfect precision. He comprehended script and print equally well, seized the sense of a question very quickly, and wrote rapidly and without the slightest hesitation. But he was unable to pronounce or articulate a word. The aphasia ameliorated gradually, so that in less than three years he was able to express himself freely. Nevertheless, attentive examination showed slight defects of articulate speech. Five years later the patient died of an aneurism of the aorta; and on autopsy there was found a patch of yellow softening situated in the posterior portion of the third left frontal convolution, and separated

from the white substance by a portion of tissue only a few millimetres in thickness. In this case it does not seem to me that there can be much doubt that a part of the area in which are stored the images of articulation was undestroyed. In no other way can be satisfactorily explained the remarkable degree of preservation of writing-capacity and the rapidity with which he regained speech possession.

It is more than likely that our attention has been centred too exclusively on the unilateral representation of the speech faculty. It is possible that the right side of the brain contains appercipient speech centres which are in direct anatomical and physiological association by means of the fibres of the corpus callosum. The apperceptive centres of the right side of the brain do not, however, constitute an autonomy, nor do they seem to have any effect in determining the execution of speech functions. Therefore when the right hemisphere is the seat of lesion in right-handed persons, there is no trouble in the execution of speech. The influence that has most to do with determining the superiority of the left hemisphere as the speech side of the brain, after ontogenetic influences, is education. Just what the antecedent factors are in this determination it is impossible to say. The general speech education, so to say, that the opposite hemisphere receives has much to do with the possibility of educating it to compensate in part for destroyed centres of the zone of language.

The capacity for articulate expression which a few, compared with the entire number of motor aphasics, retain is for a few words whose utterance partakes more of the nature of a reflex act or of an emotional possession than it

does of a process of intellection. They consist of "Yes" or "No," the patient's name or address; possessions which from long usage signify one definite purpose or thing and are uttered nearly reflexly. In like manner, they sometimes retain the capacity to use conventional expressions, such as "Good morning," "How are you?" "Very fine," etc., and these they employ for every occasion, pertinent and non-pertinent alike. Oftentimes these expressions are used by the patient in a way that reminds one of an echo.

Whether such reflex or echo-like words cannot be produced by the action of sensory impulses directly on the central executive motor speech mechanism, I have often debated. The answers that such patients give are comparable to the speech of a parrot, and no one believes that a parrot has an ontogenetic speech centre for memory images of articulation. Sensory excitations call forth uniform responses in the parrot with tiresome monotony, and it is the same way in the patients; they may respond "Tan, tan," or "Yi, yi," or "Yes" to everything. Echoing or echolalia in cortical motor aphasia is a phenomenon that is occasionally present, oftenest when the patient is recovering. The occurrence of echolalia has been thought to indicate the assumption of function by the uneducated side. The patient repeats a word which he has heard, or a sound, in the same senseless fashion and with the same tireless persistency that a child does who is learning to talk. It may possibly be an indication of the functioning of a centre on the right side of the brain which is not yet fully under control, inhibitory or exciting, of the sensory apperceptive centres. On the other hand, it may be the manifestation of a certain retention of function by the dis-

eased area. The disorganization of Broca's convolution being sufficient to rob it of all purposive activity, it retains a semblance of function pointing to what it once was.

Another very striking variety of articulate speech which patients with motor aphasia show is that to which the term lalling or lallation is applied. The significance of the term, "baby talk," is indicated by its derivation. It, like its congeners, stammering and dysarthria, is a symptom of subcortical motor aphasia, and rarely of the cortical variety.

The degree of completeness of the loss of articulate speech is, as has been said, a variable quantity. Although in cortical motor aphasia the power to make voluntary expression is usually entirely gone, the loss may be partial, nearly all words or only a certain class of words having perished. When partial, the power of expression is limited, as a rule, to one or more monosyllabic words, which the patient may use intelligently but which are not infrequently used in answer to every question—for instance, the word "No" to express negation and assent alike. In other cases, the ability to articulate certain vowel sounds, such as *a*, *o*, *u*, or some of the consonants, may be preserved. Occasionally, patients who are afflicted with complete motor aphasia are able to utter some words of the nature of an oath, which seem to escape from them in a rapid, uncontrollable way, or to ejaculate words expressive of the feelings. Such expresions are not the product of cognition, but of the emotions, and partake of the nature of reflex action. Other patients repeat continually some expressive or meaningless word or words. These repeated words or sentences, though often of no

pertinency, seem occasionally to have a definite meaning, in so much as they are connected with the onset of the patient's disease. Thus, a patient who had complete motor aphasia, observed by Hughlings Jackson, kept repeating, "I want protection." The man had received a serious injury of the head during a quarrel, and it has been suggested that in such a case (there are many of them on record) the frequently repeated word or phrase was the last one uttered by the patient before he became aphasic, and that these words, the last enregistered in the centre of articulatory memories, are the ones most promptly evoked when sensory impressions reach it.

Such recurring utterances are distinctive features of cortical motor aphasia, and not of the subcortical variety, for the latter really is a shortcoming of articulation, and if articulation is not wholly impossible the defect is manifest by some variation in the perfectness with which the word is produced.

In the ordinary case of motor aphasia, although the emotional faculties are often disturbed, the patient is capable of giving spontaneous expression to his feelings when such expression does not require the use of words. The patient's intellectual faculties do not on casual examination seem to be materially impaired, although there is usually an indisposition or lack of readiness on the part of the patient to indulge in anything requiring mental application. On that score cortical motor aphasia differs most materially from subcortical forms. Patients with the latter are as ready and willing and capacious to attack mental problems as normal man, and not infrequently transact business calling for great mental integrity and

indulge in games requiring profound abstraction, such as chess. Patients with cortical motor aphasia often show great amnesia and lack of comprehensive grasp of facts that have been communicated to them since their illness. This is easy of interpretation if it be borne in mind that lesion of Broca's area entails perversion of the function of internal language. He has lost the ability to repeat inwardly what is said to him, what he hears; and as most people obtain knowledge for storing up in this way, the person with true motor aphasia is deprived of the power of thus laying up knowledge. This, combined with the dyslexia or alexia which unfits him for obtaining knowledge by reading, is abundant explanation of the amnesia.

Cortical motor aphasia is sometimes manifest merely by a loss of substantives; amnesia of the names of the things or objects of which the patient tries to speak. For instance, a patient who wishes to request something may be able to produce every word except the one which is the embodiment of his request. A patient of Trousseau would say, "Give me my—u—u——*sacré matin.*" "Your umbrella?" "Yes, my umbrella." It seems to me very questionable, however, that this is a genuine example of amnesia of the articulatory memories for substantives, and the fact that he was able to say the first letter of the word in the name of the desired object is rather in favor of its being a case of subcortical motor aphasia, the stumbling and stammering being apparent when he attempted to utter particular names. A much more convincing example of the loss of the articulatory memory of substantives is furnished by a patient who, knowing just what he wants, instead of saying, "Give me my

watch," says, "Give me what I tell the time with," or who calls a knife "something to cut with," and so on. In these cases the amnesia for substantives may be the only aphasic symptom. Very much less common is articulative amnesia of verbs, the ability to use substantives being preserved. When it is recalled how very much more complex the formation of a mental percept of a name is than that of an object, and how much easier it is to recall the qualities, capabilities, and possessions of objects than the name of such an object, it will be understood that articulatory amnesia of substantives is much more common than articulatory amnesia of verbs.

The retention of speech capacity that patients with articulatory kinæsthetic aphasia sometimes have is very surprising. In addition to the form of which I have just spoken, in which the speech defect is confined to loss of substantives, others are occasionally observed in which the patient is unable to repeat any words except to count or to say the multiplication table. Such an instance has been recorded by Volland.[1] The patient was completely speechless, except that he could count up to one hundred and say, "Once one is one," etc. A much more interesting retention is that sometimes met in polyglots, *i.e.*, in patients who before their illness could speak with fluency other languages than their mother tongue. The loss of speech in such an individual may be complete for one or all of the foreign languages, while for the mother tongue it may be only partial. As a rule, however, in these cases the ability to speak is entirely lost in the beginning of the

[1] Volland: "Aphasie après une blessure de la tête; conservation de la faculté de compter." Münch. med. Wochen., 1886, No. 4.

disease, but after a time the patient may gradually regain the capacity to speak his mother tongue, but not the foreign languages, the languages that he acquired at a later period in life than the mother tongue. Such cases have been reported by Charcot and by others, and the subject has recently been studied very carefully by Pitres.[1] The explanation given by this writer is one that must meet with universal acceptance. The phenomenon does not in any way posit the existence of separate word-image centres for different languages. On the contrary, it is explained by the fact that the word images in one's mother tongue are more indissolubly imprinted or enregistered in the area for the storage of word images, and consequently they are the least readily dislodged or destroyed with lesions of this area that are not of sufficient intensity to destroy it completely.

Inability to recall substantives or verbs is the condition to which the name amnesia verbalis is given. This condition is to be distinguished from that to which the term ataxia verbalis is sometimes applied to indicate the disability of producing and co-ordinating the movements that subserve articulation, and which is such a prominent feature in subcortical motor aphasia that the term ataxic aphasia has been applied to it. Wyllie deprecates the fact that the term asynergia verbalis, suggested originally by Lordat to denote the disablement of speech in motor aphasia, has not been retained, as it means the want of power of working together, as this is really what exists in subcortical motor aphasia, and not that which is sug-

[1] Pitres: "L'aphasie chez les polyglottes." Rev. Méd., November, 1895.

gested by ataxia, which means want of order. But I cannot agree with the distinguished Scotchman in this, for the term asynergia verbalis, although extremely pertinent, does not encompass the defect entirely; there is ataxia as well.

According to the conception of speech promulgated by Charcot, the amount of amnesia verbalis in motor aphasia should depend very largely on the category to which the patient belongs. If he were a *moteur*, that is, if he depended largely on the evocation of articulatory memories to jut thought or memories into consciousness, then destruction of the cortical area in which are stored such memories would reduce the vocabulary of the patient much more materially than if he were an *auditif* or a *visuel*. If the patient were an *auditif* and called up words and names habitually by a revivication of auditory images, articulate verbal amnesia would be, compared with the previous condition, very slight. The logic of this is unassailable, but unhappily for the advocates of the view the premises are false, and the conclusions are not in accord with clinical experience. Probably no one speaks from a primary evocation of the images of articulation, and the loss of evocation of names and words in partial motor aphasia is due to the fact that there is always a disability in the arousal of auditory images spontaneously in this condition.

A patient with cortical motor aphasia is unable to express his thoughts in writing. His capacity to write is proportionate to the amount of derangement of internal language, and it bears a definite relation to the amount of latent or actual visual amnesia of words which every patient

with cortical motor aphasia has. In most cases the capacity to write is limited to writing the name and a few other words, such as the age, the address, and the name of the wife or the parents, that have been done so habitually, automatically, and frequently that they form a part of the patient's habitual acts, and are done almost reflexly. Motor aphasia is almost invariably associated, at least in the beginning, with right-side hemiplegia due to extension of the lesion on which the aphasia is dependent to the psycho-motor zone, and this hemiplegia prevents the patient from writing with the right hand. Although most educated people can write with the left hand, of course more clumsily, less gracefully, and with greater effort, than with the right hand, they can accomplish it; and if for any reason the right hand becomes disabled they acquire within a few weeks the ability to write with the left hand with ease and facility. But a patient with cortical motor aphasia is completely agraphic, never learns to write with his left hand, no matter how long he may live after the aphasia has developed. However, every modality of writing is not interfered with; though writing voluntarily and writing from dictation are practically impossible, yet the patient is able to write from copy.

This preservation of the capacity to write from copy is entirely in harmony with the explanation that suffices for loss of the ability to write that occurs in cortical motor aphasia. The inability of patients who have cortical motor aphasia to write spontaneously and from dictation has been recognized almost from the beginning of the history of the disease. Trousseau gave much space to a

discussion of it in his lectures on aphasia and cited many examples. In the English language the subject was fully discussed by Ogle and by Gairdner, particularly by the latter, who said very tersely and truthfully that the motor aphasic could write as well as he could speak, and if unable to speak he was also unable to write. There would seem to be entire unanimity of opinion of the occurrence of agraphia with cortical motor aphasia, but different observers put widely different interpretation on the manner of its occurrence. Those who follow the teachings of Charcot, Exner, Stricker,[1] *et al.*, and believe that there exists a special centre for the deposition and storage of graphic motor images, have no difficulty in explaining it by saying that as the graphic motor centre is adjacent and contiguous to the centre for images of articulation the one is almost always affected with the other. They experience a very serious difficulty, however, when they attempt to prove the existence of such a hypothetical centre, as well as when they seek to defend the autonomy of any one centre in its relation to finished speech or to its predominancy in furnishing us with the idea of a word. Of all the cases of aphasia accompanied by autopsical details on record there is not one that can be used successfully to prove the separate existence of a graphic motor centre. As pointed out in another chapter, the existence of such a centre is opposed to the conception of speech which it has been our endeavor to set forth in these pages.

At the time of Trousseau and Gairdner, when sensory

[1] "Zur Lehre von der Aphasie." Wiener med. Blätt., 1881, vol. iv., pp. 1,477, 1,509, 1,565.

aphasia was as yet scarcely differentiated, the explanation that was given to account for the occurrence of agraphia was more nearly in accord with the explanation that I believe to be the correct one than that which many writers give to-day. It was believed that in these cases the lesion extended posteriorly to include other areas than that of Broca's convolution. And as a matter of fact that is just what sometimes occurs. In every case of cortical motor aphasia there is some dyslexia, which shows that the visual centre in which are stored the images of the seen word (the angular gyrus) is functionally impaired. This impairment may be slight, and if the examination to reveal it is made some time after the occurrence of the aphasia it may be difficult to bring it out. It has been shown conclusively by Thomas and Roux[1] and by others that, nevertheless, it exists, though possibly in a latent form in every case. As a rule, the verbal blindness is slight and undergoes restitution even before any signs of improvement are manifest in the aphemia. Agraphia occurs, then, in motor aphasia because the idea of the word that the patient wishes to write voluntarily or from dictation is lost. In writing voluntarily it is necessary in every instance to visualize, that is, to call up the visual memory or image of the letter and the word, and these in turn, acting upon the images of articulation, evocate them, and the latter guide the motor area in making the movements that transcribe the letters to form words. In educated people and people habituated to writing, it may be necessary to arouse the articulatory images; a short circuit may be established between the

[1] Bull. Société Biologie, February 22d, 1896.

visual images of the word to be written and the motor area from which originate the movements of the part that grasps the pen, but it is extremely improbable that this takes place. It is much more likely that the sensory images of articulation are evoked in every instance. If one watches a person unaccustomed to writing, or a beginner, indite a line or a page, he will find almost invariably that either the lips and the tongue move visibly with the tracing of each letter and word, or the words are distinctly articulated. Moreover, if one asks typewriters who are accustomed to take dictation directly on the machine how they transcribe what they hear said to them, it is probable that the majority will say that they do so by spelling to themselves (but still most audibly to themselves) the words that they hear, before striking the corresponding keys. This shows that there is with the visual image of the word a corresponding evocation of the articulatory images. When the area in which are stored the articulatory images is destroyed, then a link in the circuit by whose integrity we are in possession of the proper idea of the word, and so capable of utilizing it in any form of exteriorization, is gone, and the result is failure to write it and failure to speak it.

This loss of the idea of the word is one of the distinguishing features of every form of cortical aphasia and the absence of it is the most pathognomonic accompaniment of subcortical aphasia, it matters not what the degree of the aphasia is. In subcortical motor aphasia there is no dyslexia, because there is no word amnesia of any kind, visual, auditory, or aphemic, and the patient can write with the left hand, or with the right if not para-

lyzed, with as much ease as he could before the illness. This ability to write is dependent upon the possession of the conception of the word, and particularly upon the ability to evocate the visual image of the word. When such evocation is possible, voluntary writing and copying from dictation are done without hesitation by any mobile part of the body accustomed to hold the pen. The right arm being almost invariably paralyzed in subcortical motor aphasia, this prevents writing with the right hand.

Writing from copy is possible in motor aphasia and in subcortical motor aphasia, as has already been said. Writing from copy cannot be looked upon as an act necessitating realization of the idea or concept of the word, and it therefore may be retained when such idea or conception is lost. It is probable that copying involves no intellection except that entering into seeing the word as a design, and then the copying of the design, conditioned by a psycho-motor act. The notion of the word may not enter into the internal language at all, and unless the patient reads what he is copying, and therefore revivifies the visual memory images in the angular gyrus, he may copy writing as he would copy a figure with which he is not familiar. The fact that these patients copy print in script and not in print, however, shows that they do not copy mechanically and servilely, and this is explained by the fact that in no case of motor aphasia is there absolute inability to revivify visual memories; there is only partial disability, sufficient being left to orient the patient in copying. The other reason is that man is habituated from childhood to write in script and not in print, and he does this unconsciously from such habituation. Dys-

lexia, difficulty in reading (reading to one's self), is a common accompaniment of cortical motor aphasia, although it may be wholly recovered from and often exists only in a latent state. In some instances the difficulty in reading is so pronounced as to constitute real alexia. As an instance of well-marked dyslexia, even in a case of motor aphasia that has nearly recovered, I may cite the following instance, which may at the same time serve as an example of typical motor aphasia:

Philip Masterson, thirty-one years of age, a right-handed, hard-working, temperate man, the father of three healthy children, was stricken suddenly while at the dinner table with a complete right-side hemiplegia. There was no coma, or at least it was not sufficient completely to obliterate consciousness, although he was in a dazed state for several days. Loss of speech existed from the first. A few days later he could apparently understand what was said to him, and made some efforts to answer, yet no articulate words came to him; but after endeavoring to answer for some time he suddenly blurted out the word "Lizzie" (his wife's name), and thereafter he repeated this word almost incessantly when he was spoken to and when he was not, and when he wished something or when he desired to be let alone. It partook of the nature of a recurring utterance. After a number of weeks he was able by pointing to indicate what he desired or what he meant to convey. The first word that he spoke besides the word "Lizzie" was after he had received a visit from a man with whom he had worked for many years. When the visitor had gone he very quietly articulated the word "Lynch," although there had been no conversation about the visitor after he had left. He was completely agraphic, unable to form the most crudely printed letter with the left hand.

He understood when spoken to, would obey quickly and intelligently simple requests when they were of such a nature that a partially hemiplegic man could obey, and he apparently put proper interpretation on impressions received through the optic nerves. When any one spoke to him he watched the movements of the lips very attentively, and sometimes tried to articulate, but never with any success. Six weeks after the stroke he was able to articulate the words "Yes" and "No," but in response to the invitation that he should tell something about his illness, he was quite mute. Gradually he began to use verbs and afterward to apply names to persons and things. For instance, in calling the children, he would apply the name of some person with whom he had worked, or of some unknown person, or even the name of some object in the shop where he had worked. Instead of saying, "Give me my stockings," he said, "Give me those kittens." When he asked for his vest he said, "Hand me that clock." One day he wished to have a sponge, and endeavored to convey his meaning by saying, "Where are the chickens?" Immediately after making such errors, which were oftentimes very ludicrous, he would recognize the impropriety, and particularly if he noticed some one smiling, and would immediately assume a facial expression as if searching for the right word, and wind up by saying, "That's damn funny." If he were asked the name of his neighbor, he would at once and without the slightest hesitation say, "Robinson," but would recognize his error, and on being asked if it were not O'Connor he would say, "Of course it is; I guess I ought to know." At this time he exhibited well-marked paraphasia. If the sentence contained no proper names, he would use the requisite and proper words to convey his idea, but they did not come from his mouth with the right sequence. This was some-

what more marked when he endeavored to repeat sentences and when he tried to read aloud. It was also present when he essayed to speak sentences that he had formerly learned by heart, such as the Lord's Prayer, or simple verses of poetry.

He was very loath to try to write with the left hand, and it was only with the greatest difficulty that he could be induced to write his name and address, but after some persuasion, fortified by assurance, he did so. He was able to copy, and copied script in script, and print in print, but he was quite unable to write anything from dictation.

On being given a number of detached letters, and requested to make words out of them, and then a sentence informing me when he had last been to the hospital, he framed with great difficulty the words, "Last Thursday."

On testing him for powers of association and applying proper names to certain things and objects, I said to him, "If on your way home, you see great black clouds coming up in the sky, and it begins to get dark, what do you think is going to happen?" He answered very promptly, "A storm." A number of other questions of similar import were answered correctly. One of his answers is so thoroughly in accord with the teachings that one's thoughts are largely reactionary to his environment that it deserves to be quoted. I said to him: "Suppose you were on your way here, Sunday morning, about ten o'clock; you hear the bells ringing, and see a great number of people going into a large stone or brick building, some going in singly, others in couples, some in groups; what would you call the place where they were going?" He hesitated a moment and then said: "A saloon, I suppose," and the absurdity of it was not apparent to him.

The following is a stenographic report of an interview with him: .

Patient (reading from newspaper). — Lead pipe his mande hen of powerful presses. This medi in operation before he invented the lead pipe places—press—the lead pipe—no—they was about every—s-sway—lead was cuts in pieces of the length of foot or two, which were the roller—which want, then rolled between grow and rollers into lengths of to—of about ten feets. Length being the sahder, the solder, the solder together make a marse, and some longer by another meadows, meadow—ma-madow—shaped lead was cut off, the lengths rolled up, an so—and sa—and sader—(cannot say that now)—and another sader lead pipe was drawn in thump in something the same manner that wire was drawn—ma-a—now wait—that the metis required. (Lost place entirely; cannot go on.) The lead pipe presses in on the new. No, it is not there. The lead pipe press is not new.

Q. Do you remember what you have been reading?

A. Yes, sir.

Q. You do remember what you have been reading? Now tell me slowly what you have been reading.

A. Oh, about the lead pipe and how long them are cutting it, and how big it was getting. I don't know of any more.

Q. Can't you remember anything else?

A. No, not as I know of, unless—— (hesitates).

Q. How is lead pipe made?

A. It is dumped into the boiler, isn't it?

Q. Is not lead pipe made by means of powerful presses?

A. Yes, I guess they are.

Q. Before the presses were invented how was lead pipe made?

A. Oh, I don't know how that would be.

Q. Why don't you know?

A. I don't know; but I don't remember.

Q. Have you not just read here how it was made before the presses were invented?

A. Yes, in that paper? Yes.

Q. Then why don't you tell me?

A. I don't know.

Q. Did you understand what you read here?

A. No, not at all.

Q. Why did you not understand it?

A. I don't know.

Q. Before you were sick you could read English and understand it, could you not?

A. Why, of course; I wouldn't look for anything. I would look around the front of the paper, of course, and would not read it all.

Q. The manufacture of lead pipe ought to interest you.

A. No, sir; it did not arrest us—no, we did not make anything out of it.

Q. But you are interested in learning how such a common thing as lead pipe was made?

A. No, sir; we were not exactly that; we were but lead, but copper, and rocks.

Q. Do you see the words when you attempt to read?

A. Yes, sir.

Q. You can see the words, following on one after another?

A. No, sir; I sometimes skip over a line.

Q. Why is that?

A. I don't know.

Q. Even when you read a verse, do you understand what you have read?

A. No.

This alexia which is now so manifest in attempting to read is probably explained by the fact that the patient always in reading evocated the articulatory word images. That is, the impressions received through the visual images were transformed into articulatory word images before they were carried into the consciousness, and now, when the articulatory word images are partly destroyed, visual impressions, although properly received, do not fully enter into consciousness.

Q. Now, I want you to repeat after me, the Pilgrim Fathers landed on Plymouth Rock on the 22d of December.

A. The plim—on the plim—the plim—no, the plimbic rock.

Q. Repetition of sentence.

A. The pil—the pim—the rock. The—can't get that out. The pim—— (mutters).

Q. Now we will try another sentence. The Declaration of Independence was framed by Benjamin Franklin.

A. The bec—the bec—the bet of independence—no—— (hesitates).

Q. Now I am going to ask you to repeat something very much easier. Before the foreigner can become an American citizen he must live here five years.

A. Before a for'ner who had lived here—had—(mutters) had to go back. Is that it?

Q. (Repeating the question.) You know that is the fact, do you not?

A. Yes, sir. (Tries to repeat.) Before a commoner, comner, before a commoner who had lived here—I couldn't go no further.

Q. Now, I shall give you another question. The Papal Delegate is the head of the Catholic Church in America.

A. The papal dedicate was—a—was—the—was—the papal—the pap—cake—oh, I couldn't.

Q. Shall I repeat it for you? (To wife.) You repeat it. (She cannot remember it all, whereupon the patient says very quickly: He is a head of the U. S. of America.)

Q. Repetition of sentence.

A. The Papal Delicate is the head of the United States of America.

Q. Now I am going to give you a very easy one. To go to the theatre is a pleasure.

A. To go to the theatre is a pleasure.

Q. To go to the theatre after a hard day's work is a restful pleasure.

A. To go to the theatre is a hard day's work—I don't know more—you run away too quick.

Q. (Repeats.)

A. To go to the theatre is a hard day's work is after a rest of pleasure.

Q. Does that seem reasonable?

A. Well, I don't know what word got out that time.

Q. You are not foolish, are you?

A. (Quickly.) No, not a bit, as I know of.

Q. Now I will take one more sentence. In the spring-time sparrows chirp.

A. In the springtime the—schu—eh-h-h—float—I think—ah—no.

Q. Now, Mr. Masterson, I want you to tell me slowly and distinctly how this sickness of yours came on.

A. I don't know how it came on.

Q. Describe it.

A. Only remember coming up the stairs; went to the hydrant, and came over and laid on a chair—chucked on a chair—that's all I know.

Q. That is all you know of your sickness?

A. That is all I know—that time—well, I can't move my arm.

Q. Well, tell me why you got put on a chair.

A. I don't know.

Q. What happened?

A. I could not eat any more.

Q. Now answer me this one question. Tell me what has happened to you since the time when you were first taken sick.

A. Eh-a—I couldn't tell you that. I don't know what to ask—em—is going around—I can't tell him.

Q. Why can't you tell?

A. Well, I can't very well. I do not remember it so well. I remember the day I got sick, that is all.

Q. Well, tell me everything that happened that day.

A. Well, I worked pretty hard that morning—coming down stairs—sometimes jumped down. Then went home, of course and had that—that—e-e-a-e (drawling). Did not eat dinner at all. I fell out of my hand, and I went over to the hydrant.

Q. What is the next thing that you remember?

A. That is all now, I think.

Q. Can you remember anything else?

A. Well, I was on the sofa, and I was lying there, and I could speak to them. Ever since I have been just as good as ever. I ain't done any more.

Q. Is that all you can tell?

A. Yes; I don't know.

Q. What occurred on the 4th of March in this country?

A. The President and Vice-President went in. They took the grand opera house.

Q. What do you mean by that?

A. Why, the name of the washing-house.

Q. You mean they entered the White House?

A. Yes, sir.

Q. What else occurred in the White House?

A. A ball—was there—all big women—high-toned women.

Q. Yes; but who went out?

A. Cleveland.

Q. Where did he go?

A. Oh, I don't know where he went.

Q. Did you not hear?

A. No.

To test his memory and associative faculties a few questions such as the following were asked:

Q. Name me all the four-footed animals you can think of.

A. Why— (hesitates) do you want the lion? Buffalo —eh—the lion— (mutters) did I say buffalo already?

Q. Name all the two-legged animals that you can.

A. Bird, chicken, and what is it? (mutters) I think— of 'em (hesitates, mutters).

Q. Now name all the different birds that you can.

A. Chicken, eagle—the-e—stuck now.

Q. Have you been in the country?

A. Yes.

Q. Now, sitting here, if you should suddenly hear some music, hear a tune—the tune, "On the Bowery"—where, in all probability, would it come from?

A. Why, I don't know. Somebody had an accordeon, somebody had a fiddle.

Q. Does it not occur to you that it might be a hand-organ in the street, or it might be a piano upstairs?

A. Yes, sir, come to think of it.

Q. Can you whistle?

A. No, sir.

Q. Why not?

A. Can't whistle ever since.

Q. You know tunes, don't you?

A. Well, I have not much of a fancy for music.

Q. Tell me what tune this is as I whistle it. (Whistles "Home, Sweet Home.")

A. Home, sweet mother.

Q. (Whistles another tune.)

A. Don't know that tune.

Q. (Whistles "On the Bowery.")

A. Yes, I know it, but I can't say it. It can't come into my head.

Q. It is "On the Bowery," is it not?

A. Yes, sir.

Q. (Whistles "Yankee Doodle.") What is that?

A. I don't know what the deuce it is. I don't know that one.

The essential features of this case are the total loss of articulate speech for a number of weeks following the attack of apoplexy, then the gradual possession of a few words to signify assent and negation, and later the names of his wife and of one or two of the men with whom he had long worked and lived. After a number of months his vocabulary began to increase gradually, particularly in words that signified action or quality. In the use of these words there were well-marked paraphasia and amnesia verbalis of the articulatory memory of the word. In addition to this the patient had agraphia and dyslexia, although there was no sensory aphasia, word blindness or word deafness, optic aphasia, or hemianopsia.

The most interesting feature of his present state is the agraphia and the dyslexia. He has been tested a number of times with the block alphabet, and although he can

frame simple words it requires a very long time, and he stops after he has made a few simple words, some of which are misspelled, and he insists that he is not able to go on. The dyslexia is strikingly shown by the transcript of an attempt to read a newspaper paragraph telling how lead pipe is made, a subject in which he should be interested, as he handles that article in his occupation. I have already spoken of disturbances of reading and of writing in cortical motor aphasia and have explained how they depend upon the disablement of internal language which accompanies every case of cortical aphasia.

Subcortical Motor Aphasia.

The term subcortical motor aphasia is used to indicate a partial or complete inability to externalize speech which has been properly formed in the speech centres constituting the zone of language. The lesion is one that causes a break in those speech-carrying neurons extending from the portion of the Rolandic cortex to which is allocated the representation of the different parts of the peripheral speech mechanism to the cells constituting the peripheral neurons of the same mechanism. Some recent writers, such as Ziehen,[1] use the term fascicular anarthria to indicate the same condition. The earliest important contribution to the differentiation of subcortical motor aphasia was made by Pitres,[2] in 1877, when he essayed to study the symptomatology of lesions of the centrum ovale. At

[1] Ziehen : Article "Aphasie." Eulenburg's "Real Encyclopedie," supplementary volume.

[2] Pitres : "Recherches sur les lésions du centre ovale." Thèse de Paris, 1877.

that time Pitres believed that there was no difference clinically between aphasia caused by lesion of Broca's area and that caused by lesion of subjacent white fibres. This was a legitimate supposition, in full accord with the anatomical and physiological teachings of the times. It was at that time supposed that Broca's convolution was the unique area of language and that in functioning it transmitted its influence by means of bundles of white fibres, the inferior pediculo-frontal fascicle, to the bulbo-medullary centres which conditioned the execution of speech. Therefore it was logical to suppose that interruption of the fibres constituting this pathway would cause the clinical equivalent of destruction of the cortical area of which it was said to be the projection. The conclusion would have been unassailable if the premises were correct. At the present day no one believes Broca's area to be the unique organ of aphasia, and modern investigation has shown conclusively that, even if it were, it sends no fibres into the great motor projection system that passes through the internal capsule on the way to the motor cranial-nerve nuclei and the pyramidal tracts. The completest destruction of Broca's area entails no paralysis of the lips, the tongue, the palate, or the larynx. Anatomists have shown that the speech centres in the zone of language are association centres, intimately interconnected by commissural fibres and devoid of projection fibres. The lower end of the ascending frontal convolution immediately adjacent to the portion of the area of language in which are stored articulatory kinæsthetic images (Broca's area) is the executive area of the zone of language. When this executive area is destroyed, or when

all the projection fibres going out from it to the motor cells constituting the beginning of the peripheral neurons which go to the speech apparatus are destroyed, there may be inability to articulate, which is just as complete and total as if Broca's area was destroyed, but the zone of language is intact and therefore internal speech is unimpaired.

The symptoms of subcortical motor aphasia are practically the same as those attending destruction of Broca's area, with two striking and all-important exceptions. The first of these is that the patient retains the capacity to write, which it will be remembered is lost in cortical motor aphasia proportionately to the completeness of the aphasia, and, secondly, the patient responds to the Proust-Lichtheim test; that is, he can indicate by movement in some form, by squeezing the physician's hand, by making expiratory efforts, by winking, etc., the number of syllables that constitute a given word or the number of letters. In other words, he can prove to the satisfaction of his examiners that he retains the ability to call up spontaneously the sound of the word, the visual image of the word, and the articulatory memory of the word.

One of the most interesting and instructive examples of subcortical motor aphasia in general literature, and there are many such, is that of the illustrious Samuel Johnson. He, after suffering for a number of hours with a curious sensation in the head, found that he had lost the power of articulate speech, though he could write with customary facility. This filled him with a portentous dread, although it is probable that his concern was in nowise comparable to that of Boswell, who foresaw in

this catastrophe the perishment of all that life held dear to him; the transmission to paper of Johnson's Solonic utterances. Johnson, desiring to convince himself that his intelligence was not impaired, essayed to write Latin verse, which he did with customary ease. He also indited a letter in the same language to his physician. This, in addition to being complimentary to the intelligence of the latter and to the profession of his day, showed clearly that the memory of the word in all of its forms, auditory, visual, and articulate, was intact, and that there was no disturbance of internal language. Even the occurrences attending the final attack were typical of subcortical motor aphasia. After consciousness, which had been submerged during an apoplectic attack, was regained Johnson essayed to speak in the English language. Finding that he could not do so, he tried to talk Latin, but found this impossible. He then uttered a few words in Greek with greatest difficulty. Notwithstanding this difficulty in communicating his thoughts by articulate speech in any of the languages that had been familiar to him, he wrote a Latin hymn addressed to the Creator, the prayer of which was that so long as the Almighty should be pleased, graciously, to permit him to live, he should be allowed to have the enjoyment of his understanding, that his intellectual and bodily powers should expire together. This shows satisfactorily that, although he could not manifest thoughts in external speech, internal speech was intact and that it could be externalized by writing.

In discussing the clinical manifestations of subcortical motor aphasia, I shall record a few cases, beginning with the simplest, in which the phenomena are closely restricted

to inability to execute the movements of articulation. I shall then take up the more complex forms of subcortical motor aphasia. The following case, which occurred in the practice of Dr. J. C. Kendall, and to whom I am indebted for the notes, may be cited as one of the most restricted examples of subcortical motor aphasia that I have seen. The history of the case is much abridged.

A seventeen-year-old schoolboy felt quite well on the 19th of May. On the following day he noticed that his right arm was clumsy and awkward, and while in school he was not able to use the pencil dexterously. On the following day there was much impairment of strength of the right side of the body, and he dressed himself with a great deal of difficulty and got down stairs. On attempting to cross the floor, he fell. Examination of the patient soon afterward showed a right-side hemiplegia, the arm being most affected, the face less so, and the leg still possessed of sufficient motility to be flexed and extended. There was slight twitching of the right angle of the mouth. The tongue protruded naturally, and the pupils were of equal size and responsive. At this time it was distinctly noticed that there was no impairment of speech. On the following day the right leg became paralyzed, and his vocabulary was limited to the use of the words "Yes" and "No." He understood everything that was said to him, and was keenly appreciative of his surroundings and environment. During his illness he diverted himself with pictures, and often read, which he seemed able to do understandingly. Impressions coming through the visual apparatus and auditory apparatus were heard and interpreted. On the following day he could not articulate a word. He understood words readily; he could indicate the number of syllables in a word by pressing the physi-

cian's hand the same number of times as there were syllables in a spoken or written word. The hemiplegia remained the same. Five days later he was able to make slight movements with the toe of his right foot. The next day, that is, one week after speech disturbances first manifested themselves, he articulated the word "Paper," pointing to a newspaper that was lying on a distant table, and which he wished to get. The next day he said "Yes," in response to a question. From this time on he regained the ability to speak and at the end of a week he was able to answer and talk voluntarily in a stammering, hesitating manner. He never misplaced words, nor did he use words incorrectly. The hemiplegia disappeared rapidly, and a month from the time when he was stricken he could walk and run, and use his right upper extremity, even to perform such complex movements as writing, although this was very awkwardly done. Six months after the attack there were no trace of hemiplegia, except a slight exaggeration of the knee jerks on the right side, and no trace of speech disturbance.

The clinical history of this case, particularly its mode of development, course, and termination, indicates that the pathological lesion was of the nature of a thrombosis, while a study of the speech defects shows that the patient was in possession of the faculty of language in all its components, including articulatory kinæsthetic images, but that he was not able to give expression to his properly formed thoughts and words on account of some interruption of continuity between the area where such articulatory kinæsthetic images are stored and the peripheral executive apparatus of speech. In short, the case is one of subcortical motor aphasia, or pure motor aphasia of Dejerine. Although this is fully apparent from a study

of the speech defect alone, a consideration of the manner of his recovery makes the truth of this much more apparent. If the cortical area in which are stored the sensory articulatory images had been destroyed, the mode of recovery would have been quite different. In such case learning to talk would have been a slow and tedious process.

It may possibly be urged that the thrombotic lesion interfered with the vascular supply of Broca's convolution, and so temporarily—that is, during the period when the vessel was thrombosed—prevented this convolution from functioning; but the fact that the patient recognized the number of syllables in a word, thus showing the possession of articulatory images, is opposed to this view. The case, then, is one of strictly motor aphasia in the same sense as the paralysis of the right arm was motor, and it is explained in exactly the same way. The motor projection fibres that convey the speech impulses were temporarily interrupted just as the projection fibres that convey simple motor impulses were interrupted.

Writers are by no means in accord as to the location of the lesion that causes subcortical motor aphasia. Pitres, in a very recent contribution to the subject, contends that destructive lesions of the white substance adjacent to the foot of the third frontal convolution on the left side cause aphasia which cannot be differentiated from true cortical aphasia, and that the symptoms of subcortical aphasia are dependent upon lesions situated in the central part of the cerebral hemispheres and contiguous to the internal capsule. Very few writers are willing to concede this. It is undoubtedly true that it is oftentimes ex-

tremely difficult to differentiate between aphasia due to lesion of Broca's area and subcortical lesions subjacent to it; but it is my belief that in every instance it can be differentiated if the patient be studied with sufficient care and persistence, particularly with reference to his internal language, the ability to read, to write with the detached alphabet, etc., not to speak of the Proust-Lichtheim test and the information to be derived from objective study of the patient. It is not denied that lesion of the projection tracts at the level of the capsule sometimes produces a symptom complex very similar to that attending lesion of the white substance immediately subjacent to the lower end of the area of Broca and the ascending frontal convolution. Cases in proof of this have been recorded by Banti, by Dejerine, and other most trustworthy writers on this subject.

The case of Banti is a very typical one. A man, sixty-two years old, who had never learned to read, was stricken with apoplexy, followed by right-side hemiplegia and inability to speak. He understood everything that was said to him, and he endeavored to respond, but the only result of such efforts was a sound comparable to "tititititi." When words of one syllable or individual letters were spoken before him and he was requested to try to say them, he could articulate some of them so that they could be understood, if he did it very slowly and with great deliberation. He died five years after the apoplectic attack, there being in the mean time very little change in the state of his expressive faculties. On autopsy, there was found an apoplectic cicatrix, of a brownish-yellow color, at the level of the internal capsule, between the lenticular nucleus and

the thalamus—that is, in the anterior part of the posterior segment of the capsule.

Another case related by Dejerine is even more instructive, for the patient was a man of some education and the Proust-Lichtheim test was applied. The patient, sixty-seven years old, had right-side hemiplegia and aphasia of many years' standing. There was absolute impossibility to speak aloud, but he could whisper words that were intelligible. He was able also to indicate very well the number of syllables in words that he was entirely unable to pronounce. There was no agraphia or paragraphia. The autopsy, made eleven years after the first apoplectic stroke, showed three small foci of softening situated in the anterior of the hemisphere, one in the middle of the internal capsule, one in the caudate nucleus, the third in the fibres of the white substance subjacent to the foot of the third frontal convolution.

The recitation of one case or of a hundred cases of subcortical motor aphasia in which the autopsy showed the symptom to be dependent upon a lesion of the projection tracts at the level of the capsule in nowise militates against the statement that lesion at other levels higher than this, even up to the cortex of the Rolandic area, from which starts the projection tract, may produce the symptom complex of subcortical motor aphasia, and therefore I venture to think that Pitres takes an entirely unjustifiable and untenable position in saying that "it occurs only when the lesions are situated at the level of the internal capsule or at the origin of the capsular irradiation," unless the capsular irradiation begins at the motor cortex. It would seem to me that if the following case, recorded by Dejerine, were

the only one at our disposal it would disprove Pitres' contention. The patient had incomplete hemiplegia of the right side; no sign of verbal blindness or verbal deafness. The patient understood everything that was said to him. He was unable to pronounce a single word, to read in a loud voice, or to repeat aloud. The motor word images were meanwhile intact, because when he essayed to speak he made as many efforts of expiration (Dejerine's application of the Proust-Lichtheim suggestion) as there were syllables in the word. He wrote easily with the left hand. At the autopsy, an incision, passing horizontally to the foot of the third frontal convolution and not implicating the caudate nucleus, showed an oval spot of softening three centimetres long and two wide beneath the foot of the third frontal, reaching into the subjacent white substance as far as the lower extremity of the ascending frontal and parietal convolutions. I believe, moreover, that in many instances cases of subcortical motor aphasia due to lesion at the level of the capsule can be differentiated from those due to lesion closely subjacent to the cortex, and from a consideration of the objective symptoms alone.

Before taking up an analysis of these differentiating symptoms, I shall cite two cases of subcortical motor aphasia, the first an example of a subcortical lesion situated near to the cortex, the second due to a lesion at the level of the capsule. Opportunity was given to study the following case through the courtesy of my friend, Dr. Joseph Fraenkel:

M. W——, thirty-three years old, married; the father of healthy children; a native of Polish Russia; by occupation a tailor; and of good family and personal history.

Four years ago he was carrying a sewing-machine in the street, and fell, on account of weakness. He went home, complained of severe headache, was not able to be about, and the next day sent for a physician, who, after attendance upon him for a day or two, diagnosticated typhoid fever. Five days after he had taken to his bed, he became unconscious, and so remained for about sixty hours. When he regained consciousness, he was paralyzed on the right side and unable to speak. After six weeks his general condition began to improve, and in a short time he was able to be taken out of bed. Paralysis of the right side of the body persisted.

Examination shows a well-nourished, intelligent-looking man, of good disposition, who is apparently able to look after himself. He goes all over the ward and out of doors in a wheel chair, which he manages with great readiness. Aside from the spastic hemiplegia of the right side, which, however, does not involve the face, and the speech defects, his physical and mental conditions are very satisfactory. He has been examined repeatedly, and the results of these examinations are monotonously uniform.

What is your name? A. (Shakes his head and smiles sadly.)

How old are you? A. (Holds up three fingers twice, then puts his hand to his lips; then quickly reaches for the pencil with which I am writing; takes the pencil and writes "33.")

Are you thirty-three years old? A. (Nods affirmatively and nonchalantly.)

Are you married? (Affirmative nod of the head; then, when he notes that I am still waiting, he writes "Yes." Then, without being asked, he holds up in succession five fingers and four fingers, to indicate that he has been married nine years.)

Have you children? A. (Holds up one finger.) Is it a boy or a girl? A. (Smiles and makes a nod of negation.)

I want you to tell me if your child is a boy or a girl? "B' B' ba" (that is, he endeavors to say "boy," which is the correct answer, but he is unable to articulate it.)

How old is he? (Holds up five fingers and then three fingers to indicate that he is eight years old.)

What is your wife's name? A. (Reaches for pencil, and writes in distinct Hebrew characters the word "Rachel.")

It should be said here that the patient is fairly well educated in the use of his own language. He has learned to write with the left hand. He spends his time in writing letters to his family and friends, and in putting on paper his memories, thoughts, and hopes. The Hebrew language readily lends itself to construction by the left hand, its continuity being from right to left, and this patient writes with great distinctness and accuracy. It would seem on testing him a great many times that he writes spontaneously with more ease, accuracy, and correctness than from dictation. When, however, dictation is slow and he is not hurried, he reproduces most commendably. Many of his written productions he carries with him, and when they are read it is seen that not only the thoughts but the diction are very creditable. In response to simple questions or suggestions, such as, "How would you ask the nurse to get you a glass of water?" he fixes the nurse in his gaze, nods to him expressively, points first to a glass, then in the direction of the hydrant, and then to himself. Afterward he makes a movement of carrying the glass to his lips. When asked to put out his tongue, to close the eyes, to squeeze my hand the same number of times as

there are syllables in the word "Constantinople," he does so very intelligently and correctly. The nurses and orderlies in the hospital say that he never makes use of articulate words.

The sensory side of his speech mechanism is apparently intact. On being asked if he understands everything that he reads, he nods his head in the affirmative, and writes "Yes." On being asked to read a paragraph from a Hebrew newspaper, and then to write its significance or meaning he does so consistently, but uses nearly the same words that he has read. Numerous attempts were made with this patient to determine whether or not internal speech was intact. On every occasion he was able to indicate the number of syllables in a spoken word, the number of letters in a word, to write quickly the names of objects. In response to the question if he hears in his ears the sounds of the words that he reads, he writes "Yes." As to his hearing them with the same distinctness as when they are spoken, he answers "No," but that he hears them within himself. It may be said that he hears whispers with the same ease as loud-spoken words and sentences. He readily interprets familiar sounds, and when his eyes are bandaged and a watch, a bell, or the like impinges its sound upon his ear, he smiles and then quickly writes the name. In the same way he interprets the names and uses of familiar things that make impression upon his visual, tactual, and olfactory apparatus. When shown a handful of coins and requested to pick out all the five-cent pieces, or twenty-five-cent pieces, or cent pieces, he does so with accuracy. He likewise matches pennies, according to the heads or tails, dates, etc. When a twenty-dollar bill is taken in one hand and a number of bills amounting to eighteen dollars in the other, and the patient asked which he would rather

have, there is no hesitation in his decision. Quality goes before quantity with him every time. On being given a number of bills and coins, and asked to tell how much they aggregate, he writes down the correct answer. Simple columns of figures are summed up with accuracy but slowly, and other problems in mental arithmetic are performed in such a way as to indicate the workings of the associative faculties. He can hum the national anthem, can whistle in unison with another, and plays dominoes, cards, and penochle with a great deal of skill. On being asked if he would like to be able to speak again, he looks suspiciously at his interlocutor and smiles in a most discouraged sort of a way; but after a moment he reaches for the pencil and writes "Yes." On being told that it is very probable that he could learn to speak some words, he quickly joins in the physician's effort to repeat after him simple sounds, such as "o, a, n," and when he is shown how to place the tongue and when the lips are fixed in the proper position for him he makes intelligent efforts to produce the sounds into words, but persistent effort to teach him to speak has not been rewarded by material progress.

This is a case of subcortical motor aphasia. It is the purest and most uncomplicated that I have ever seen. It is not associated with agraphia or with amimia, and the sensory side of speech production seems to be absolutely intact. Whether or not it was formerly, before he came to the hospital, I am unable to say, for critical examinations of his speech defects had never been made. His case illustrates with great force the fact that in subcortical motor aphasia loss of articulate speech may be the only expression of inability to project outwardly states of mental content. He writes with accuracy, both sponta-

neously and from dictation. He expresses mental feeling by means of mimicry which is very easy to comprehend, but with the one exception of the word "Yes" and a sound resembling "No" he makes no use of articulation. His writing shows no evidence of repetition, of transliteration, or of agraphia. It shows, however, an uncommonly accurate portrayal of cognitions and feelings, for one of his class.

To summarize the clinical conditions in this patient, they are: Loss of volitional speech; the images of articulation are preserved and he responds to the Proust-Lichtheim test. On account of inability to speak, he cannot repeat from dictation. He can hum the air of a song, but not the words. He is unable to read aloud. On the other hand, there is no alexia or dyslexia, there is no agraphia; he can write voluntarily, from dictation, and from copy. He comprehends spoken speech, written speech, and gesture language. The word memory is not disturbed, the intrinsic speech mechanism is intact; none of the word memories, visual, auditory, articulatory, are in any way destroyed.

The left motor area or the projection fibres going out from it are destroyed, and this is indicated by the spastic hemiplegia which the patient has of the right side of the body. The fact that there are complete inability to articulate a sound and pronounced inability to co-ordinate linguo-buccal movements, as in whistling, without facial paralysis, leads me to the belief that the lesion is of the cortical Rolandic area to which are allocated the representation for the vocal cords, tongue, lips, etc., and not lower down in the projection tracts. For if the lesion

were of the latter it is probable that there would not be such complete aphemia as there is, but that there would be pronounced dysarthria or stammering. In this case, it would seem that the word impulse representing the idea left the zone of language properly and completely formed, but, on being handed over to the cortical executive mechanism, it could not be started down the emissive pathways, the projection tract, on account of destruction of that part of the cortex.

The next case, in which I believe from analysis of speech the lesion is lower down, is as follows:

Mrs. S——, a Polish Russian, thirty-eight years old. There is nothing of interest or of import in her family or previous personal history, with the exception of the event with which her present illness is associated. From members of her family it is learned that she was in perfect health until immediately after the birth of her last child, five years ago, when she was stricken with an attack of apoplexy, followed by right hemiplegia and inability to communicate thought by means of spoken language. Examination shows, as will be seen hereafter, that she is in possession of the most complete understanding of everything that is said to her, and that she has the faculty of making manifest such understanding and her wishes by means of the most expressive gestures. Her whole vocabulary is contained in two or three words, which she uses for every occasion.

What is your name? A. Frieda—, Frieda—, Frieda-Perl. (With a look of discouragement) Ich kann nicht.

How old are you? A. (In a sing-song tone.) Five, and five, and five, and five, and five, and five, and five (and then she holds up three fingers, at the same time smiling very knowingly).

How old did you say you were? A. (Starts to repeat it.)

Are you thirty-nine? A. No.

Are you thirty-eight? (Quick as a flash.) Yes (this being the number of years indicated by the use of the seven fives plus three, reckoned on the fingers).

Are you married? (In a sing-song way.) Oh, yes, yes, yes. Then she begins repeating, "Five, and five, and five, and five" in a sing-song way, and then holds up one finger, all of which indicates that she wishes to say that she has been married twenty-one years. On being asked if she has been married twenty-one years, she responds very quickly and accurately, "Yes."

How many children have you had? (She holds up one finger.) One? A. Yes.

What is your child's name? A. I can't say.

Is it a girl or a boy? (Points to herself to indicate that the child is one of her own sex.)

How old is she? A. Five, and five, and five; then she holds up three fingers, which, of course, means that her daughter is eighteen years old.

Is she married? A. Yes.

What is her husband's name? A. I can't say (ich kann nicht).

Has she children? In response to this question, the patient makes the most exquisitely intelligent and expressive gestures that I have ever seen; her face is wreathed in smiles, she places her left hand over her heart, and her right hand a short distance from the floor, as if to lift up the baby, and then she carries the imaginary baby to her breast, caresses and kisses it; then she puts it down and kisses her hand to it; and then she waves kisses in the direction of her previous home in the lower part of the city. After doing this, tears are seen

welling up in her eyes, but this condition is not the lachrymose uncontrollable attacks of depressed emotion, such as are sometimes seen in hemiplegia.

Interpreted, these gestures mean, in the most inimitable way, that she has recently become a grandmother; that the little child is just beginning to creep; that she would like to press it to her bosom; that it is the embodiment of her maternal affection; and her tears come because she is separated from it. If it is suggested to her that this is the proper interpretation of her gestures she gives the most appreciative and grateful assent. If some other interpretation is suggested to her, she laughs scornfully.

Tell me about your sickness. Oh, yi, yi, yi, yi, yi, yi, yi, vergessen (forgotten), kann nicht (I cannot) früher (before), weiss es nicht (I do not know), später (later), in, vergessen (forgotten), Frieda Perl (her first name, to which she attaches the word "Perl," for what reason I have not yet been able to fathom), finger. All this is said in a singing tone. After she repeats these words over for a time, she ceases to speak, as if discouraged with her own efforts. She then makes most suggestive gestures of combing her hair and then of toppling over, which means that her attack of hemiplegia came on without warning in the morning five years ago, while she was combing her hair, that she toppled over, and that she was unconscious for quite a long time.

On examination of the patient's physical condition, it is seen that she is a well-nourished woman, that she has the gait and station of one with partial right-side hemiplegia, and that the remains of the paralysis are more marked in the right upper extremity than in any other part of the body. The patient is able to perform all the general movements of the tongue and lips, and there is no difficulty of swallowing, no dribbling of saliva, and

she goes about and takes care of herself as does any patient whose mental integrity is undisturbed. As has been said, the language of her emotions is not at all impaired, and she smiles, frowns, laughs, and cries, whenever there is cause for any of these outward expressions, but she does not manifest them without cause. She understands all that is said to her, and detects with the greatest readiness unreasonable or ridiculous statements or speeches. The sensory side of her speech faculty is apparently intact. The patient has never been able to read or write, but she recognizes the letters of the Hebrew alphabet and words which form parts of familiar prayers, and she recognizes and puts the proper interpretation upon pictures. For instance, shown the picture of a girl, she smiles, and points to some young female patient in the ward, or goes through the gestures mentioned above to indicate that one of the kind in the shape of her little granddaughter is in her own family. When shown the picture of a cow she tries to pronounce the word, but when she cannot she reaches for a glass and by carrying it to her lips aims to indicate that the cow gives milk; and so on, with all the objects or subjects with which she is visually familiar.

Sounds and words apparently make natural and proper impression upon her auditory peripheral and central apparatus, and the tick of a watch, the sound of a bell, the paring of an apple, the note of a pianoforte, are all heard and properly interpreted. As I have said, she is wholly uneducated from a scholastic standpoint, and unable to write. Naturally, therefore, it is impossible to say whether or not there is agraphia or paragraphia. We may infer, however, that had she been able to write there would have been no defect in the graphic representation of thought except that due to the immobility of the right hand. It

is perhaps unnecessary to state, after what has been said, that the patient responds with alacrity to the ordinary commands, such as "Put out your tongue," "Close your eyes," "Bring me a piece of paper," and the like, and conducts herself in every way like a person in the fullest possession of her mental faculties.

Repeated examination of this patient, week after week, shows no material change in the responses to various tests.

The age of the patient, a consideration of her previous history, the physiological effort immediately antecedent to the attack, the mode of onset, duration, symptoms, and termination of the attack, and the degree of physical and mental recovery which followed it, all point unerringly to an embolus of one of the lenticulo-striate arteries as the cause of her apoplexy. It is not my purpose to pursue further a discussion of this question here, first, because it is not patent to the symptom complex of subcortical aphasia, which now interests us, and second, because the onset and course of the hemiplegia were typical of cerebral embolism.

A retrospect of her history shows that in the beginning there was complete aphemia, and that now, some five years later, the aphemia is still present, but is not absolutely complete. She can articulate a certain number of words; she can use properly the words "Yes" and "No;" and almost always the sentence, "I can't say." The few words of her vocabulary are used with conventional significance; but they are produced in a sing-song fashion, that is, the sounds are intonated and the words are repeated, and the rhythm of their emission is irregular.

Really, the most striking and interesting side of her

possessions in the way of projecting mental contents is her ability to use pantomime so expressively and intelligently. It is not necessary, in this connection, to say again that thought is expressed by movements. Movements are particularized in three ways, namely, by speech, by writing, and by pantomime, and inability to express thought by any one of these three ways is known respectively as aphemia, agraphia, and amimia. In some cases of aphasia, all of these defects are present, in others one may be complete, and the others only partial; while in subcortical motor aphasia, of which this case is an example, one of these faculties, oftentimes two, are entirely spared, while the third is completely destroyed. In this patient it would seem that the projection areas of the cortico-nuclear neurons which serve motor speech production were interrupted by the vascular lesion that occurred five years ago, but that the cortico-nuclear neurons which conduct impulses that are manifest by pantomime have remained intact. Sufficient has been said in speaking of the examination of the patient to show that the associative faculties which are the basis of intellectual activity have not been seriously impaired in this woman, nor has the internal language. Her mode of answering questions into which the element of time is interjected shows that she is capable of reckoning. I might say in this connection that if she were asked to compute simple problems in mental arithmetic she would indicate the correct answer by holding up successively the fingers of the hand, or by intonating, "Five and five and five," and so on, until she approached as nearly as possible to the correct figure by using the multiple; then she would complete the answer

by holding up one, two, three, or four fingers as the case required.

The distinguishing features in these two cases are that in the first there is no agraphia and the patient responds to the Proust-Lichtheim test, and in the second there is no disturbance of internal language. If it be a fact that a lesion of any of the speech centres entails some perversion of internal language in every instance, these cases do not belong to the category of cortical or true aphasia. They differ one from the other by the fact that the first patient is absolutely speechless, and the second can say a few words but only in a sing-song manner. When she says, "Fünf und fünf und fünf und fünf," etc., she does not speak it, she sings it. The other words that she uses are pronounced very imperfectly and indistinctly. It is impossible to indicate this on paper, for she speaks the Yiddish jargon and it has to be translated into English that it may mean anything on paper; but as a matter of truth the words that she uses are very imperfectly articulated. It is this imperfectness of articulation of words that she can say and the sing-song way of making replies that lead me to believe that the lesion in her case is at a lower level than in the case first enumerated. That is, I believe that in the second case the words may get started down the projection tracts on their way to be externalized, but they meet with an interruption in the shape of the lesion at some lower level. Those fibres that are not entirely severed allow the transmission of the impulses that are externalized by the patient's few words.

When the lesion causing subcortical motor aphasia is situated at a level as low as the internal capsule, then a

diagnosis can often be positively made, not alone from the employment of the Proust-Lichtheim test, but from study of the trouble of articulation. Such a patient has all the components of internal language absolutely intact, inclusive of the articulatory kinæsthetic images of words. He hears, sees, writes, mimics, and in other ways gives evidence of intellectual integrity. He is incapable only of causing the co-ordinate movements which subserve articulate speech. He may not be totally devoid of power of articulation; his incapacity may vary from simple slurring and elision of certain syllables and words, through dysarthria, dysrhythmia up to complete anarthria and arhythmia, and thus complete speechlessness. This partial loss of the capacity for articulate speech is well illustrated by the first case of Dejerine cited above.

In the conventional use of the term, this condition is aphasia; but it is not true aphasia, for true aphasia occurs only with lesion of the area of language. Yet it simulates true aphasia so closely that a differential diagnosis can be made only after very careful study. The nearer the lesions to the cortex, the more difficult will this differentiation be for such cases. All the projection fibres coming from the executive articulatory area are more likely to be involved, and with it there may be some functional perversion (possibly transitory) of the zone of language.

The differentiation of cortical and subcortical aphasia sometimes becomes of inestimable importance from a medico-legal point of view. For instance, a patient who has the symptom complex of motor aphasia due to a subcortical lesion may be just as competent to make a will

and dispose of his possessions as a man who has hemianæsthesia due to a central lesion; but a man who has cortical motor aphasia, and thus a derangement of his internal language, entailing some deviation from normal in every component of speech, be it in hearing, in seeing, or in expressing himself (including writing), may be quite incapacitated from such disposition, according to the interpretation of the law.

Cortical motor and subcortical motor aphasia are both almost invariably associated with right hemiplegia, and are dependent upon the same lesion.[1] In the cortical form the hemiplegia is apt to be less complete, and the spasticity of the paralyzed parts great. Moreover, it usually follows immediately after the stroke, although it may occur with epilepsy, tumors, abscess, foci of inflammation, or other conditions which slowly destroy Broca's area.

[1] In Appendix II. a case of cortical motor aphasia is cited in which there was no hemiplegia. It may be of interest, or instructive perhaps, to read it in connection with this chapter.

CHAPTER VI.

Sensory Aphasia.

1. General Considerations. 2. Sensory Aphasia: Word Deafness or Auditony Aphasia, and Word Blindness or Visual Aphasia. 3. Subcortical Sensory Aphasia.

As motor aphasia is used to designate those disturbances of speech expression in which the chief difficulty is in making speech, sensory aphasia, as a term, is applied to those cases in which imperfections of language, disability or inability to speak, are due to interference with the reception of speech forms; that is, to lesion of the perceptive areas of the brain and the immediate incoming and commissural pathways of such areas. The perceptive centres by whose functioning speech is ontogenetically developed are the auditory and the visual, and sensory aphasia is thus practically auditory and visual aphasia, and it is as such that I shall describe it, after a brief introduction to the subject.

Sensory aphasia may be defined as loss of the understanding of words, due to interference with the formation of associations necessary for complete perception. Anatomically speaking, cortical sensory aphasia might be defined as aphasia due to lesion of the posterior part of the area of language, and cortical motor aphasia as due to lesion of the anterior end of this zone. The subcortical

forms of each variety occur when there is lesion of the pathways which carry impressions into and away from the zone of language.

When Wernicke first described sensory aphasia it was as a symptom complex characterized chiefly by loss of comprehension of words, heard and read, often associated with paraphasia and agraphia. Clinically speaking, this is what is understood by the designation, and although sensory aphasia is subdivided into auditory and visual, it is only that the subject may be easier of comprehension and more in accordance with the symptoms that our cases present. The separation of sensory aphasia into auditory aphasia and visual aphasia is quite natural, in view of the fact that physiology and pathology are in the fullest accord in granting separate allocation to the two functions, seeing and hearing, whose integrity is so essential to the development and maintenance of speech. As the cortical representation of these two functions is independent, it follows that one may be diseased without the other. In such case the resulting symptoms are predominantly of the centre diseased, but nevertheless there are always some symptoms referable to the centre not the seat of lesion, for none of the speech centres are autonomous and lesion in one entails perversion of the function of all the others. In other words, in order that speech may be perfect, whether it be internal speech or external speech, it is necessary that the entire speech mechanism be intact. For the occurrence of a physiological reflex it is necessary that the afferent tract, the centre, the efferent tract, and the part in which the reflex act is manifest be intact; and if any one of them be disorganized the reflex act will be cor-

respondingly abnormal. Each element entering into the constitution of the reflex arc is an entity, if one chooses to call it so, and each one of its parts may continue to discharge certain functions after another part is diseased. but it can no longer contribute its share to the performance of a compound or a conjoint act after such disease. In the same way speech becomes disordered when one of the components entering into the speech mechanism is diseased. Nevertheless, when the auditory area is alone the seat of destructive process, there is no reason why the visual and articulatory kinæsthetic memories should not remain intact, and so they do; but when memorial recalls from these centres are intercalated into the speech circle, the auditory link is missing, and the circuit cannot be completed; the result is disturbance of speech, referable particularly to the hearing, because that is the element that is wanting. Moreover, as the primary revival of words takes place in the vast majority of peoples by the initiative of the auditory centre, when this is diseased internal speech is very defective.

The blood-vessel supplying the zone of language is the left middle cerebral artery, the so-called Sylvian artery. Lesion of different parts of its course entails different results. Disease of the anterior part and branches is apt to cause motor aphasia; of the posterior part and branches, sensory aphasia. In the beginning the sensory aphasia may appear to be equally of both the auditory and the visual centres, but, as time goes on and nature strives to overcome the lesion, the affection of one of these centres of language may be less serious than that of the other, and therefore the symptoms referable to it will be soon cleared up.

The symptoms referable to the other sensory centre may be caused by lesion of a branch of the vessel going directly to that centre, and therefore these symptoms are continuous and persistent. Thus it is readily seen that a degree of auditory aphasia and visual aphasia, or word deafness and word blindness, as they are unfortunately called, are frequently coexisting phenomena. The predominance of the one, or the apparent individual occurrence of the other, depends upon the seat and the intensity of the lesion. Many cases of sensory aphasia, especially those due to vascular lesion, have in the beginning symptoms pointing to implication of both the auditory and the visual centres; yet after the condition has existed for a time the symptoms referable to one of these centres may be reduced or even become latent, while those referable to the other centre persist and dominate the clinical picture. Thus auditory aphasia or word deafness and visual aphasia or word blindness are differentiated remains of one and the same sensory aphasia.

Sensory aphasia possesses certain very definite general features, which I propose to enumerate briefly before taking up the separate discussion of auditory aphasia and visual aphasia. These general features are in many ways materially different from the general features of motor aphasia, and they are explained by the location and relationships of the different centres. Motor aphasia is almost invariably associated with hemiplegia, while sensory aphasia is rarely accompanied by hemiplegia unless the lesion is a most extensive and severe one. In the case of motor aphasia this is easily explained by the proximity of the convolution of Broca to the motor centres in the

Rolandic region and to the motor projection tract. On the other hand, if the lesion be confined to the posterior part of the zone of language, the cortical motor area and the projection tract constituted by its central axones may be entirely spared. Compared with motor aphasia, sensory aphasia is relatively more often associated with lesions that are not primarily vascular in origin. In other words, it is more likely to occur with encephalitis, with tumors, with injury, and with certain degenerative diseases of the brain; and thus its evolution is often very different from that of motor aphasia. Whereas the former is almost always abrupt, and consequent on an apoplectic stroke which usually entails more or less prolonged loss of consciousness, sensory aphasia not infrequently unfolds itself slowly, and even when due to a vascular lesion it oftener comes on progressively or in repeated accessions than does motor aphasia.

Another very striking feature of sensory aphasia is that in the beginning it is the aphasia of comparative speechfulness, while motor aphasia in the beginning is usually absolute speechlessness. Sensory aphasia is characterized by logorrhœa, motor aphasia by alogia. Then the career of sensory aphasia is most instructive. The unfortunate patient starts in with his senseless loquacity, and week after week, sometimes day by day, one notes the shrinkage of his useless vocabulary, through the stages of babbling, of lalling, and of echoing, down to absolute mutism as complete as that produced by total destruction of the articulatory kinæsthetic area. The course of a sensory aphasic may be compared to that of a runaway engine. Some accident opens the throttle during the absence of

the driver, and away it flies without regard to destination, to danger, to results. Gradually, as its steam becomes exhausted, its pace becomes slower, it becomes less boisterous, it is more easily overtaken, until finally the last atom of steam is exhausted and it comes to a dead halt. There is nothing to stir it up and start it on. It stands ready, however, at any moment to be made a useful agency; it requires but coals and water. If it remain unused for a long time, rust and other accompaniments of time so incapacitate it that, even if these elements be given to it, it is no longer a useful agent, and it stands a useless wreck of its former self, a wreck induced by loss of the agencies necessary to drive it, all dating back to the accident that opened the throttle.

It is the same with the patient who has had a lesion of his auditory speech centre which is sufficient to derange it without completely destroying it. From the beginning of the person's ability to speak this centre has exercised the influence of a director-general over the articulatory speech centre, inhibitory and excitatory. Now this influence is taken away. The result is that all the inhibitory influence is destroyed by one blow, and the work of years of experience on the part of the articulatory centre in the endeavor to become a centre of the primary reviver of words begins to dissipate like a corundum wheel when breaking, and the result is logorrhœa. After this automatic storehouse (which is always small, even in the most expert linguist and most highly educated man) is exhausted, the vocabulary begins to shrink, and, as the centre which has always acted as the stimulator in the memorial recall of words is destroyed, the patient gradually approxi-

mates mutism, although the articulatory kinæsthetic centre is itself intact, ready to do its work on the reception of the proper incentive. But if this incentive has been long withheld, like the engine that becomes incapacitated by rust and other ravages of time, it will be incapable of doing it, even though such incentive should be given to it long after. This is but another bit of evidence in favor of the interactivity of the different speech centres and against the autonomy of any one of them.

Patients with sensory aphasia are very rarely reduced to a condition of mutism by such lesion alone, because destruction of the auditory centre is rarely complete. When the lesion of the auditory centre is slight, the most striking abnormality in voluntary speech is the inability to use words with their proper signification, although the words that are used are articulated with as much clearness and distinctness as in the normal state. The patient may utter words that are entirely the opposite of those which he intended to use. No more striking example of this can be given than a reference to the history of a lady cited by Trousseau,[1] who, arising to greet a visitor, would with courteous bow and apparent welcome say in the most matter-of-fact way, "Pig, brute, stupid fool," which when interpreted by one of her family meant, "Madame begs you to be seated." Happily the misuse of words does not often take this decidedly frappant form. It may be apparent only by a change of position of words in a sentence, or by the use of one word for another that has a somewhat similar sound or beginning. The condition is one that admits of ready explanation: the sensory centres being de-

[1] Trousseau: "Clinical Medicine," fourth edition, vol. ii., p. 674.

stroyed, their images can no longer be evoked to act as a check or control on the articulatory centre, the images in which are always evoked before the thought that is to be externalized can be expressed.

The defect of speech known as "jargonaphasia" occurs oftentimes with sensory aphasia. This may be considered a degree of paraphasia, although the latter is properly applied to a condition in which words are used in an incorrect sense. Jargonaphasia consists of the production of a jumble of words all forged into one, the syllables of which may be articulated, but the words have no similarity to words as usually spoken. Indeed, so wholly dissimilar are they that some cases of jargonaphasia have been considered evidence of a supernatural influence manifesting itself in the sudden acquisition of a language undecipherable by the most profoundly versed in strange tongues.

Reports of these cases not infrequently find their way into newspapers, and excite great attention and are much marvelled at by the laity. They are oftentimes investigated by committees made up of men imbued with praiseworthy scientific zeal, but oftener by persons who have exhausted their interest in things that conform to the established laws or courses of nature and who find inclination only in the supernatural. Jargon speech is occasionally an accompaniment of disordered cerebration dependent upon altered states of consciousness attending acute sthenic and asthenic states.

It is not alone in spontaneous speech that these perversions are manifest; they are as evident when the patient attempts to repeat what he hears (indeed oftentimes very much more so, particularly if the patient is partially word

deaf). In singing also the condition of paraphasia and even of jargonaphasia is sometimes very striking. The patient remembers and is able to hum the air of a familiar tune, but the words that he attempts to fit to it have no sense, no propriety, no reason. The occurrence of paraphasia and of jargonaphasia on reading aloud is also very striking, but as this subject will be considered in more detail under the subdivisions of sensory aphasia—auditory and visual aphasia—I shall merely make mention of it here.

Not secondary in importance to the information that may be obtained from a study of articulate speech is that which is to be had from an examination of the spontaneous, dictated, and copied writing. Patients with pronounced sensory aphasia are not usually hemiplegic, so that tests for defects of writing can be undertaken without trouble if the patient be made to comprehend what is wanted. Defects in writing are most striking when visual aphasia is the prominent feature of the sensory aphasia, although they occur in every case of sensory aphasia. The degree to which spontaneous writing may be preserved, or lost, in sensory aphasia varies with the patient, the seat, and the intensity of the lesion. Oftentimes there is preserved, even in cases of genuine visual aphasia, the ability to write a few words spontaneously, such as the patient's name, address, the name of some member of his family, and possibly a few other words; but even in the production of these the patient gets the surname following on the family name, and in other ways shows the condition known as paragraphia. If the patient has destruction of the angular gyrus, there will be practically total agraphia, because he cannot revive the visual memories that are necessary to be evoked in order

that the mobile part of the body carrying the pen may be directed by the thought to inscribe them. If the lesion is principally a destruction of the auditory area, there will be absolute inability to write from dictation; and even though writing spontaneously may be preserved to a very limited extent, the words or sentences produced will be markedly disordered in their arrangement.

Writing after copy is preserved in every case of genuine sensory aphasia, but the patient copies in a way that at once puts the stamp of his infirmity upon his work. He copies letters the way a beginner does a drawing, line for line, curve for curve, angle for angle, and makes an exact reproduction of what is before him in the same manner as does an engraver or a forger of a man's signature. These variations in writing will be discussed particularly under visual aphasia, in which variety of sensory aphasia they are most pronounced.

I shall cite the following case as a typical example of sensory aphasia, even though the symptoms were predominantly referable to that subdivision of sensory aphasia known as visual aphasia. It appears to me much more instructive to give accurate reports of interviews with such a patient than merely to state the results of such examinations. These reports demonstrate more conclusively than can any description, the shortcomings of speech as they really exist. This case has been of great interest to me as one offering difficulties of interpretation, and I have many stenographic reports of interviews with the patient, two or three of which I shall now present.

L. B——, female, thirty-nine years old. Said to be married. Admitted to my wards in the City Hospital,

July 8th, 1896. History: One year ago on awaking in the morning she could not move the right side. The lower extremity was not so helpless as the upper. Since that time she has not been able to talk understandingly. About eight or nine months later she had an epileptic attack, and since then she has an attack about once a month. Practically nothing is known of this patient's history. The facts just mentioned were obtained from the registrar of the hospital, to which she had been taken immediately after the beginning of her illness. These facts were given by the individual who brought her to the hospital. Since she has been in the City Hospital we have not been able to find out anything about her from herself, nor has any one been to visit her from whom such information could be obtained. In discussing the case I shall limit myself, therefore, to the results of examination.

A general survey of the patient shows that she is able to walk without the aid of a stick; she swings and drags the right leg and foot, not in a characteristic hemiplegic way, yet with a considerable paresis and spasticity. The right upper extremity is in a state of contracture and very nearly completely paralyzed. There is no involvement of the face or cranial nerves. To convey a proper idea of her speech disturbance, I shall give a stenographic report of a few examinations.

Examination, September 4th.

What is your name? m'm'm'y name, m'name's Sampson, my name's's—I don't know. I can't tell that.

You heard the doctor read the name? No, that I know.

Is your name Lucy Brown? Yes.

Is your name Lizzie Brown? No.

What is your name? Lucy.

Lucy what? I can't say.

Is it Lucy Black? No.

Is it Lucy Brown? Yes.

How old are you? I can't tell you, mu mother, my mother, my g'mother go.

Are you twenty? I wouldn't say.

Are you more than twenty or less than twenty? Oh, yes; twenty-one, twenty-two; I had children you know.

Are you forty? Oh, no.

You are seventy-five? Yes, ma'm.

Is your mother living? Yes.

When did you see her last? She comes on the first m'm'mth.

Was she here the first of this month? No, the last of the month.

Are you older than your mother? I can't tell that much.

Is your mother twenty? (Putting hand to throat) I can't tell that much.

Is she fifty? Of course I can't—boys—got——

Are you married? Yes, I'm married about o'o'o'—can't call it now.

How long have you been married? This boy—twenty-one—boy.

How long have you been married? I told you, I couldn't tell you — I couldn't tell; sometimes my speech——

Why can't you tell me? What? (in surprised tone).

Why can't you tell me? Married I tell you, please (pointing toward city) mother-care.

Here she began to talk of her paralyzed side—"My hand s'clame [lame] sometimes s'clame, sometimes strong."

I do not know yet whether you had one child or twenty? I have had two.

Do you understand all I say to you? Oh, me. Why yes, ma'm; I was sleeping, that's all.

Where are your children? Boy and a girl, boys, boys, girls.

How big are they? One boy is dead.

How big is the other? Twenty-one.

That is right, is it not? Yes, sir, right.

How long have you been in the hospital? Well, I think it is two months, I can't tell, Miss Buckley (the nurse) knows.

Have you been here a year? Oh, no, sir; no, sir.

Where did you live before you came here? Its, its, its, in though its, its—I can't tell it (in despair).

Where do you live? I can't tell you, but I can tell you tame.

Do you remember how the house looked? Oh, yes, sir; yes, sir.

Would you know it again if you saw it? Yes, sir; yes, sir.

Can you remember the house next door? I don't know, I think so.

Can you remember how your mother looked? Yes, ma'am; my m'm'm'oth other was lighter woman.

Can you remember the sound of her voice? Mercy, yes; oh, yes.

What was it like? White woman.

What was it like? What do you mean? My mother was a white, my mother was a white, light woman.

Can you remember the scund of a bell? Oh, I guess so.

Can you imagine that you hear a bell now? No.

What is a voice? Vcice, voice.

Yes; is my voice like the voice of that gentleman? (Smiles.) Voice, voice.

Have you ever heard that word before? No answer.

I then spell v-o-i-c-e and ask her to repeat it, but she cannot.

When she tries she says p-u-c-c-b.

You can say that pretty well? Yes—I can't once in a while, I have to suffer.

Do you know the letters of the alphabet? I can say them.

Say them! C, c, c, no, no, is that right? C, u, t, v, u, c, o, n, u, p, e, u, t, m, n, o, t, c, t, o.

Does the alphabet not begin, a, b, c, etc.? Yes, that's it.

Now let me hear you repeat the letters of the alphabet after me. A? u. B? c. C? u. D? w. E? w. F? i. G? c. H? h. I? i. J? j. K? k. L? o. M? i. N? u. O? o. P? u. Q? yaw. R? r. S? t. T? a. U? t. V? u. W? w. X? x. Y? a. Z? c.

You recognize that the alphabet starts off a, b, c. Yes, sir, I recognize but I can't say them, that's all.

It is noticed that she watches very closely the lips of her questioner.

Test for Recognition of Objects and Naming Objects.

Holding up a watch. Clock.

Chain? Clock, it's a clock.

Key? It's a clock (said very hesitatingly and starts to correct herself after the word is partly out, but at last lets it go).

Holding up another and more typical key? It's a key.

Pencil? 'S'key, it's what's its name, can't tell it now. It's a clock, can't tell it now.

Knife? Knife.

A roll of bills? It's a dollar.

A two-dollar bill? I can't tell, a dollar.

A twenty-dollar bill? A dollar. 'Tain't, can't do it now; I know it but I can't do it now.

Count on your fingers how many dollars this is (showing five-dollar bill). Doesn't understand and cannot be made to, and even after I show her how to do it and illustrate what I mean she cannot do it. Once she recognized and named a five-dollar note. Although she seemed to know the value of different denominations, she could not pick out the money when bills were called by face notation.

Match box? Match box.

Cigar? P-p-five, pipe.

Is it a pipe? (With emphasis) No, sir.

It is a cigarette? Oh, no.

It's a cigar? Yes, it's a cigar.

What am I doing now (lighting a match)? Match, turning it up, lighting.

How old do you think I am? I don't know.

Eighty-six? (Scornfully) Oh, no.

How many eyes have I? Two.

What is the color of my beard? No answer.

Black? No. Yellow? No. Green? W'w'ee, I can call it, but I can't say the name.

Is it red? Y-e-s (doubtfully).

What day of the week is it? Wesday (it is Sunday).

What day? W-e-s—, yes, sir, Sunday.

What do you do on Sunday? Sleep.

Are you tired? Oh, no, no, sir; oh, no.

Can you move your right hand? That one (pointing).

Apparently she cannot tell right from left with accuracy.

(Shown a picture of a man.) What's that? (Hesitates.) Is it a cow? Oh, no.

Is it a woman? It's a man.

(Shown a picture of a woman.) Sat's boy.

Why, that is a woman. Yes, I say it's a boy.

(Shown some letters, words, a book.) Points at them

all indiscriminately, yet still she seems to recognize some difference between them.

(Testing letters.) A? u. N? o. H? c. E? t. R? r. S? r. E? t. M? c. I? —. N? —. E? r. W? n.

What did you have for breakfast? (No answer, seems to be thinking.)

Stewed kidney (most improbable)? Oh, no.

At what hour do you dine, as a rule, Lucy? (Greeted with laughter.)

What do you like to eat? I'm not pertickler.

Test for hearing. A tuning-fork held to the ear seems to annoy her, as if there was great hyperæsthesia of hearing. She can detect a bell and the jingle of coins in one ear as well as in another, but she cannot say what they are. She hears the lowest whisper and apparently comprehends, and if one says she does not she assures him that she does. Calls a watch a clock when put to ear. She could not tell the time, but seemed to recognize the error if one said it was a different hour than indicated by watch.

(A cat walks across the floor). (Suddenly) Lucy, what is that? (Flabbergasted.) Is it a dog? Oh, no, no, sir. Is it a cat? Yes, sir. What kind of a cat? (Smiles.)

Lucy, do you take in washing here? No, sir, not here.

Do you drink beer? Once in a while. Whiskey? No, sir.

What is your husband's name? Lucy Brown.

There is no tone deafness and she can hum tunes in unison when some one whistles; can detect tunes but cannot say what they are.

A knife, a pencil, a tuning-fork, a spool of thread, a book, a pair of scissors, a small bell and a penholder were put in a row in front of her, and she was asked to pick them up as their names were called. She did this with

considerable accuracy, particularly after she had done it once or twice. In the beginning there was some hesitation, a searching look directed toward me, as if to ask, "Is it right?" and a clumsiness in picking them up, but she soon selected them without mistake or appreciable uncertainty.

Examination, September 14th.

What is your name? Well, tell him, m'u mame can't you (last addressed to nurse).

What is your name? I can't, can't, can't tell it now.

Why not? I can't, I can't, my name is oh, o-, o-, o-, o-, I can'——.

How old are you? Ma' mother's name, my mawth' name is ma'name.

How old are you? My mother got it down.

Can't you tell me? No, ma'am.

Have you a father and mother? My father is dead, my mother is dead.

What was your father's name? Reilly.

His first name? Can't tell that.

Your mother's name? M'a mother, Margaret Reilly (bit off).

Are you Irish? I don't know, but that is my name though.

What is your name? M'm'ma name is—I can't tell you. I can't call it. I know what it is though.

Is it Lucy Brown? Yes, sir (slowly).

Did you ever go to school? N'o, not much; I used to t'k washing.

Could you read? Yes, ma'm (with a glib, cock-sure manner).

Could you read? Little bit.

Could you read? Write, oh, no, sir; no, sir, no.

Are you married? No, I told you yes.

Were you married? Yes, sir; two children.

What was your husband's name? Sampson.

His first name? I can't call that name, but I can say it.

Is he living? Yes, ma'am, but I don't know where he is tho'.

How long were you married? Two children, I can't catch that.

What were your children? Two boys.

You told me the other day they were a boy and a girl. No, sir, I didn't.

I beg your pardon, you did. I did not (beginning to cry).

What was your first boy called? Sampson, after his mother.

The second boy? Little boy was dead, was dead anyhow.

What do you mean by that? I can't tell.

Was he born dead? Yes, sir.

How long is it since you had a child? I can't say, I can't call; fo', fo' years, I can't call it—but anyhow my boy, ah, u, oh, ah (sort of a revery).

Will you count for me? 1—2—3—4—5—6—7? 9—11--11——I can't.

What is that figure (large 4)? No answer.

Same for other figures.

(Showing coins) What is that? Money.

How much is that? Five cents (right). That? Twenty-five cents (right).

Please pick out a dime? Selects a penny.

Pick out a ten-cent piece? Does it correctly.

Pick out a five-cent piece? (Picks out a ten.)

(Showing two-dollar bill) How much? I can't tell, I can't call it.

Is it money? Yes, but I can't call it.

Is it a five-dollar bill? Yes, I know it now, it's a one-dollar bill; no, it's a five-dollar bill, yes.

(Holding up key) What's that? Key.

(Penknife.) (After some hesitation) Knife.

(Pencil.) Key—isn't it; no, was do you do with it?

Interlocutor recites first line of Longfellow's "Bridge," also Lord's Prayer, and asks her to repeat the first line, then half a line at a time. It is absolutely impossible, she makes the mouth go and occasionally makes an audible sound, but in no way repeats.

Say birds? Words.

Rat? Cat. Mother? Mother. Steamer? Simmer. Kettle? (In amazement, and does not repeat.)

Says wasin for basin, richer for pitcher, and in other ways simulates the sounds of words.

A number of familiar things are spread out before her, and she is requested to pick them up as their names are called. Pick up the key? Picks up scissors. Spoon? Correct. Glass? Correct. Hair pin? Correct. Looking-glass? Pincushion. Pencil? Correct. Key? Knife. Lock? Bell. Thimble? Correct. Looking-glass? Fumbles pin cushion, then picks up mirror. (This is in marked contrast to the successful efforts of the last examination.)

I then said to the house physician that she had mistaken the pincushion for the mirror before, and then she began to say something very inco-ordinate, like "I said, you, well, some, befo'," etc., all of which might be construed as an explanation of her failure to pick things up properly. There was marked confusion of names of things. After she had picked them up once, she seemed to be able to do it more accurately the second time, but after picking them up a number of times she got more confused.

She seemed to have the faculty of tracing simple figures such as squares, circles, and crosses, but she could not copy simple printed letters, although she could trace them fairly well. When asked to make a square or a circle after she had been shown a drawing of one, or to make such figures from memory she failed in the attempt.

To-day she seems to be able to match pennies with considerable accuracy. If I put down three pennies and ask her to put down an equal number, she does so. She also matches heads and tails very well. She cannot call the notation of money.

She does not appear to be so well to-day as heretofore. She is emotional and several times is about to cry, and when starting to cry she says something about children.

Examination, November 4th, 1896.

What is your name? Lucy.

Is that all of your name? Lucy Reilly.

Is it not Lucy Black? No, ma'am.

Is it not Lucy Brown? Lucy Brown.

What is your name? Lucy Reilly, I told you (indignantly).

You said it was Lucy Brown? Me (in tone of astonishment)? No, sir.

Well, is it not Lucy Brown? Yes, I told you it was.

How can you have two names, Lucy Brown and Lucy Reilly? m'm'w son.

What about your son? He's dead, he is.

Well, what about him? Nothing at all.

Then what did you mention him for? Oh, I don't know.

Did you ever go to school? Not much; no, sir.

Why not? Don't want; the doctor told me, washing all the time, my mother.

Can you read? No, sir. Why not? Cause I could.

How old are you? Ma answere, mo amte got my age.

How old do you think you are? I can't tell.

Are you a hundred? Oh, no.

Are you more than one hundred? Yes, sir.

Less than one hundred? I don't know. (This is rather characteristic.) She apparently appreciated the ridiculousness of asking her if she were upward of one hundred, still in the very next breath she said that she was. Then she denied it, and if her questioner looked astonished or incredulous she became embarrassed or indignant. One is almost sure that she does not understand the question, but she assures him that she does. Then the next moment she answers a question not only properly but very quickly; for instance:

How do you feel to-day? First rate, only I want to go home, that's all.

Where do you live? I, I'v, I told, I can't tell you, I can't tell you now, but I know the name of the street all the same.

Tell me how your sickness came on.

She hesitates, and after a time, during which she seems to be getting ready to talk, she begins: "One day—I had —the morning—I was on street—swashing—one day—I was—my mouth—come—she said I was so tizzy—I, I, I, —morning—I forget the rest."

All this is told in a long drawn-out way, as if she were telling a story that she had memorized.

Test for Recognition of Money.

(Handful of change, eighty-one cents.) How much? Fifty cents. (This after she picks the coins up in succession, looks at them, and feels them with great de-

liberation). Then I ask again, How much? She hesitates.

(Holding up twenty-five-cent piece) How much? Three, I told you.

One cent? Three cents.

Ten? Three cents, f', f', f', f' (as if endeavoring to say fifty or five).

Fifty? M'o, five, no.

Is it fifteen? No.

Is it ten? Yes (correct).

Examination, November 25th.

How many fingers are held up? One, two, three, four, five, six, seven, seven, five, seven, five, five fingers (latter correct).

(Holding up seven fingers) How many? Nine. (It seems that she cannot tell without reckoning on her own fingers, and then not correctly.)

I hold up five fingers, and she counts them slowly and correctly.

I hold up ten fingers, and she responds "nine."

Ten, wasn't it? Yes, sis.

She is now given a roll of money and asked to count it. She turns it over, separates the individual bills, then pays no attention to it until told to go on with it again. There are fourteen dollars in the roll. She counts the four one-dollar bills, but hesitates when she comes to the ten-dollar bill, and then says: Dollars, dollars, you know what I mean.

Show me with your fingers how many dollars that bill stands for? She makes no reply. She is either unable to respond or she does not comprehend the question, and emotionally there is no evidence that she does understand.

It is a twenty-dollar bill, isn't it? Yes, yes, sir.

It is a twenty-dollar bill? No, sir.

Well, you said it was a twenty-dollar bill? (Exclamation of indignation and surprise.) I did? No, sor; no, sir; (mumbling) yes, sir; no, sir.

Did you ever go to school? Once in a while. Couldn't; working all the time. Well, when I was a girl, playing all the time. Yes, sir, working.

A cat walks into the room, and I ask her to tell me what it is. She replies quickly and promptly, That is a book.

It is a little book? Oh, no, sir. Or, no. No.

Well, you said it was a book. (Indignation) I? Oh, no, sir.

Well, what is it? (Hesitatingly) a child. A little girl.

What color is it? I can't call it.

I kept urging her to name it, and finally she burst out, "It's a little cat."

Is it the same color as that (holding up brown paper)? A. No, sir. As you? Yes, sir.

She not infrequently uses "Yes, sir," for "No, sir," and *vice versa*, and sometimes there is a repetition of words—"intoxication" by a word.

What's that (pointing to bird in cage)? Bird.

What kind of bird? I know but can't tell.

It's a robin, isn't it? Yes, sir.

It's a canary bird? Yes, sir. I knew it was. A robin. What is it?

A robin? No, I tell you it's a canary. Yes, sir; I said it was a robin (indignant tone of voice).

(Pointing to a pair of shoes.) What are those? Ladies' shoes.

(Pointing to an artificial orange.) Ladies' shoes.

(Pointing to a mirror?) Ladies (hesitates).

(Pointing to a purse.) Can't call it.

(Pointing to a book.) Bo', bo', bo', I can't call it, I can't. (Stops abruptly.)

Lucy, tell me again about your sickness? One day wa' was washing, and all I tell you that, I don't know—I tell you, but—was, s s washing, com—par—my daughter, the girl—me, and I should—I can't, I can't, and then the girl, washing, oh, o-h, the boy—other, some stuff, and I look, that's all, got me—I said it, see? Well, tant, I can't tan—o-h, I can't tell that.

Tell me what you did yesterday. Warden, on—I—sway—all—looking—warden—names—fighting, fighting, fighting, I'll go home anyhow. Se doctor—names—(it is thought that the patient is endeavoring to say that some of the other patients in the ward taunt and tantalize her, call her nigger, etc., and when she is asked if this is so she responds promptly that it is.)

I ask her to repeat the following sentence, "I am delighted to make your acquaintance." Way, m', me, me, m, once, me (continued to get more monotonous and lower).

Lucy, what day of the week will to-morrow be? Fure, fure, not Friday, I can't call it.

Friday? No, sir.

Saturday? Yes, sir. (After a little bit, when her interlocutor makes no reply, she promptly says, "No, sir.) Thus she is absolutely unable to repeat. She is also completely unable to read words and letters, and I believe unable to read figures, although I have never been able to convince myself of the latter. Sometimes she apparently recognizes the figures on a banknote. Certain it is that she cannot read figures on a test card. She is unable to write a letter spontaneously or from dictation, and she is unable to copy, except the simplest lines. She is, however, able to trace.

It is now for the first time made quite certain that she has typical right-side homonymous hemianopsia. This was thought to be the case after the first examination, but the paraphasic answers on the part of the patient and the difficulty in making her comprehend what was desired of her when objects were brought into the visual field, made the test very uncertain. To-day objects thrust toward the right sides of both eyes, as if they were going directly into the eye, do not cause the slightest blinking. A candle brought toward on the right side is not perceived until it gets beyond the mid line.

When a summary is made of the results of repeated examinations of the speech defects of this patient, we find that the most striking defects of spontaneous speech are amnesia of words, particularly of nouns, and paraphasia. The patient can usually tell her name, but she cannot tell the names of members of her family or her residence. In fact, even to-day we do not know where she lived or the names of any of her friends. The articulatory images seem to be preserved. If she can call up what she wishes to say, there is no difficulty in saying it. It is impossible for her to repeat the simplest sentence after me. This is well illustrated by her effort to repeat the conventional phrase, "I am glad to make your acquaintance." All that she produces is an unintelligible mixture of sounds constituting gibberish. There is relative preservation of the melody of some songs with inability to use the proper words. She can sing the airs of hymns and popular tunes, but she cannot get the words in properly. There is inability to read aloud or to herself. She cannot write spontaneously or from dictation, and there is very great diffi-

culty in copying. In copying simple geometric figures, if the copy is removed for a moment she cannot go on with the delineation. There is word blindness, a loss of the comprehension of written and printed words. She is still capable of distinguishing one object from another, one color from another; and she can match pennies with some certainty. Figures on a banknote are recognized but not mentioned. During one test she seemed to have little or no notion of the difference between a letter and a word, and in looking at a printed page she could not point out single letters as different units from the individual groups of letters constituting single words. There is right lateral homonymous hemianopsia. There is a slight degree of word deafness, which becomes more apparent the longer the case is studied. This word deafness does not seem to me to be due so much to a destruction as to a functional degradation of the auditory word centre. This functional degradation is the most patent cause of her amnesia. The amnesia verbalis which she has, *i.e.*, of persons, places, and things, is the usual kind of amnesia associated with slight disorder of the auditory area. The symptoms which she presents are characteristic of sensory aphasia, and I believe it to be a case of sensory aphasia in which lesion of the visual centre and the subadjacent white substance is responsible for the leading speech disturbances. Her capacity to speak and her vocabulary vary from day to day, and vary considerably with her disposition and general tone. She has shown herself able to articulate with distinctness at times words presented to her consciousness by voluntary recall of past impressions, by auditory impressions, and by association; *i.e.*, those sug-

gested to her mind by an appropriate question. She frequently says, "I can't tell," and oftentimes in such situations she manifests the state that is best termed amnesia verbalis. This amnesia is most marked for proper names and nouns, and very slight for verbs, adjectives, and pronouns. It is not easy to decide whether this be due to conceptual, to kinæsthetic, or to auditory amnesia, and no tests so far have positively settled which one of these important elements is most disturbed, but it is very probable that the amnesia is predominantly auditory. On the other hand she at times indicates that the word is just on the tip of her tongue. It appears as though the word were mentally formed. She gives every indication of knowing what she wants to say and of having the feeling of making the proper innervations; but the wrong word comes out. In other words there is paraphasia. This wrong word frequently presents interesting relationships with the correct word; if she is asked to say B, she may say C instead. On one occasion when asked to say X, she called out I. Here there was a double relationship, first to the contiguous letter Y, and then to the similarly sounding letter I. Many contrast relationships manifest themselves, such as saying "yes" when she means "no;" "mam" instead of "sir." The wrong word was also frequently associated by similarity of sounds with the proper one. The influence of words just heard, or just thought of, also manifests itself occasionally, simulating an enforced echolalia. There are many instances, moreover, which suggest that the right word is just about to be spoken after a number of struggles, without any result; "wed" has been enunciated for "red," and through a series of approxi-

mations of this sort, the patient has frequently been able to arrive at the proper word or words.

She seems to have hyperæsthesia to tone vibrations, and they appear to produce a disagreeable tickling sensation in the ears. This is mentioned because it has been associated by Freund and others with verbal perceptual deafness, without general acoustic deafness. The patient, despite this intelligent hesitation in responding to questions, shows at times a faulty perception of the significance of the words of others. I say to her, "You said yesterday that you had three children," and she will say, "Yes, three." I say, "What were they, "two boys and a girl?" She will say, "No, two boys." I then say, "You have three children, both of whom are boys?" She will say, "Yes," even repeating the statement after me. If I then say, "You do not mean three children, you mean two," she will say, "Yes, two." In repeating words and letters her greatest difficulty seems to be on the articulatory side. Nevertheless, I feel certain that there is some perceptual word deafness, but it is the slightest of her deficiencies. She seems to call up with a great degree of certainty the use and significance of objects, and even their names. At times, however, she shows marked deficiencies amounting almost to complete perceptual blindness. I have seen her overlook an object that was held in the hand, which I named with distinctness and with which she had shown herself quite familiar. She tried to strike a match on the inside of a box, although she had just shown that she understood the significance of a match and understood what striking it meant, and it was not until I had told her that she should

strike it on the outside and move the box about a little that she seemed to perceive the side of the box, felt of it, and then ignited the match.

The mental power that seems most persistently deficient is the ability to grasp ideas of number. If I say, "Match that penny," she will put the penny down, correctly matching it. If I say, "Put down as many pennies as I do," she is utterly at sea. She seems to have no notion of number. This is manifest also in regard to relative ages. She seemed only to be sure that her mother was older than herself. A fairly simple task she performs with ease; but even a small complication throws her off the track. She matches one penny at a time with ease. Put down three pennies and ask her to match them with an equal number, even though the pennies are widely separated, she will put down all the coins in her hand, at the same time making no attempt to match them; or, if she does, failing signally to do so. The case seems to present some symptoms of restriction of consciousness and of detachment of consciousness, such as are met with in cases of hysteria. Then again she shows, but only very occasionally, a blank stupidity, absent-mindedness, or mental vacuity and consequent confusion.

Auditory Aphasia. Verbal Deafness. Word Deafness.

In discussing the localization of the various sensory and motor elements involved in the faculty of speech, we distinguish three distinct speech centres: the articulatory, the auditory, and the visual centres. Strictly speaking, the latter centres, in so far as their functions concern the faculty of speech, are not sensory areas but areas of per-

ception, or, better still, of apperception. The so-called sensory aphasias do not present disorders of sensation, nor yet of simple perception, but disorders of the interpretation or understanding of certain classes of perception. Destruction of these centres produces conditions in which memory significance of spoken or written speech is lost. Though the sound of the words and the images of the words may be clearly perceived, there is a loss of co-ordination which prevents these simple receptive centres from arousing other elements in adjacent centres, whose activity is necessary in order to awake the memory impressions stored there as the result of past experience with the spoken and written symbols of speech.

In this form of aphasia, first differentiated by Wernicke and given the name word deafness by Kussmaul, there is inability to understand spoken words. This is dependent apparently upon the total loss of auditory verbal memory images. It is one of the uncommonest forms of aphasia, and it rarely occurs individually, being frequently associated with some degree of visual aphasia or motor aphasia. Strictly speaking, the defect is not word deafness at all, for not only do such patients hear spoken words, but frequently they show no diminution in the intelligent appreciation of the significance of simple, co-ordinate, or purposive sounds.

Such patients, although word deaf, may be as alert as ever to the significance of a shrill blast of the whistle from an approaching engine. They detect sounds, even the slightest, as quickly as does the person whose auditory mechanism is intact, and they seek the origin and the significance of such sounds. In fact, because they are un-

able to interpret spoken words they are often apparently more keenly alive to sounds. It is true that an aphasic patient may also lose the significance of differentiated or purposive sounds but such an occurrence would needs be in a more pronounced case of aphasia than that which is typified by the term auditory aphasia. Such sounds are not usually complex and highly differentiated, therefore loss of the capacity to interpret their significance is rare.

There are as many kinds of auditory aphasia as there are varieties of interpretation put upon symbolic sounds by our consciousness. The seat of lesion which causes auditory aphasia has already been pointed out to be the cortex of the middle and posterior portions of the first temporal convolution, extending over into the second temporal and upward into the supramarginal convolution, where it impinges upon the cortical area for visual verbal images. Lichtheim[1] has reported a case of so-called subcortical auditory aphasia in which there was word deafness with no disorder of speech. The lesion was thought to be of the auditory projection system, in the white substance within the left cerebral hemisphere. Wernicke and his pupil Freund[2] report cases of extracortical peripheral lesion, located by them in the labyrinth, which manifested symptoms of word deafness, very similar to those reported by Lichtheim and others, as due to subcortical and transcortical aphasia. It would appear that pseudo-aphasia symptoms, which must be very carefully distinguished from those of true aphasia, may be due to actual lesion or to functional defect in the sense organ or in part of the subcortical receptive tract.

[1] *Loc. cit.* [2] *Loc. cit.*

The symptoms of auditory aphasia are subjective and objective. If the aphasia is limited to simple word deafness, the patient hears the voice in which words are spoken, but the words convey no idea to him and he has no more comprehension what they mean than if they were spoken in a tongue which he never before had heard. He, however, recognizes the significance of other sounds, unless it be that the memory pictures for such sounds are also lost. Naturally there are different degrees of word deafness, depending upon the extent of the lesion or the destruction of the auditory area. In some cases the extent is so great that the sound of the voice which speaks them is simply perceived as a sound, and such patients do not recognize the sound of their own name. In other instances they recognize the sounds of their own names and possibly the names of other members of their family, their places of residence, business, etc., words that have for them a much wider significance, and are more deeply imprinted in their memories than is the ordinary concrete word. In other cases the limitation of the lesion in the auditory area allows the patient to comprehend that he is being spoken to, and possibly to understand a word here and there. It is often very difficult when the patient gathers the significance of a single word to say just how complete the auditory deafness is, unless the physician be very careful in controlling suggestive gestures and facial expressions, for some patients quickly interpret in part the significance of what is spoken and guess the rest. If this form of aphasia exists for a long time, the patient may acquire considerable skill in lip reading.

In the milder forms of word deafness it is often neces-

sary, in order to estimate correctly the degree of word deafness, to test the patient carefully and repeatedly. For instance, if the patient is asked to protrude the tongue and he does so; to extend the right hand and he obeys, it might lead one far astray if he were to make a note, "The patient has not lost the comprehension of spoken words, for he obeys requests." Patients with word deafness are often keenly alive to their infirmity, in fact morbidly so, and they strive to conceal it by adopting any ruse that occurs to them. As protruding the tongue, extending the hand, etc., are customary accompaniments of a doctor's visit, they guess the meaning of questions which convey no ideas to them, and sometimes the responses are pertinent, either by accident or clever gesture reading. In those cases in which the word deafness is slight, the patient understands one or two words of a question, and pieces it out in his own mind accordingly. The simulation can be easily exposed by changing the sense of the question, while employing practically the same words or a number of the same words. It is then apparent that the patient does not recognize the different questions, for he answers as before.

The concomitant accompaniments of word deafness, inability to write from dictation, defective comprehension of what is read, imperfect writing, paragraphia, etc., may be disposed of very quickly. Inability to write from dictation, which a patient with auditory aphasia presents, needs no explanation. If the sound of the words do not revive the memorial significance of these words in the auditory centre, no impulses inciting the visual centre to visualize the word proceed from the auditory centre.

Internal reading is disordered, because the primarily excited visual word centre, in transmitting the impulses to the auditory area, finds the latter disordered, and there is in consequence defective revival of corresponding word memories and lack of comprehension of what is read. The paragraphia is an expression of the disorder of internal language, which is always present in true auditory aphasia.

A patient with word deafness, having a lesion that cuts him off from the significance of all that is said to him is practically rendered deaf. And as the catastrophe comes suddenly, after he has for years been accustomed to the delights of auditory sensations, he is naturally very much changed in manner, in appearance, and in demeanor. He is quiet and observant; his glance betrays suspicion or fear, and his demeanor is often one of trouble and unrest. This change in demeanor and manner, combined with the paraphasia which is often strikingly manifest, the inability to repeat from dictation, and the profound diminution, even to complete absence, of spontaneous speech, have often led physicians and laymen alike to look upon these unfortunate patients as insane. It need scarcely be said that such an implication is unjust.

As has already been said, word deafness rarely, if ever, exists alone. It is often associated with cortical motor aphasia, and frequently, on account of the proximity of the auditory area to the visual area, with some degree of word blindness. Wernicke and Lichtheim claim to have seen cases in which there were absolutely no defects of articulate speech. If the first of these two conditions exists, the patient is unable to communicate thoughts in

writing and is dyslexic, while if the last be present he is word blind as well.

Examination of patients with word deafness reveals different objective conditions dependent upon the degree and completeness of the word deafness. Even though the patient does not grasp the question that is addressed to him, he will endeavor to give answer, but the answer has no pertinency to the interrogation; in the first place because the patient does not understand the question, and in the second place because emissive speech is dependent upon the integrity of the auditory centre, that is, upon the revival of auditory memories and their transmission to the articulatory kinæsthetic centre. On account of the fact that the centre for articulatory memories is guided and correlated in action by the auditory centre, disease of the latter causes not only amnesia of words, but misuse of words, a condition to which the name paraphasia is given. I have said before that a patient in whom the primary revival of words has been through the auditory centre will be more incapacitated by a lesion that interferes with his auditory centre than will one who relies in part or largely on the revival of visual memories of words, or for the reinforcement of the memory of words by visual images. Such individuals are rare compared with those who revive words through auditory memories, but nevertheless they exist. An illiterate man who gets word deafness becomes almost speechless, because the illiterate man is then deprived of his only mode of recalling words to mind. According to Hughlings Jackson, Ballet, Stricker, and others, words may be revived as motor processes, *i.e.*, by stimulation of the centre of articulation (which they consider motor). It

is perhaps unnecessary to say here that the writer does not admit this, for it is one of the fundamental ideas in the conception of aphasia that he has put forth that Broca's area is not motor but sensory, as has been contended by Bastian for more than a quarter of a century. Moreover, in another place it has been pointed out that, developmentally, in the child the articulatory power is conditioned by audition. If the centre in which are stored the articulo-kinæsthetic memories of words—that is, Broca's centre—is not affected, articulation of words revived in thought by visual impressions may be but little impaired. This is especially noticeable in response to questions the significance of which the patient gets through the visual areas; that is, questions addressed to him by writing and by pantomime. But in such replies the words may be transposed, because the patient is not in possession of the faculty to know whether or not they have the proper sequence, for he can hear his own words no better than he can those of others, and, the auditory centre being destroyed, the articulatory centre is deprived of the directing control which the former exercises over the latter. It is very necessary that this statement should be as lucid as possible. If when one is speaking aloud a word is misplaced or a word is not used in its proper sense, if there be made what is called a *lapsus linguæ*, the auditory area, which is keenly alive to the slightest misuse of words, quickly detects the error and communicates it to the intelligence or carries it into consciousness. This in turn calls up the articulatory image of the proper term, which is then articulated. The sound of every articulated word acts as a stimulus to the auditory centre for the next. If the auditory centre has

been destroyed there is no such leader in the memorial order of words, and the frequent occurrence of *lapsus linguæ* constitutes paraphasia. The patient is usually not cognizant of the mistakes that he makes in speaking, though sometimes when he is he treats them lightly and essays not to notice them, or he is loath to express himself in words at all, providing he be in no way demented. Usually, however, the patient with partial auditory aphasia is loquacious, the logorrhœa being devoid of sense or pertinency. Occasionally one encounters a patient with word deafness who prefers to answer questions in writing, because the intactness of the visual memories of words enables him to control his output. In a similar way the auditory centre guides the action of the articulatory centre in the employment of internal language. In other words, I do not believe that the motor centre of speech ever becomes independent of the sensory centre which presided over its education, as is claimed by Bernard in his extension of Charcot's teaching.

If the visual area is coincidently diseased and the patient has right-side hemiplegia as well, such a patient will be, objectively, as one without a mind. The important receptive avenues of speech are closed to him and the emissive, likewise. He may be able to make known some of his thoughts or wants by means of pantomime, but just how much it is very difficult to say, except by the study of each individual case. As an instance of a case of this kind I may cite the following, which I saw with Dr. H. P. Hirsch:

A. M——, German, fifty-nine years old, married; by occupation a merchant. His wife has had four miscar-

riages and has borne one full-term child, which has since died. The patient, who had been a temperate, well-preserved man, of a kind and lovable disposition, was in excellent health until about three years before the onset of his present illness, when he had an attack of rheumatic sciatica. Since then he has had recurring attacks of "rheumatics" in the arms and legs. About two years after the attack of sciatica he had a mild attack of endocarditis. During the year preceding the present illness his entire disposition and temperament underwent a change. He became irascible, irritable, worried over trifles, and got fatigued more easily than formerly. He never complained of headache, nor were there symptoms that led to an examination of the urine. Attacks of the blues came oftener and stayed longer.

On the afternoon of the 21st of November the patient came home from business, apparently in his usual health. He sat in an easy chair and read a paper. Suddenly without warning his wife noticed his arms drop, his eyes roll up, his respirations become dyspnœic. She applied restoratives and in about five minutes he recovered consciousness, asked his wife what she was troubled about, did not seem to recognize that anything had befallen him, averred that he was all right, and did not want a physician. If he appreciated that he had had an attack of unconsciousness he did not say anything about it, and when dinner time came he went to the table as usual and partook of the meal. It was noticed by the members of the family that he did not use the right hand so much as usual, and he said that it felt somewhat stiff and heavy. Before retiring he took a hot bath, and on getting out of the tub he had another syncopal attack of very short duration. He passed a restless night, and in the morning on arising he found that his right hand was very stiff, un-

wieldy, and lame. He seemed to be in other respects quite well. There were no noticeable change of facial expression, no hesitation of speech, and no difficulty of getting about. On the afternoon of the 22d, Dr. Hirsch, a friend but not heretofore his physician, was sent for. He noted that the pulse was rapid and hard, the face flushed and anxious, and the patient evidently in a very anxious state. Examination showed a paralysis of the right arm which, although not complete, prevented the patient from using the arm except slightly. There was no involvement of the face or of the right leg. There were no sensory disturbances. The patient seemed to be in fullest possession of his faculties, talked distinctly, and explained rationally to the physician why he had been selected as the medical adviser; told of his symptoms, showed that he was able to read understandingly, and in other ways demonstrated that he used words at their proper worth.

The physician was sent for again about seven o'clock that evening. The patient had become unconscious while in the bathroom. He remained profoundly unconscious for upward of seventy-two hours, during which time his temperature was subnormal. He then recovered consciousness gradually. He was completely paralyzed on the right side, unable to speak, and apparently unable to understand anything that was said to him. For the next six weeks he was completely speechless; then he began to say the words "Yes" and "No" and some other monosyllabic words, but he did not use them correctly. He has not been able to understand spoken or written words since the attack. Occasionally, if commands are repeated a number of times, such as "Put out your tongue," etc., he will obey, but almost always it is necessary to show him pantomimically what is meant. After the second month his newly acquired, limited, monosyllabic vocabulary began to shrink,

and then he began to "babble" and to echo words that he heard. The babbling became almost continuous for a time and it was thought that the patient was insane. After a while it became less frequent and was produced only in response to or after a question.

One day, about six weeks after the attack, he suddenly began to sing a German song, which he had often sung, "O Tannenbaum, O Tannenbaum, bau, bao, ba, ba, ba, bo," ending in a laugh; and on a number of occasions he essayed to sing when the piano was played, but he never got beyond a few words.

Status, March 16th, 1897, nearly four months after the beginning of the present disease. There is hemiplegia of the right side of the body, the right side of the face being involved only to a very slight degree. The paralysis of the extremities is not pronouncedly spastic, but the tendon jerks are very much exaggerated. The heart impulse is weak, about ninety times a minute; there is a harsh systolic murmur, heard with greatest intensity over the aortic valve and the second sound is accentuated. The pulse is small and feeble.

On being asked his name he begins to whine and intone sounds which may be expressed by the following words: "Nein, nein, na, no, no, nein, bettau, betta, tau, tau, nein, nein," etc., beginning in a moderate tone and then getting higher pitched, and ending in a babble. All the time the musculature of the face is in such a state that on looking at the patient one would suppose that he is crying, but in reality there are no tears.

"How old are you?" No response, but after a few moments he begins the above senseless, articulated babble.

"Are you ninety years old?" Does not recognize that he is being spoken to.

In fact, test as long and as completely as we may, it

is absolutely impossible to convey any meaning to him by spoken word. He is alive to the slightest noise, and turns his head when a person comes in the room or on the occasion of a slight noise the origin of which he does not understand.

It is possible that there is right homonymous hemianopsia. It was difficult to get positive proof of this, as it is in all cases in which the patient cannot understand a word that is spoken to him and does not comprehend writing. The wife, however, suggested its presence by insisting that when she approached anything to his mouth from the right side, such as in giving him a spoonful of medicine, or to wash his eye, he did not see her until after she had got it immediately in front of him. On bringing the finger or fist quickly toward the right side of the eye, the patient does not blink or make any movement to indicate that he recognizes the approaching object which is seemingly going directly into his eye. When the same test is tried on the other side, the eye blinks at once, and he even indicates that he sees objects, such as the light of a candle brought into the visual field from the left, but in no way gives heed to it when brought into the visual field from the right.

There is complete alexia. A letter from his family, who are in Europe, is given to him, but although he takes it in the hand he does not recognize a word. It is the same with print. There is total agraphia; he cannot make a stroke with the pencil put in the left hand. When the hand is made to grasp it and the physician's hand guides it to shape a word, he takes no interest in the matter, apparently not having the slightest idea of what is being done, and begins to say, "Nein, nein, na, na, bateau," etc.

There is in addition to this some mind blindness, but this does not seem to be so pronounced now as it was a

few weeks before, when he many times endeavored to drink out of the urinal, and in other ways showed that he did not recognize the uses of objects. Nevertheless the apraxia is not complete, for when given his eyeglasses he places them astride his nose, in a particularly dexterous and intelligent way. This, however, may be an automatic act.

Voluntary speech is entirely lost, except the words that we have given, which he uses on all occasions. Repetition of speech is also lost, but sometimes he surprises the examiner and the family by echoing what is said, particularly if it be said a number of times and in a loud voice.

For instance, if one says "Good by" or "Good morning" a number of times, he may respond like a parrot, "Good by, ba, ba, ba, ba," and he will take up the old refrain, "Na, nein, nein, betteau," etc.

In this way he occasionally says, "Pfui" (first used by his wife, on one occasion when he was about to drink from the urinal); "Lieb," after the question, "Liebst du mich? Hast du mich lieb?" Without in the slightest manner understanding the question he may articulate with considerable clearness, "Lieb, lieb."

He apparently has some recognition of music, for when his daughter plays a familiar air on the piano he is attentive and seemingly follows it.

Examination of this patient eight months later reveals practically the same condition as above stated, save that the word deafness is, if changed at all, more complete. The hemianopsia is very difficult to demonstrate, and, if it exists, it is very slight. The only change of any import is a marked echolalia that he has developed. If one says, "How old are you?" he repeats over and over, "You, you," with a rising inflection on the last letter. "How is papa?" "Papa, papa," repeated and repeated. Usually he takes the last word of the sentence that he hears and

echoes it, occasionally the last two words. Such as "Will you have an orange?" "An orange, an orange," he repeats--the "an" with great vigor and clearness of enunciation and with a rising inflection on the last syllable of orange. Complex words he occasionally attempts to echo, but he does not succeed in so doing. There is still a degree of that condition known as mind-blindness, but it is not so conspicuous as when he was first seen.

No general description of auditory aphasia can be given to cover every case, so great is the variation in individual instances. In some cases there is only inability to interpret a certain language, or a certain dialect, or a certain number of words. For instance, there are a number of cases on record in which persons seemingly as familiar with several languages as with one, have had word-deafness for all of them except one, and that one the mother tongue, the one acquired first. Some years ago I had a patient who was employed as an official interpreter at the Immigration office. He developed a very slight right-side hemiplegia, which soon ameliorated so much that it was scarcely to be noticed unless examined for, but he had a marked degree of word deafness for all languages except Swedish—his mother tongue—a language which he had in later years rarely used, and never except when occasion or necessity compelled him. He was partially word deaf for Swedish. This language having been first acquired, it is logical to infer that it was most indelibly imprinted in his speech area and least easily destroyed.

I have said before that there must necessarily be as many forms of auditory aphasia as there are distinctive symbolic sounds. Spoken speech is the most highly sym-

bolic; the next most differentiated is music. To the form of aphasia in which there is deafness for musical notes, the designation tone deafness (musical deafness) is given. Amusia as an accompaniment or as an integral part of aphasia has been studied carefully by Edgren in recent years. In his article a number of cases are cited in which the auditory form of amusia has been subjected to critical examination, and some of them were studied in the light of post-mortem examination.

Musical deafness is almost always associated with word deafness, but there have been a few cases recorded in which it occurred apart from the latter. Such is an instance cited by Brazier,[1] in which a famous tenor of the Opera Comique was suddenly seized during a performance of an opera with complete amnesia of words and music; neither the orchestra nor his fellow-singers, who tried to prompt him, succeeded in reviving his memory. Another instance in which the tone deafness was not associated with word deafness was that of a man who suffered from attacks of ophthalmic megrim, during which there were passing attacks of motor aphasia, lasting from four to five hours. On one occasion there was no aphasia but he could not distinguish musical airs. The "Marseillaise," on being played by a military band, was not recognized; although he could hear quite well, he did not know the tune. He knew only that it was a noise of brass.

The clinical forms of amusia are strikingly analogous to the clinical forms of aphasia, and they generally accompany the latter, although the different varieties of amusia

[1] Brazier: "Amusie dans l'aphasie." Rev. Philos., October, 1892, p. 337, t. xxxiv.

have some clinical independence. The cases of word deafness and tone deafness reported by Serieux[1] and by Dejerine[2] may be taken as classical examples. In the case reported by the first mentioned there was total loss of the conception of spoken words. The patient remarked that she could hear the words very well, but that she did not understand them. The most familiar tunes when played on any instrument were not recognized. "Au Claire de la Lune" was said to be a "dead march." Cafe chantant music was designated church music, etc.

Lichtheim[3] has reported a very instructive example of amusia. His patient was a teacher and journalist, who became completely word deaf after a second attack of apoplexy. Communication with the patient could be made only in writing. He heard when one sang or whistled, but he did not recognize the melodies. Concert singing by his children was most annoying because it was "so noisy." The most familiar melodies, such as "Rufst du mein Vaterland," were not recognized.

This case is worthy of remark, in so much as the patient wrote facilely and correctly, and understood everything that he read. These features, I venture to think, stamp the case as one of subcortical sensory aphasia.

A still more striking example of subcortical sensory aphasia with notal amusia is that reported by Pick,[4] in which there was loss of musical recognition with preserva-

[1] Serieux: "Sur un cas de surdité verbale pure." Revue de Médecine, 1893, p. 733.

[2] Dejerine: "Cécité verbale." Mémoires de la Société de Biologie, February 27th, 1892.

[3] Lichtheim: "Ueber Aphasie." Deutsches Archiv f. klin. Med., vol. xxxvi., 1885, p. 238.

[4] Pick: Archiv f. Psychiatrie, 1892, p. 910.

tion of musical expression associated with word deafness. All other disturbances of speech and writing could be excluded. When the brain was examined it was found that it had its usual normal configuration, save that the convolutions were rather small. The patient had had a left-side hemiplegia, and changes were found in the right hemisphere to explain its existence. In the left hemisphere there was a subcortical softening of the posterior half of the first temporal convolution and of the medullary substance of the adjacent supramarginal convolution, which accounted for the preservation of internal speech associated with word deafness and loss of musical recognition.

Much evidence might be cited to show that there is a definite representation of musical memories, viz., auditory perception of notes, accords, and melodies, in the first and second temporal lobes of the left hemisphere. Edgren[1] believes, after careful weighing of the facts bearing on this allocation, that it is immediately in front of the area for word memories. The indications are that it is a part functionally and anatomically of the auditory centre.

Visual Aphasia—Verbal Blindness—Word Blindness.

This is a form of aphasia in which there is loss of the significance of written or printed words, although the words themselves can be seen with the usual distinctness. The designation "word blindness" or "verbal blindness" to indicate the inability to recognize words and letters, and interpret what they stand for, is a very unhappy one, because the term blindness has here a very different significance

[1] Edgren: "Amusie." Deutsche Zeitschrift f. Nervenheilkunde, vol. vi., 1895, p. 1.

than that given it in every-day use. In the form of aphasia which is described under the caption of verbal blindness the patient can see the word perfectly, but he gathers no meaning from it. The peripheral visual apparatus is intact. A printed page of a language previously entirely familiar to the patient suffering from this form of aphasia conveys no more meaning to him than does a page of Greek or Hebrew to the illiterate, or a page of Chinese symbols to him who reads only English, although he sees with the customary distinctness the letters printed or written, and he may even be able to tell the handwriting of one person from that of another. As in word deafness, in the literal interpretation of the term, the defect is not word blindness but loss of the significance of words. Words seen do not arouse a corresponding content of consciousness.

Word blindness may be classified according to the degree of its completeness and according to the kind of concrete written or printed symbols which we associate with ideas, such as algebraic symbols, musical notes, geometrical figures, hieroglyphics, etc., that the patient is unable to recognize. When the unmodified term word blindness is used, it is understood that other forms of printed and written symbols than letters and words are seen and interpreted, and that they call forth corresponding ideas. Many cases are on record in which a patient absolutely word blind was able to have roused in his consciousness certain ideas or thoughts leading to efforts of judgment by such printed or written, verbal or numeral notation. Dejerine has described a man with complete word blindness who was able to interpret the markings on goods in his

shop, and to tell customers the price. In other words, written letters having an entirely different significance than that ordinarily attached to them quickly called up a content of consciousness which he was able to associate with previously acquired knowledge.

I have already mentioned that blindness for words may be met with as an isolated condition. Broadbent in 1872 was the first to note such occurrence, but it was not until Kussmaul's discussion of it that it began to be carefully studied. The visual area is in the posterior lobe of the brain. It is made up of two more or less distinct centres: a visual perceptive centre and a centre in which are stored the visual memory of words and other symbols. The former is situated on the mesial surface of the occipital lobes in the enviromental area of the calcarine fissure; the latter (usually known as the visual centre) is in the posterior portion of the inferior parietal lobule, the angular gyrus, and the adjacent margin of the supramarginal convolution which curves over the posterior extremity of the fissure of Sylvius. Destruction of this centre produces a form of sensory aphasia in which there are inability to put interpretation on words seen and consequent inability to read—the condition known as word blindness, alexia, but it causes no loss of visual acuteness. It will be seen later on that the primary visual area and the higher visual centre are frequently diseased simultaneously, but the symptoms produced by each can be differentiated. In these cases it is to be understood that no lesion exists in the peripheral visual apparatus, although the condition known as homonymous hemianopsia, which will be referred to hereafter in some detail, oftentimes exists.

If cases of uncomplicated word blindness existed, the study and interpretation of their symptomatology would be a very simple matter; but such cases do not exist. There is almost always some association of motor aphasia, agraphia, and word deafness, and these coincident occurrences make the interpretation more difficult. In addition to the fact that word blindness is thus complicated with other conditions, it is to be remembered that word blindness due to lesion of the zone of language is sometimes associated with right homonymous hemianopsia or concentric limitation of the visual fields, on account of the juxtaposition of the optic radiations of Gratiolet to the angular gyrus on their way to the cortex around the calcarine fissure. Or, to express this anatomically, there is frequently some destruction of the optic projection fibres constituting the radiations of Gratiolet and those connecting the area in which visual memories are stored, the angular gyrus, with the primary visual centre in the left occipital lobe, which mirrors objects in the right visual fields of both eyes. Recent and trustworthy observations prove beyond cavil that destruction of the higher visual centre in the angular gyrus and supramarginal convolution does not cause hemianopsia, nor does it cause concentric contraction of the visual fields. In every case, therefore, in which hemianopsia and concentric limitation are present, the lesion must involve either the primary visual centre in the cunei or that band of white fibres which is the backward prolongation of the optic tract connecting the external geniculate body, the anterior quadrigeminal body, and the thalamus with the primary visual centre, and known as the radiations of Gratiolet. I am at a loss to

understand why Brissaud,[1] while admitting that there are cases of cerebral hemianopsia without verbal blindness, denies that cases of verbal blindness, and by that I mean literal verbal blindness, may occur without hemianopsia—facts laid down by Prevost and substantiated by the work of Dejerine and Serieux. These observers have put on record a number of cases which lead them to conclude that lesion of the visual centre, the angular gyrus, causes word blindness and agraphia, but does not cause hemianopsia if the lesion is limited to the gray matter, and that a lesion of the primary visual centre in the left occipital lobe alone causes right homonymous hemianopsia, but if limited to that the higher visual centre remains intact and the patient is not word blind so long as the latter is in connection with the primary visual centre in the other occipital lobe; but if a lesion should cut across the connection between both primary visual centres and the centre in the left angular gyrus, the patient is word blind but not agraphic.

Before proceeding to a discussion of visual aphasia I wish to record the following case of true sensory aphasia in which visual aphasia was the leading feature. I am under obligations to Dr. Joseph Fraenkel for opportunity to study the patient.

R. M——, a right-handed woman; native of Russia; forty-five years old. She is married and has borne several children. There is no history of miscarriages or of syphilis. She has now what seems to be an atypical form of Basedow's disease, the only attributable cause of which is emotional shock. She complains of fever and pain

[1] Brissaud: "Traité de Médecine," vol. vi.

around the heart. Objectively tachycardia, exophthalmos, and enlargement of the thyroid gland, more pronounced on one side, are very evident. A year or more ago she was quite melancholy; then she got very much better, and remained fairly well for a few months. The symptoms from which she now suffers came on very abruptly, about eight months before she came to the hospital, and followed an attack of pneumonia. Shortly before she came to the hospital she had a transitory attack of right-side hemiplegia; the right side of the face and the right arm were particularly involved, and the right leg very slightly. Admitted to the hospital, June 16th, 1896; died, October 24th, 1896. The notes of the house physician previous to my first examination are as follows:

"Futile attempts to get a history, patient being aphasic, this condition having come on, it is said, on the way down from the ward to the office. She appears to understand when spoken to, but her answers show paraphasia and motor aphasia. She cannot read; does not understand written words; laughs frequently; is very active; soils the bed; has dyspnœa.

"Examination: Weight, seventy-five and three-quarter pounds; pulse 148, arhythmical and irregular; respiration, 42; temperature, 99.5° F.; skin hot and dry. Patient fidgety; slight bed sores; thyroid gland enlarged; has Basedow's disease. When asked to do simple things, such as 'Put out the tongue,' 'Give me your hand,' etc., she seems to understand, and does it quickly and hastily. Favors the left hand; the right arm hangs down from the body.

"July 20th, 1896 (one day later).—Aphasia exists; considerable psychical bewilderment; her vocabulary, compared with that of yesterday, is diminished. There is well-marked motor paralysis of the right upper extremity, and

the forearm is kept in a semiflexed position. The knee-jerks are nearly equal on both sides, and they show the peculiarity of being increased with a flaccid condition of the lower extremities. There is distinct hemianæsthesia on the right side, but it is difficult to ascertain of what nature this is, on account of the jabbering character of the responses. Examination of the urine reveals the presence of albumin, the specific gravity being 1.028."

Since she has been in the hospital she has had one or two recurrences of the hemiplegia, but like the other attacks they were transitory. The most striking feature of the case in looking at her is her intense restlessness and the rapidity with which every movement is performed. She is either continually moving and agitated, or talking strings of words that are wholly unintelligible. She gives the impression of a person who is on the verge of bursting into an attack of acute mania, but who still has some control of herself. On my first examination, I found the patient to be an emaciated, excited-looking woman, who, on account of the bulging of the eyes, the never-ceasing physical activity, and the verboseness, presented a striking picture. At this time there was no trace of the previous hemiplegic attacks, the last of which occurred only a few weeks before the examination. The patient was sitting up in bed; she held continually a folded handkerchief or a towel against the face and mouth with the right hand, for what purpose I could not learn, but there was no drooling. Her speech possession is best indicated by the following stenographic report:

What is your name? Tanes—tanes—tanes. (Then she smiles, looks distressed, turns abruptly and reaches for the card which hangs over her bed and on which her name, age, period of admission, etc., are written. This she holds out to her interlocutor and laughs.)

How old are you? Sex—sex—vier—vier—fünf—fünf —(then, with the same rapidity as before, points to the card).

Are you married? Sure—sure.

Are you one hundred years old? Sure, sure. (It must be marked here that the lower class of Polish Jews give wider significance and usage to the word sure than does any other race, and it is necessary to bear this in mind in this patient, with whom the word "sure" seemed to be a recurring utterance.)

How many children have you? Four. Sure, sure, sure. (All this with the greatest facial and bodily activity and emotional display—smiling and laughing.)

What is the name of the first? Sexel.

What is the name of the second? Vier.

What is the name of the third? Fickel. (These are possible Hebrew words, but they have no appropriateness.)

What is your husband's name? Fickel.

What is your mother's name? Finckel.

Now, Mrs. M——, quiet yourself (it should be said here that the patient is very verbose, continually emitting a string of words which have no sense or meaning), and tell me slowly all about your sickness. My husband—my husband. He will say, explain. I can't. Sure, sure. Yesterday morning early—(then she repeats a string of words of which it is impossible to make a report, which here and there can be recognized as elements of Hebrew jargon, but the larger number of them are not a constituent of any language, nor have they any connection. Their production is accompanied by great motor activity, restlessness, gesticulation, and occasionally the word "sure.")

(Holding up a spoon) What is that? A book.

Is it a spoon? Yes. (Says it with pleasure.)

(Holding up a cup.) A cup. (It is probable that she heard the word cup.)

(Holding up a watch.) A thing to look for the hour.

(Holding up a match.) To rub. I understand all.

(Holding up a pencil.) To write.

(Pointing to some bread.) To eat. (After hearing the word "bread" uttered, she says it very quickly and with avidity.)

(Holding up a key.) Pickle (German, Schlüssel). (Then replies) What one opens with.

(Holding up a knife.) (Making a motion as if to open it) To eat.

Is it a knife? Sure (repeats jargon).

She is given a handful of coins and asked to select all the five-cent pieces, but she is entirely unable to do so. She starts in as if she fully understood what she was requested to do, but she is unable to do what is asked.

How much is there there? Twelve, six, four (and then she begins to laugh).

(Shown a two-dollar bill) How much is that? Six, six, six, six.

Will you please count for me? Six, six, seven, eight, one (then gets rather emotional).

On being asked to repeat the line, "Aus tiefem Schlaf bin ich erwacht," she was unable to do so. She started with "aus" and then poured forth a jargon interspersed with "sure," all the time smiling, grimacing, gesticulating.

Can you read? Yes. (She is given the text of a very familiar Hebrew prayer, and asked to read it, but she cannot repeat a word correctly. She is unable to decipher either words or letters.)

When an interpreter who is very familiar with her jargon asks her her name, she repeats, "Weitl, sceitl, heitl,

weitl." (It is thought that she is endeavoring to say "Rachel." All this time she is talking rapidly and gesticulating.)

How old are you? Six and four, and two, and sure, and sure.

Are you married? Sure, sure.

How long? Sixty-four, sixty-five. I can't remember.

Now try and count again for me? four, six, seven, six, seven, five, six, eight, seven, seven, eight, eight, sixteen, forty-six, six and four and five and six.

Can you repeat the letters of the alphabet? She begins to say the same figures as above.

Her people say that she was formerly able to read and write. She is now given the printed page of text that she should be familiar with if she had been able to read a word. She cons it very studiously and then begins, "Six and four and four and six," and so on in the most mixed-up fashion. She then takes an individual line and points the words out with the finger, and as she points to a word and makes some articulate sound she looks up at the physician inquiringly as if for corroboration. It is impossible to say from examination of the vision whether or not there is hemianopsia. It is oftentimes very difficult to say just how much she recognizes through the visual sense. I ask her to look at my watch and tell me what hour it is. She looks carefully and says it is nearly five (correct time, 4:30). Is it ten minutes after five? Yes.

And sometimes on being given a number of coins and requested to match them she does it very accurately. It is absolutely impossible for her to repeat a sentence or a few connected words.

On being handed a pen and asked to write she makes a show as if she were about to write (that is, she seems to recognize fully the use of the pen or pencil), but nothing

in the shape of writing results. There is complete agraphia. Of course it must be kept in mind that she has a very severe tremor; but this is not sufficient to prevent the formation of letters. She can copy, but in the most laborious and servile way.

The efforts at copying figures and numbers are somewhat more successful, but she cannot call the numerals that she is trying to write. A tuning-fork, a watch, etc., held to the ear she heard, but she apparently did not associate them with any distinct source or sound. It is impossible to test her for possession of associative faculties, for aside from the fact that she does not indulge in foolish actions, there are no means of judging.

In order to show the slight variation in her symptoms I append a protocol of an examination made some weeks later on the 24th of September:

What is your name? Heitem, weitel, sure. Can't say it. (All the time she is talking some sort of jargon of which neither I nor any of the attendants can make out a syllable, and she gesticulates at the same time.)

How old are you? (Apparently does not understand the question.) Question repeated, and she looks inquiringly at the nurse and at me, and then says, "Six and four and four and four," and then all at once as if she suddenly interpreted the question she points to her card.

Are you married? Sure.

How long? Six and four and four and six. I can't tell, I can't remember; sure. (Always after an apparent reply she goes on with considerable jargon, then smiles, laughs, looks around. She keeps the corner of her shawl, or a napkin, or whatever she may have in hand, up to the right side of the mouth.)

How long have you been married? Six and four, sixty-five, I can't say it.

She does not comprehend questions that are addressed to her in writing, and although she essays to read such questions the answers are not at all *à propos*. Interrogated in this fashion she looks at the writing eagerly, quickly, and knowingly, but when she starts to answer it is always the same, "Six and four, weitel," etc., etc. If one smiles incredulously, he is pretty apt to hear "Sure," etc.

Tests to make her repeat after her interlocutor resulted the same as determined previously. Sense of smell seems acute, but whether she detects the individual substance held to her nostril is not clear, for she cannot name it properly.

She seems to know the use of things.

Autopsy (Dr. Fraenkel): Asymmetry of the skull, shown by bulging of the right parietal boss. Dura and sinuses normal. Pia of the convexity normal. The Sylvian vessels as well as their branches are normal. Along the sulcus of the right insula there are three yellowish-white papules on the surface of the cortex, each about three millimetres in diameter. These lie either in the pia or upon the extreme surface of the brain, or in both places. In the left hemisphere there is seen at the posterior portion of the inferior parietal lobule a soft pultaceous yellowish mass, which is slightly depressed beneath the surface of the brain cortex. This measured four centimetres in diameter. Where this softened region joins the surrounding brain cortex it is less yellow and more firm. The pultaceousness is most pronounced at the centre. It occupies the angular and supramarginal gyri, not completely effacing both, and has slight impingement on the superior temporal in its posterior portion (Fig. 12). The depth of this softened region is so great that it extends through and involves the outer portion of the posterior arm of the

internal capsule just as these fibres enter the basal ganglia. The occipital lobe is slightly encroached upon. The right hemisphere shows a recent softening of the posterior portion of the third frontal convolution. This grayish mass is about two centimetres in diameter.

The areas of softening in the right side of the brain were of very recent date apparently, and they, like the

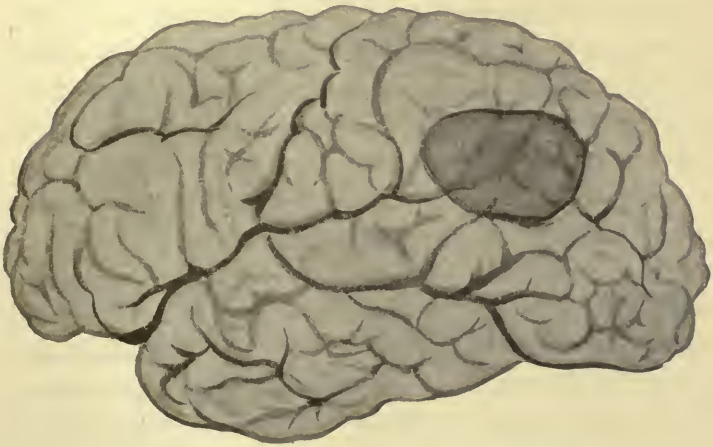

FIG. 12.—Shaded Area Indicates Spot of Softening.

one on the left side, can probably have their origin traced to emboli coming from the hypertrophic and thrombotic auricular appendix, and the chronic endocarditis. It is probable, moreover, that the lesion on the right side of the brain had nothing to do with determining either the aphasia or the paralysis, and the case is one of true sensory aphasia, the word blindness being the most prominent feature. The comparatively slight degree of word deafness that the patient presented was dependent upon the impingement of the area of softening in the inferior parietal lobule upon the posterior end of the superior temporal.

The total agraphia and alexia are of course significant of destruction of the angular gyrus, and the paraphasia and jargonaphasia are likewise most interpretable in the light of the autopsical findings.

Word blindness in its simplest form entails alexia, inability to read, or inability to get any information from written or printed symbols. Naturally there are various degrees of intensity of word blindness. The patient may be unable to read words, and yet retain the faculty of recognizing letters; or, on the other hand, this may also be lost, constituting literal as well as verbal blindness; or he may be able to recognize letters and unable to join them in syllables (asyllabia). For instance, Mierzejewski [1] has described a case as a form of cæcitas syllabaris et verbalis sed non literalis, and Badal,[2] in his monograph, mentions another case which was carefully studied by the author. The patient studied by Badal could read individual letters, but he could not combine them or keep them long enough in memory to form a word. This, it will be readily seen, is merely a difference in degree and not a difference in species. It has been noted in exceptional instances that a patient who has verbal but not literal blindness is able, if his auditory centre is intact, to have the meaning of the word made evident to him by spelling it out. For instance, though he cannot read the word "cat" he can read c-a-t and comprehend what the letters stand for. This conservation of the ability to read

[1] Mierzejewski: "Ein Fall von Wortblindheit." Neurologisches Centralblatt, p. 750, 1890.

[2] Badal: "Contribution à l'étude des cécités psychiques; alexie, agraphie, hemianopsie inférieure, trouble du sens de l'espace." Arch. d'Ophthal., p. 97, 1888.

individual letters when the sight of words calls up in mind no corresponding ideas is not difficult of explanation. It is dependent upon the mode of education of the individual. Most children, and indeed all until recent years, learned to read, that is, learned to attach certain significance to printed and written words, by first learning to recognize the individual letters of the alphabet and to differentiate them one from another. They are not supposed to associate any significance with such acquisition. Recognition of the letters of the alphabet is attained as the result of prolonged and tedious effort. Later, the child joins a number of letters together to form words, which he may or may not have previously heard, but which have a visual and auditory individuality and which give rise to a distinct content of consciousness, and the visual and auditory memory of them leaves its impress upon the cortex of the angular gyrus and the first temporal convolution of the left hemisphere in right-handed persons; of the right hemisphere in left-handed persons. The early acquisition of letters, and the primitiveness of their registration, explains the greater tenaciousness of their possession and the greater difficulty of their disintegration.

At the present day, one of advanced pedagogic enlightenment, children are no longer required to learn letters before syllables and words. It is probable that in a person thus educated there would be letter blindness coincident with word blindness. A condition somewhat analogous to that of variation in intensity of word blindness is that in which a patient becomes unable to read the letters and words of one or more languages that he had previously been able to read and speak perfectly,

while still retaining the capacity to read other languages. Cases of this kind are by no means common, but a number of them have been reported, particularly by Charcot and Pitres. It is not often that such a condition is the only disability, object blindness and some degree of mind blindness are generally associated with it. In the case cited by Charcot, for example, there was loss of visual memory for form and for color, for objects and for places. The monuments, houses, landmarks, streets, etc., of the town in which the patient had lived for many years, and with which he had been very familiar, all seemed new to him. Moreover, he did not recognize the members of his own family; they seemed to him like strangers. Some writers make the very grave error of confounding these cases of object blindness and mind blindness with cases of word blindness.

In many cases of visual aphasia, or word blindness, the patient, although absolutely unable to recognize anything else, still tells his own name when he sees it written. But unless he has been accustomed to see it in print, he will not recognize it. The only explanation for this fact that can be offered is that the individual's name, because of his long experience in seeing it, in writing it, in hearing it, is more deeply printed on the specialized sensory area of the brain to which it has been carried by the hearing and seeing apparatuses. In such cases it is understood that the angular gyrus is not completely destroyed. Oftentimes the patient will preserve a recognition of a number of other words, particularly of names with which he has been for a long time familiar, such as those of the members of his family, his business, his place of resi-

dence, the church of which he is a member, etc., and for precisely the same reason that he recognizes his name.

Occasionally cases are met with in which the verbal blindness is so very slight that it requires careful and persistent examination to reveal it. This is particularly true of cases in which the symptom of word blindness unfolds itself slowly, and of cases in which there has been a considerable degree of recovery. In such cases the patients may be able to recognize one or two words of a sentence, especially the substantives, and from them they gather the sense of the phrase. If the patient is not an educated person, the examiner may be misled at first by the readiness with which the patient essays to read, but there is that about his actions and demeanor that will suggest to the experienced physician that the patient is guessing. He very rarely reads right along unhesitatingly, as one usually does. He watches the expression of the person for whom he is reading, and at the end of every sentence or line he looks up and asks, "Is that right?" heaves a sigh of relief if it is, and turns to again.

It is a well-known fact that the acquisition of, and the memory for, figures and numerals are a different process than that for letters, and it is also known that the memory for the latter may be preserved and the former lost. Indeed, it is not so very rare for a patient to be word and letter blind but not figure blind. A very instructive case is one recently reported by Hinshelwood, in which a man although absolutely letter blind could read figures quickly and with the greatest readiness.

The term dyslexia is one that was first used by Berlin,[1] of Stuttgart, in 1886, to indicate a form of word blindness which differed materially from the ordinary forms. The first patient described by Berlin was a man sixty-six years old, who had been forced to abandon his occupation because the reading of printed and written characters had become quite impossible. He was not word blind in the usual sense of the term. He could pick up a paper and read five or six words quite correctly and get full appreciation of their meaning, and then he would have to stop, as the words had no longer any meaning for him. After he had rested for a short time he could go on and read a few words more. There was precisely the same difficulty with letters of every size. On being asked why he could not read he gave no satisfactory explanation. The letters did not become dim or confused, he simply could not read them. Efforts to read were most obnoxious to him, so keenly alive was he to his infirmity. Later, the patient developed other cerebral symptoms and eventually died from apoplexy. Berlin[2] looks upon the symptom as a special form of word blindness due to interruption in the conductivity of the connecting fibres of the visual centre, the angular gyrus. It does not seem to me necessary to look upon the symptom as an exclusive "aphasia of conduction," and I see no reason for not believing that it may be due to a partial impairment of the molecular function of the centre for visual memory, a condition that may result from injurious agencies that pervert the function of

[1] Berlin: "Weitere Beobachtungen über Dyslexie mit Sectionsbefund." Berliner klinische Wochenschrift, p. 522, 1886.

[2] Berlin: Archiv f. Psychiatrie, pp. 289–292, 1887.

this area without causing anatomical destruction of it. In such a condition the centre is capable of being aroused for the memorial recall of letters and words for a short time, then it becomes exhausted. After it has time to rest, a brief period of excitability follows. I am inclined to this view of it particularly from the fact that the symptom is not accompanied by symptoms pointing to impaired functioning of the auditory centre, which would be the case if the disturbance was in the internuncial fibres connecting these two centres. This view would seem to receive further corroboration from the fact that the symptom has been observed a number of times in alcoholic patients, and it has disappeared when the use of the toxic agency was abandoned. This view is in harmony with the genesis of alcoholic visual hallucinations which are so often of an extraordinarily vivid character, and with the occurrence of alcoholic retrograde amnesia.

Hinshelwood[1] has published a very instructive example of dyslexia due to alcohol. His patient was a tailor, forty-nine years old, who before his present trouble was a very capable workman. Latterly, on starting a piece of work, he forgot how to proceed, and every step had to be pointed out to him as though he were a beginner. Even then he would make the absurdest mistakes, and after he had sewed parts together they had to be ripped. The greater part of his time in the shop was spent in looking for things that he would put out of his hand, such as the thimble, needle, glasses, etc., so that finally he had to be dismissed. A rather remarkable feature of the case was that he frequently lost his way in the most familiar

[1] Hinshelwood: Lancet, December 21st, 1895.

parts of the city, and he could find his way home only with the greatest difficulty, even over a route that he had travelled for years. The difficulty of reading was very similar to that described by Berlin. The patient could read a few words and then he would completely lose the capacity to proceed. The letters, though seen with distinctness on first endeavors to read, would lose all meaning when he continued in his attempts to read. There was no blurring or running together of the letters. There was no disturbance of speech or deterioration of mental power, while memory for past events was not at all impaired. The patient recovered under tonics.

This case is one that admits of interpretation along the lines already suggested. The effects of the alcohol would seem to have been first and particularly upon the centre for the memorial recall of printed and written words, which were followed later by affection of those parts in which are stored the visual memory of objects, and finally, and to a comparably slight degree, upon the primary visual area which reflects the images of things. Hinshelwood contends that memories for form, color, etc., are stored up in both occipital convolutions, and the symptoms of his case would lend credence to this view. It would seem warrantable to suppose, in this case at least, that the seat of the disturbance was the entire visual area, the primary centres, and the centre for the visual memories of words. The integrity of speech and the fact that there was no loss of memory for past events show that the auditory word memories could be revived with their customary vividness and transmitted to the articulatory kinæsthetic centre. It is to be regretted that careful tests were not made to de-

termine the patient's capacity to write, for the auditory centre, acting with its customary force, should send impulses, which have a marked influence in arousing the memory of graphic images, to the visual centre.

If the angular gyrus is completely destroyed, the faculty of writing is lost with it, or, perhaps I should say, if the power of visual memorial recall is lost, then writing is no longer possible. In those cases in which voluntary writing is preserved, the lesion involves the primary visual centre, and, as this lesion is so often associated with right homonymous hemianopsia, the patient begins to write at the extreme left side of the sheet and stops in the middle of the page, unless he takes the trouble to inform himself by mediation of the tactual sense that there is still room on the line and pushes the sheet toward the left in order that it may be brought into the visual field. These patients, being unable to read what they have written, are totally unconscious of any errors of spelling or phraseology that they may make, although they may put the words on paper in as orderly a fashion as they were able to do before the development of the aphasia.

If the auditory centre is uninjured, the patient is able to comprehend what is read to him, and if his own handwriting is read he may be able to detect errors of sequence, of diction, and of spelling, but he is unable to take a pen and correct them, because he does not recognize and appreciate the value of a word or a letter. Patients who have word blindness will occasionally take a paper, a book or written matter and essay to read aloud, but naturally what they produce has no connection with the subject matter before them. Why they do this is difficult

of explanation. In many instances it has been the reason for considering the person to be demented.

Patients with word blindness are sometimes able to read written or printed words and sentences by tracing the word (which, it is to be remembered, they see with customary acuteness) with the end of the index finger or with a pencil. This is a most instructive phenomenon, and, although it is not often elicitable, it is by no means very rare and it should be studied carefully in every case. Such patients by utilizing kinæsthetic stimuli excite previous kinæsthetic memories, which in turn react upon or act conjointly with auditory and articulatory memories to revive the mental concept of the word, the idea that it represents. In other words, the kinæsthetic apperceptive area acts vicariously for the visual, and if it acts more slowly and with less certainty it is because it is neither ontogenetically nor phylogenetically intended for that purpose, while the visual area is. This should not be construed, I think, as a revivification by "motor processes," as taught by Stricker and others. The idea of the word is revived by kinæsthetic stimuli which consist of sensory impressions coming from the muscles, joints, and skin of the hand and upper extremity. Patients who practice perseveringly this method of conveying to their minds the meaning of written symbols, often acquire great facility with this mode of interpretation. The principle involved does not differ materially from that by which the congenitally or accidentally blind acquire ideas of form, of space, of size, of quality, and even of color, in which association becomes established between the auditory centre and various parts of the somæsthetic area.

The fact that such patients interpret by tracing written characters more readily than by tracing printed, indicates that muscular sense and the centre of graphic motor co-ordination are the elements that mediate the connection of the word and the concept, for in every person the training of the hand has been to trace written and not printed symbols. These patients read the movements of which the letters are the tracing, the usual pathway between the word and the concept in the revivification of visual images being destroyed. In some cases there is complete inability to read printed or written characters, while the recognition of musical notes, of figures, and of various other expressive symbols is preserved. In other words, there is a differentiation of graphic symbols, just as we have seen a differentiation of auditory symbols, and the more highly differentiated these symbols are the more completely and easily are they lost. The denomination of money and its representative value may be quickly and thoroughly grasped; the patient may retain any skill which he may have possessed in playing cards and in giving perception to symbolic formula other than those represented by letters, all of which tends to show that the storage of written symbols is not the same as it is for other forms of notation.

It should not, however, be understood that the patient may not be blind to all forms of notation, graphic and symbolic representation. Thus, there may be sensory amusia, sensory asymbolia, sensory amimia, etc. The occurrence of these will vary with the composition and character of the patient's intellectual possessions. A musician who has been accustomed to carrying simple or most in-

tricate themes by means of musical notation may gaze on scales without their having any other signification for him than they have for a person who has never seen a page of music. He may also watch the movements of a leader's baton while they give no more orientation for the execution of a piece of music which he may be able to play by revivification of auditory memories than would a person who had never heard of *tempo*.

Musicians, by virtue of the acquisition of a unique method of eye externalization, are in possession of specialized sensory images, visual and auditory, entirely divorced from the memory of letters, of words, and of figures, which probably posit for their possession different cells or association tracts, and the one may fail to be revivified when the eye falls on it, as it appears on paper, while others are called up by gazing on their like.

It is my desire to make as clear as possible that the patient with visual aphasia, word-blindness, sees with perfect distinctness the letters in his visual field. This is done by virtue of the primary visual area in the occipital lobe, but the reflection of letters by the primary visual area does not convey anything to the intelligence, it does not call up any memory pictures, for the area in which they have been stored is destroyed. The primary visual area reflects letters and words as a mirror does an object held up before it, except that there is no reversal, and as reflections by a mirror make no impression which remains after the object reflected is taken away, neither do words make any impression because of the impingement of their images on the primary visual area. Destruction of the primary visual area produces blindness in the literal sense

of the word; there is loss of perception of luminous objects, virtually the same kind of blindness as when the retinæ are destroyed. When the primary visual area of

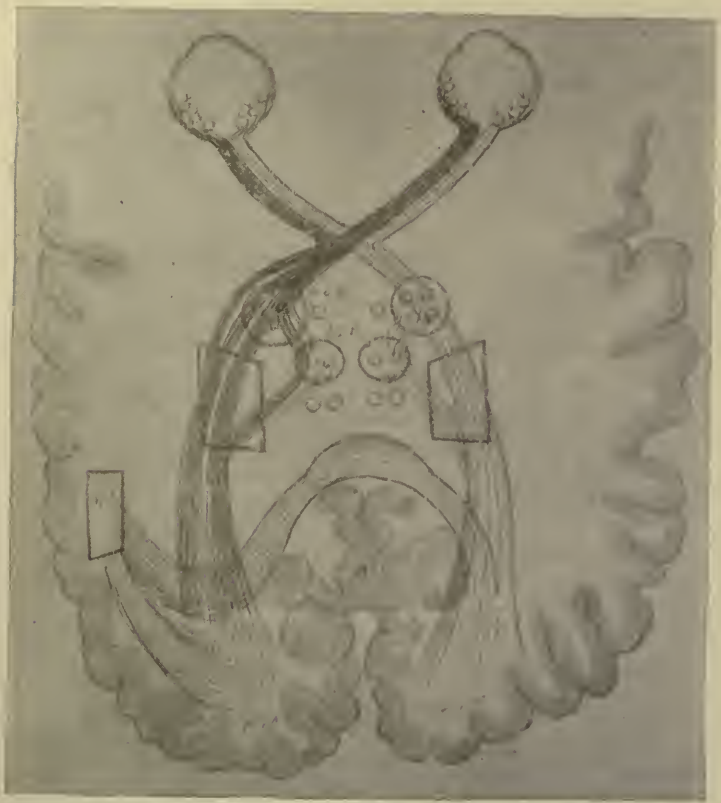

FIG. 13.—Course of the Optic Fibres. *EGB*, External geniculate body; *AQB*, anterior quadrigeminal body; *P of T*, pulvinar; *VC*, visual centre; *HVC*, half-vision centre.

one side is destroyed, the blindness that follows is of half of each retinal field, and on account of the partial decussation in the chiasm this blindness is homonymous, that is, of the same half. This visual defect is shown graphically

by the adjoining figure, which is based upon recent knowledge of the course of the primary optic neuron.

There are two or three subdivisions of visual aphasia, to which I shall refer. The first is a form in which there is loss of the perception of the word, not only of the visual impressions corresponding to the word, but a loss of the value of the symbol in arousing the idea of which it is the written representation. This form of sensory visual aphasia is entitled to the name loss of word visualization, verbal amnesia, or psychic blindness of words. Unlike word blindness, the patient interprets letters as letters and words as words. They are not simply marks on paper, and he can read them and copy them, but they convey no meaning to him after he has read them. When they are pronounced before him he hears and interprets them very readily, but he has no idea that they are the same words that he has been reading or copying unless he is so informed. It has been suggested that psychic blindness of words is a phenomenon exactly analogous to that which occurs on reading a book when our attention is absorbed with something entirely apart from the subject matter before us; we read on, line after line, we reach the bottom of the page, and not until the necessity for turning the page arouses us do we find that although the page has been faithfully read not a word has entered into consciousness, nor can we cite a single fact that the writer attempted to convey.

It is unnecessary to dwell upon the difference between this and letter blindness and word blindness. It is readily seen that the lesion which produces such a condition must be one that interferes with the pathway that conveys

the sensation from the printed word or object to the idea, or to where the idea is formed; and we are warranted in saying that such interference is nearer the seat of consciousness, wherever that may be, than it is to the seat of memory images of words.

A second subdivision is that in which the patient on looking at an object, which he has previously seen and used is unable to call up its name, although he is in condition to utter the name if he could call it up. This is the condition to which the name optic aphasia has been given by Freund. The striking symptom is the inability to name things. This inability is not difficult of interpretation. It depends upon an interruption of the pathways that unite the seat of cortical visual representation and the seat of cortical auditory memories. It is necessary in order to enunciate a name to influence articulatory images through the auditory centre; in other words, the impulse that starts the externalization of a name travels along the intercentral (or internuncial) auditory-articulatory pathway, and if auditory images are properly revived, and there be no disturbance of the articulatory kinæsthetic area, the name will be uttered. If, however, something prevents the impulse from passing through the visual apparatus by which one gets a visual concept of the object and thus from reacting on the seat of auditory speech images, then calling objects by their names, when such are presented to the peripheral organ of the visual apparatus, will be entirely impossible. On the other hand, if the object is of such a nature that it can appeal directly to the auditory apparatus, the auditory mechanism being intact, the name may still be produced. One or two simple examples

will suffice to show this clearly. If a photograph be held before an individual and he is asked to designate it by name, an impulse travels from the percipient ocular apparatus to the primary visual centres and to the higher visual centre, and the impression thus produced will excite the auditory centre, to which the articulatory kinæsthetic area is subservient, and the result will be the enunciation of the word "photograph." On the other hand, if the visual mechanism is destroyed, and the photograph is held to the ear, the patient will not be able to name it, although the auditory mechanism is intact. The photograph has no qualities that appeal to the ear. If, however, the object used be an apple, a patient with loss of visual memories, but with auditory memories intact, may still be able to name the word "apple" if the object be pared and sliced within his hearing. The occurrence of this condition, with its sharply defined symptomatology, has led certain writers to allocate special portions of the speech area as a "concept centre" and a "naming centre." Broadbent was the first to advance this view, in a paper published in 1872.[1] In his own language: "There is a primary or rudimentary perceptive act in which the external cause of a given set of sensations is recognized as such, and in which the simple attributes, as of form, color, hardness, etc., are perceived. And there is a higher degree of elaboration in which, by the combination or fusion of perceptions derived from the various organs of sense, a conception or an idea of an object as a whole is obtained. This is a new and distinct process, and is usually accompanied by the affixing of a name to the object. The higher

[1] *Loc. cit.*

elaborations and the fusion of various perceptions together, and the evolution of an idea out of them, will be accompanied not by radiation from one perceptive centre to all the others, but by convergence of impressions from the various perceptive centres upon a common intermediate cell area, in which a process analogous to the translation of an impression into a sensation and of a sensation into a primary perception will form a part of the supreme centre, and will be situated in the superadded convolutions which receive no radiating fibres." In the further elaboration of this theory, Broadbent postulated a "proposition-izing" centre, in which words other than substantives were registered and in which words were arranged orderly preparatory to being uttered.

It seems to me unnecessary to point out how completely at variance this is with the conception of speech that I have attempted to portray in another chapter, and how contrary it is to the idea that the various speech centres have an integral part in the conception of words, the symbols of thought. The existence of such a centre is, I believe, contrary to the conception of the psychogenesis of speech. The reasoning on which its existence is posited is fallacious, and the cases that have been cited to establish its autonomy are made to support it only by putting unwarrantable interpretation upon accompanying symptoms. If such a centre existed, it is reasonable to suppose that cases of aphasia would have been described before this in which the symptoms pointed to the destruction of this area, especially considering the autonomic activity that its sponsors give it. I have been unable to find any such cases; that is, no case in which the speech disturbance

could not be explained by a lesion of one of the speech centres or of its afferent and efferent pathways. The case cited by Mills[1] in support of this contention is reported in such an incomplete and fragmentary fashion that I am astonished that one of his clinical astuteness and erudition should consent to offer it in evidence. The following is a *résumé* of his case:

A woman, forty-five years old, complained of numbness in the back of the head and neck, vertigo, and vomiting. Soon after this it was noticed that she did things differently from her usual custom. She hung upside down a certificate of membership in a society without realizing her mistake. She had to forego her occupation, that of dressmaking, because she no longer got the parts together properly, etc. *In other words, she developed optic aphasia.* (If she had been tested there would have been found, in all probability, dyslexia or alexia, but no information is given concerning the reception, interpretation, or emission of speech.) About three years after these symptoms were first noticed, she had an epileptic fit, and after this forgetfulness of words became very evident. Examination showed left lateral homonymous hemianopsia, word blindness, but not letter blindness, inability to name objects, it mattered not through what receptive avenue she got information concerning them, although she knew their uses. I infer there was considerable agraphia, although nothing is said about it, for a fac-simile of the patient's signature shows the last name unfinished. There was slight paraphasia in spontaneous and repeated speech, but the former soon became limited to the use of "Yes" and "No," which she used properly. About ten weeks before death there developed slowly a right-side hemiplegia. On

[1] Mills: Journal of Nervous and Mental Disease, p. 1, 1895.

autopsy, which is most meagrely reported, there was found, on cutting into the temporal lobe, a hard tumor, yellowish-brown in color. Its hardest and apparently oldest part was in the middle of the third temporal gyrus, but a firm, nodulated, more or less hemorrhagic condition extended backward as far as the white matter of the occipital lobe and forward toward the anterior ends of the second and third temporal convolutions. It was thought that the disease started in the third temporal convolution, at a point in line with the posterior extremity of the horizontal branch of the fissure of Sylvius.

I find it difficult to convince myself that the gradually developing optic aphasia and word blindness in this case were due to anything else than a gradually progressive lesion which encroached upon the projection tracts of the visual fibres, the optic radiations, which lesion was manifest by such infallible evidence as homonymous hemianopsia, and which later severed the connections between the area of visual and auditory speech memories, the invocation of which by the former is necessary before concrete words can be used. (Mills reports that the patient had left homonymous hemianopsia, which I take it refers to the retina, and not to the visual field; in other words, blindness of the right side of the fields.)

The patient showed optic aphasia by her inability to recognize that a certificate was hung upside down, and by her inability to go on with dressmaking. This symptom, in all probability, coincided with disturbance between the cortical area for visual reflection in the cuneus and the area in which visual memories are stored up in the angular gyrus. That the interruption became more profound

and extensive is shown by the development of word blindness and homonymous hemianopsia; that it never became absolute is shown by her continued ability to recognize letters; that her sensory speech area became gradually destroyed is shown by the fact that she became almost, if not absolutely, unproductive of voluntary speech. The area of articulatory images was cut off from the centres that excite it to activity and control, and the result was almost the same as if there had been no articulatory kinæsthetic centre—a locomotive with steam but without a driver. Moreover, the patient was not wholly agraphic, although she wrote with great difficulty. Now, if there is any one fact substantially proven in sensory aphasia, it is that the cases of aphasia in which the patient is word blind, but still not agraphic, are dependent upon a lesion in the white matter of the occipital lobe which severs the fibres connecting the higher visual centre with both of the primary visual centres in the two hemispheres. The lesions found on post-mortem examination in this case show, so far as they indicate anything, that they severed the connection of the auditory centre with the visual centre, and the connections of the latter with the primary visual areas.

In addition to the kind of visual aphasia in which there is loss of memory for written and printed letters, and to which the name verbal amnesia or psychic blindness of words has been given, there is a form of less common but more striking occurrence, known as psychic blindness or mind blindness, the "Seelenblindheit" of the Germans, "Cécité psychique" of the French, a condition not infrequently associated with the ordinary form of word blind-

ness and letter blindness. In this condition the patient not only does not recognize the significance of letters, but he loses the power to differentiate between familiar objects or persons and to distinguish the use of things. He looks at a member of his own family and sees, apparently, a stranger. He does not recognize the house in which he has lived for years, neither from the outside nor from within, and frequently asks when lying in his own room when he is to be taken home. Such patients do not recognize a washbasin from a drinking-glass, and drink as readily from one as from another. They have no more conception of the use of a fork or of a spoon than has an aborigine.

The condition known as apraxia, the inability to comprehend the usage of ordinary objects and things to which one had been accustomed, is analogous to this. Its occurrence and misinterpretation in former times were often responsible for getting the unfortunate possessor into an asylum. This is not at all surprising when we consider that a very similar symptom often occurs during the very early stages of general paralysis. A patient with the latter disease, now under my care, first showed symptoms of mental illness by persistent and repeated refusals to enter his own house, insisting that the one in which he lived looked entirely different. Even before any other symptoms of mental disorganization were noted, he showed himself quite incapable of finding his house or the street in which it was located, or of recognizing it when brought to it. A similar condition is often seen during the occurrence of very severe illness, such conditions being looked upon as the visual hallucinations of delirium.

The nature of the condition is probably an abolition of the visual memories of objects, which memories have been stored up in the higher visual area, a condition analogous to that of word blindness. The patient who has this condition may see the object; that is, he sees it objectively, but he does not see it subjectively. When we are as yet small children, we have no conception of the uses of a great many things that impinge themselves on our visual apparatus. which afterward become very familiar to us. In the acquisition of this familiarity there is stored up in the higher visual centre not only an image of the object itself, but with it a memory picture of its use. This latter may be a combined kinæsthetic-auditory-visual one, the latter being for most objects by far the most important one. When the visual image is lost, the memory picture cannot be rehabilitated. As Wyllie has remarked, the imprintation or storage of the images of objects does not require such close attention as the imprintation or storage of words, and the images of objects and things are stored in both hemispheres of the brain. This would account in a measure for the infrequent occurrence of this condition even in cases in which the sensory speech area is very largely destroyed. It would also lend itself to the interpretation of the occurrence of this symptom in the early stages of general paresis, in which the lesion is a widespread degeneration of the cortex.

CHAPTER VI.—(*Continued*).

Subcortical Sensory Aphasia.

The subcortical forms of sensory aphasia, pure sensory aphasia of Dejerine, are analogous to the subcortical forms of motor aphasia. In discussing subcortical motor aphasia, it was said that the symptom complex attending that condition was the result of a lesion that prevented the idea, properly and completely formed, from being externalized in a word or words. In similar fashion the lesion of subcortical sensory aphasia is one that interferes with the passage of the spoken and written word to the idea of the word or to where the idea is formed, it being understood that the structures by whose functioning the idea of the word is formed are intact.

The real components of sensory aphasia are visual aphasia and auditory aphasia, and it follows that the visual cortical area and the auditory cortical area are the parts by virtue of whose activity one gets a visual and auditory idea of words. Therefore, there may be a subcortical interruption of the visual and auditory pathways which causes a subcortical visual aphasia and a subcortical auditory aphasia. I shall not attempt to discuss these *in extenso* here, as I have already referred to them in discussing the true forms of sensory aphasia.

The symptoms of subcortical or *pure* visual aphasia will be readily understood if it be borne in mind that the vi-

sual centre itself is intact and ready to functionate and that it only awaits the impulses inciting it to function which proceed from the half-vision centres. In subcortical visual aphasia connection with the primary visual centres is severed, and consequently the higher visual centre, although retentive of its anatomical integrity, is perverted in its physiological ability. The connection of the visual centre with the other speech centres is not disturbed, except in so far as the latter are not properly and customarily influenced on account of the fact that the visual centre itself does not itself receive customary stimuli. This accounts for the occasional occurrence of shortcomings of speech (slight paraphasia, such as noted by Bramwell), which might otherwise seem paradoxical.

The symptoms of subcortical visual aphasia vary somewhat with the seat of the lesion, *i.e.*, with its proximity to the left angular gyrus. Generally speaking, they are verbal blindness, always associated with right lateral homonymous hemianopsia, as the lesion is either of the primary visual area, in the cortex of the occipital lobe bordering on the calcarine fissure, or of the optic radiations connecting the occipital lobes with the left angular gyrus, the higher visual centre. The patient looks at printed and at written letters and sees only black marks on a white surface; he is wholly unable to interpret them, because the visual impulse is prevented from reaching the left angular gyrus, in which such impulses are interpreted by comparing them with the residua of other impulses the significance of which have been registered in consciousness and which are known as memory pictures. The fact that the centre in which are stored such visual memory images is

intact, and the images are therefore preserved, can be shown in a variety of ways. In the first place, they can be revived through kinæsthetic stimuli. If the patient has been long habituated to writing, it may be possible for him to interpret the significance of written letters and words by tracing them with the finger end. Printed symbols remain unintelligible to him, because he has not been accustomed to tracing printed characters, and he has no kinæsthetic memories and association tracts for such impulses which can proceed to the angular gyrus. It is not always possible even in one accustomed to writing to revivify visual images in this way. In the second place, patients with subcortical visual aphasia retain the capacity to spell. Spelling consists, in most persons, of the memorial recall of auditory images which are sent to the other two speech centres, the articulatory and the visual. As these centres are intact, capacity to spell is undisturbed. Patients with subcortical visual aphasia are able to copy, but they delineate without deviation from the copy before them, and copy print in print, script in script.

These are the prominent symptoms. Spontaneous speech, except occasionally the slight paraphasia already referred to, and capacity to repeat are intact. The patient is able to write voluntarily and from dictation, but he cannot read what he has written any more than he can read what some one else has written, except in those instances in which the sense can be gathered from tracing each letter with the finger tip. The patient understands readily what is said to him and can reply intelligently and correctly.

Subcortical visual aphasia may or may not be accom-

panied by a degree of optic aphasia manifested by inability to name objects. Whether or not these patients have any difficulty in spelling needs further investigation. In all probability they do not, save in rare instances. Bramwell has recorded an example of this form of aphasia in which the faculty of spelling was preserved to such a remarkable degree that the patient became the speller for the family.

As an example of subcortical verbal blindness which is every way typical I shall cite the following instance studied in my clinic at the Post-Graduate Medical School and later in the City Hospital.[1]

The patient is a male, fifty-eight years old, by occupation an artisan, whose life has been one of uniformity. Although he has not had syphilis, rheumatism, or gout, his blood-vessels show well-marked arterial sclerotic change. His present infirmity dates back five months. He had been complaining somewhat of not feeling well, of headaches, and of some vertigo, when without other warning he became unconscious and lay in a semi-unconscious, semi-delirious condition for about three weeks. There was no evidence of hemiplegia. He recovered his mental balance after three weeks, but has not since that time been able to return to his occupation. His wife says that this incapacity is the result of bodily weakness, forgetfulness, and because he does not remark anything. He comes to the clinic on account of a stupid feeling in the head, and because he is unable to work. Examination shows that voluntary speech is without notice-

[1] I regard the case as particularly valuable because of the corroboration which the examination of the brain, thus far made, gives to the statements made from clinical data alone. The patient died six months after the monograph had left the author's hand, and, as will be seen on further perusal, the autopsy fully bears out the diagnosis.

able defects. He talks in a somewhat more confiding way and with a little more emphasis on words than one usually does, and occasionally he misplaces a word. His wife again comes to our aid and says that he misuses words at home, although I must say that I have not been able to remark any considerable paraphasic speech disturbance, although if he is not understood quickly he seems to get impatient and with a gesture significant of impotence he turns to his wife. When his wife is asked to cite specific examples she says there are many, but she has difficulty in recalling one. Finally, she says that if she sent him to a shop for soap he would be quite likely to bring back flour. This surely cannot be cited as a paraphasic manifestation. In repeating sentences there is occasionally sight misplacement of words, but no more, I think, than would be expected in one of his intellectual attainments. Pantomime and mimicry he does not indulge in. He understands what is said to him, although he is not quite so alert as formerly. He says that he has to be attentive to what is said to him, and that if the sentence is very long he loses the connection. Examination of the eyes shows a complete lateral homonymous hemianopsia, which is shown by the accompanying chart. The patient is unable to read, that is, there is complete word blindness. Occasionally he can make out a letter here and there, but there is almost complete letter blindness. He sees the letters very distinctly and is able to tell how many letters there are in a word, but he is totally unable to mark off a word into syllables or to take a number of detached letters and construct a word or syllable out of them. There is no object blindness. He recognizes things and calls them by their right names. Voluntary writing is preserved, but, with the exception of his name and address and a few words, he is not productive. After

writing a few words, he foregoes further efforts with some despairing remark. He writes badly to dictation after the first few words, the writing from dictation showing only two striking features: first, he is apparently unable to retain in mind a sentence sufficiently long to write it; and, second, he stops the line before he gets to the right side of the page. Writing from copy is very defective; what he copies he does laboriously and servilely, the let-

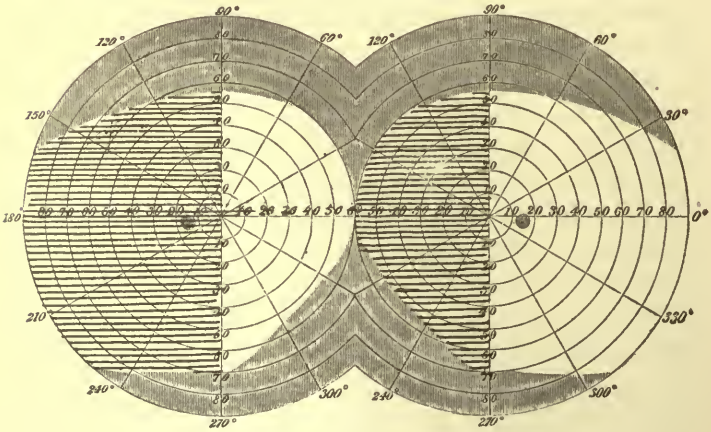

FIG. 14.—Complete Lateral Homonymous Hemianopsia.

ters being an exact reproduction of the copy. Script is copied as script and print as print. There is no optic aphasia or mind blindness. The patient is quite as intelligent as are most people of his age and social position; he is able to compute mentally and to spell, and is in fair possession of his associative faculties.

In the examination of his eyes, it was interesting to note that when a lighted candle was brought into the right visual field the light of the candle was not seen until after it had passed the median line, although the patient detected at once that the atmosphere was brighter as soon

as the candle was brought into the space of a normal visual field. In other words, as soon as the illumination struck the retina he said, "It's brighter," but he did not see the light.

A specimen of his voluntary writing is reproduced here. It is an attempt on his part to tell me in writing

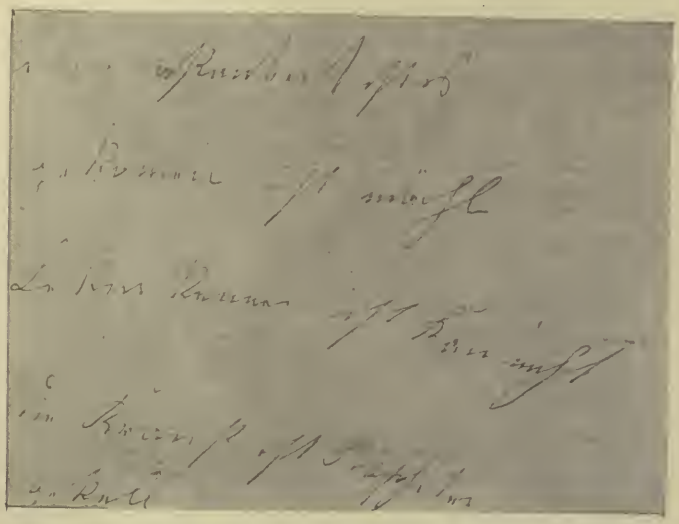

FIG. 15.—Specimen of Voluntary Writing.

how his sickness came on—to tell me all about it. The words have no significance. So far as they can be made out they are: "Dare durg [the last letter is really nothing] akenbeit ist es gekommen ist möchte lesen, ist kann nicht. Die Krankheit ist," etc. When I ask him to read what he has written he begins pointing to each word in turn "Ich möchte gern lesen, ich möchte gern lesen"—all this slowly and with some emphasis. On being asked to spell the individual letters entering into the formation of each word he says, pointing to the first, second, third, and so

on, "That is e, that is also e, that is e, that is also e," and so on, until he seems to become tired.

I repeated slowly the first lines of Schiller's poem, "The Casting of the Bell":

> "Fest gemauert in der Erden
> Steht die Form aus Lehm gebrannt;
> Heute soll die Glocke werden," etc.

and asked him to write them. Although this was repeated line by line and he could say it himself quite well, the following is a fac-simile of what he produced when he endeavored to write it from dictation (Fig. 16):

The letters are formed, and some of the words are real words; there is no sense in them or any suggestion of the original. He does not give the slightest heed to commands addressed him in writing, such as, "Put out the tongue," although he responds quickly when told to do so in spoken words.

In this case the symptoms are, briefly, word blindness, alexia, right lateral homonymous hemianopsia, inability to copy, and defective writing from dictation. Spontaneous speech and repeated speech are not disturbed, nor is there noticeable defect in the interpretation of spoken words. These symptoms and possessions point to a lesion between the angular gyrus and the half-vision centres in the occipital lobes, situated in the posterior end of the left hemisphere. The fact that he does not write facilely either spontaneously or from dictation, leads me to believe that the lesion is situated close to the angular gyrus and is perhaps encroaching upon it.

The lesion, which was originally either a hemorrhage or a thrombosis, must have so completely implicated the white matter of the occipital lobe that it cut across the fibres

passing from both occipital lobes to the left angular gyrus, involving the optic radiations of Gratiolet. The fact

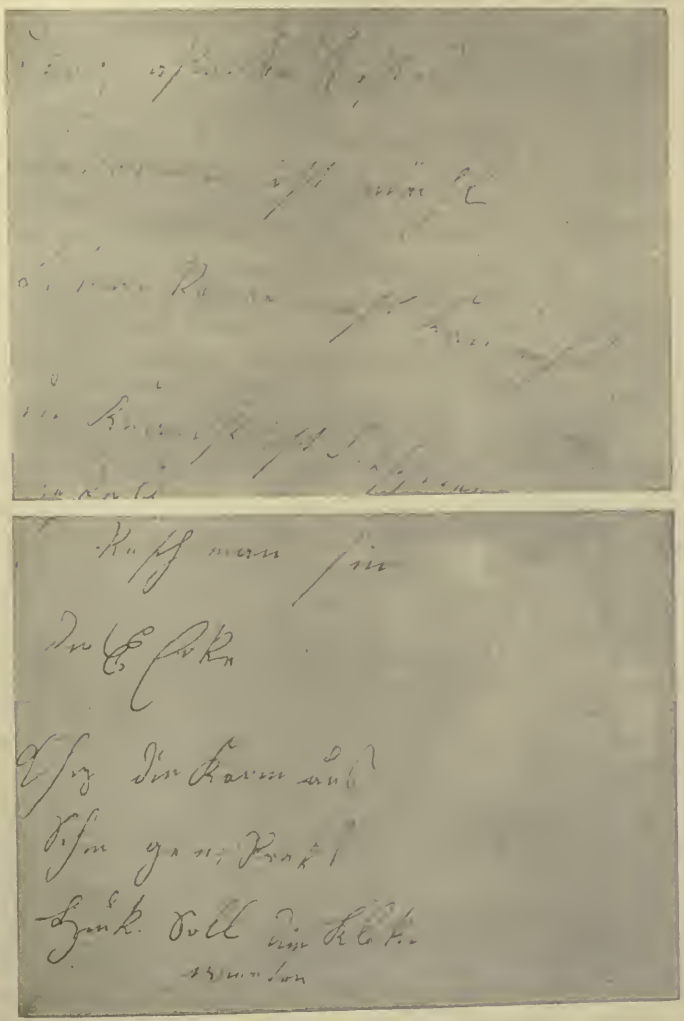

FIG. 16.—Specimen of Writing from Dictation.

that there are word blindness, left hemianopsia without agraphia, but no object blindness, shows that the lesion cannot be either in the primary visual centre or in the higher visual centre. For if it were in the primary visual centre there would be object blindness; the lesion to produce hemianopsia would not cause word blindness, because in such a case the higher centre would still be in connection with the primary occipital area of the other side. The lesion is not in the higher visual centre, because if it were there would necessarily be agraphia and pronounced disturbance of internal language, while in reality neither of these exists. The general mental infirmity is no greater than would be expected from an intracranial lesion so grave as this must be, and in a patient who presents the striking manifestations of vascular degeneration that this one does.

So much for the clinical deductions. The patient was admitted to the writer's wards in the City Hospital, where repeated examinations showed no material departures from the above-stated findings. Although apparently a docile, tractable individual, it was found that he had but slight control of his temper, and when aroused, oftentimes even when not excited, he indulged in profane and vituperative language, which was directed against his attendants and those about him. His ability to create internal language and to externalize it in spoken words was fully evidenced by numerous letters sent to his family which were dictated to a fellow-patient. His health was considered to be in fairly satisfactory condition until November 8th, 1897. On the afternoon of that date while sitting on a bench in the garden, he fell over suddenly

and had a more or less generalized convulsion accompanied by frothing at the mouth. He was got into bed immediately and seen a few minutes later by the house physician, who reported that the patient did not utter a word or apparently recognize any one after the attack, but gradually sank into unconsciousness, and meanwhile a right-side hemiplegia developed. The pupils were uneven, owing to contraction of the left pupil; the face was flushed and showed a right-side palsy; the axillary temperature was 98.2° F. and alike on both sides; the respirations were stertorous, and 32 per minute; the heart was working laboriously, while the pulse, beating 84 times a minute, was very hard and firm. Both knee jerks were absent. The unconsciousness deepened, the patient vomited copiously, and soon the left side of the body ceased to indulge the continuous movements that were first noticed. Two hours after the apoplexy he became quadriplegic, and died two hours later, about four hours after the onset of the symptoms.

An autopsical examination was made a few hours after death, and, aside from the changes in the brain about to be described, there was no noticeable abnormality save an advanced degree of arterio-sclerosis, most noticeable in the heart and large blood-vessels and in the kidneys.

The skull is of the customary thickness. The veins and diploe are well filled. The dura is easily detached and not thickened, and the brain *in situ* looks normal. The pia is smooth and glistening and its vascular arborizations are very distinct. Even before the brain is removed from the calvarium, but more conspicuously after removal, a most striking abnormality is seen at the posterior pole of the left hemisphere. The entire occipital lobe looks reddish-

yellow, is extremely soft to the touch; in fact, the lobe is replaced by a cystic formation, all save the posterior extremity, where there is a slight cortical mantle, evidently not yet implicated. When the fluid of the cyst escapes the cortex of the occipital lobe sinks in, and the superior surface of the left cerebellar hemisphere juts prominently into view. The same yellowish-red color and the same pultaceousness to the touch are apparent on the internal surface of the brain, where the destruction of tissue involves nearly all the lingual gyrus and the cuneus, except a very small thin surface at the posterior pole of the latter, which seems to have retained a fairly normal appearance to the naked eye.

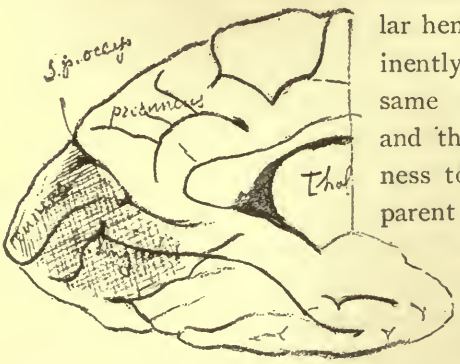

FIG. 17.—Shaded Area Shows Extent of Lesion on Mesial Surface.

In short, the cystic formation involves the whole posterior part of the left hemisphere, save the very apex of the cuneus and particularly the inner surface, being limited on the mesial surface (Fig. 17) anteriorly by the parieto-occipital fissure, and on the external surface by the prolongations of the same fissure. The gyri involved are the first, second, and third occipital, the cuneus and lingual gyrus. The remainder of the left hemisphere is apparently normal to the view and to the touch. The accompanying illustrations, made at the time of the autopsy, show the

location and the extent of the lesion. It will be seen that the destruction of tissue extends anteriorly as far as the posterior limits of the inferior parietal lobule, the precuneus, and the angular gyrus, but spares these absolutely. The temporal lobes are likewise quite unimplicated.

The depth of the lesion, originally a hemorrhage which had undergone cystic transformation, was almost through

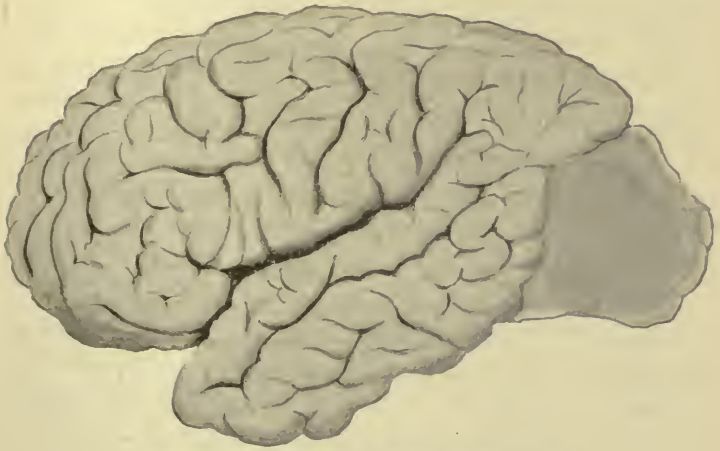

FIG. 18.—Shaded Area Represents Seat of Lesion.

the entire thickness of the white matter and into the roof of the ventricle, so that the optic radiations were completely cut across. When the isthmus of the encephalon was separated by a horizontal cut of the pons anteriorly to the apparent origin of the trigeminal nerve, the cross-section showed a distention of the aqueduct with sanious fluid. When the two hemispheres were separated both lateral ventricles were found filled with clots, the left much distended, the foramen of Monro and the middle ventricle also distended, the primary fatal hemorrhage apparently

being in the left lateral ventricle. The hemispheres were then cut according to the method of Flechsig and the brain was placed in Müller's fluid for hardening preparatory to further study.

This observation needs no further remark, save to say that the symptoms which had been present were so accurately substantiated by the post-mortem findings so far determined that it amounts almost to the exactness of a mathematical demonstration.

Subcortical word deafness, or *pure* word deafness of Dejerine, is characterized especially by inability to understand spoken words, and naturally by inability to write from dictation, because the spoken word of another cannot get to the part of the brain in which the idea of the word is interpreted. It is of much less frequent occurrence than subcortical visual aphasia. The feature that distinguishes it from cortical auditory aphasia is the fact that spontaneous speech is preserved, there is no amnesia verbalis, the patient is able to read aloud, to write voluntarily, to copy, and to read understandingly what he and others have written. As in every other form of subcortical aphasia, aphasia in which the zone of language itself is not diseased, every constituent of internal language is intact. It is rarely if ever associated with paralysis of the extremities.

A classical example of this form of aphasia is that cited by Lichtheim.[1] A journalist had an attack of apoplexy which was accompanied by pronounced sensory aphasia, from which he made a partial recovery. Five years later he had a second attack and again recovered, but this time

[1] *Loc. cit.*

more completely than before, so that now there was no difficulty in speaking, in reading, or in writing. He went on with his work as a journalist. He was, however, absolutely word and tone deaf, although hearing remained quite undiminished.

It has been mentioned casually in another connection that recently Bleuler,[1] Freund, and other writers have contended that the customary conception of subcortical auditory aphasia is entirely too narrow, confined to too limited an area, and that it should be enlarged to include disease of the extracerebral neural structures whose functioning conditions audition; at least, that disease of the peripheral auditory neuron, including the termination of the nerve in the organ of Corti, thus taking in labyrinthine diseases, should be included.

Freund,[2] in a recent monograph on this subject, cites the following case, in which bilateral disease of the labyrinth following epidemic cerebro-spinal meningitis caused a form of word deafness:

A youth, twenty-two years old, by occupation a watchmaker, developed after prolonged physical effort, followed by rapid chilling from lying on the cold, damp earth, symptoms that led to the diagnosis of epidemic cerebro-spinal meningitis. The disease was of the severe type, and the patient did not convalesce until the sixth week. It was then noticed that, in addition to vertigo and profound disturbance of equilibration, hearing was very much impaired. Soon after this he was treated in the otological clinic of Professor Gruber for deafmutism.

[1] Bleuler: "Zur Auffassung der subcorticalen Aphasieen." Neurologisches Centralblatt, 1892, No. 18.

[2] C. S. Freund: "Labyrinthtaubheit und Sprachtaubheit," Wiesbaden, 1895.

An examination made five months after the beginning of the illness showed: Loquaciousness; speech rasping and of harsh intonation; no paraphasia; no optic aphasia. The patient appeared with a pad and pencil for the use of his questioner, as he claimed to be wholly unable to hear spoken words. There was no disturbance of motion, sensation, or vision. There were slight vertigo and difficulty of maintaining equilibrium. Otoscopic examination of the ears did not reveal any departure from the normal. Examination of the sense of hearing showed absolute deafness on the left side, while on the right side a few vowels, such as *a*, *e*, could be heard when they were shouted into the ear. Aerial conduction of notes of a tuning-fork was entirely unperceived. Bone conduction of the same was unperceived, although the patient was sensible of the mechanical vibration.

There was no dyslexia or agraphia. His speech was interfered with only in so far as understanding of spoken words was entirely lost, and naturally there was inability to repeat spoken words and to write from dictation.

All acoustic impressions impinging on the right ear were, when not too low, perceived by the patient, but they were not properly interpreted.

In order to determine accurately what degree of comprehension he had for articulatory sounds, the patient was subjected to a painstaking examination. The vowels and consonants were shouted separately in the right ear, and the patient was apprised in writing how to indicate when he had heard them, what he had heard, and made to understand fully the test that was to be made.

The examination showed that the patient, who was apparently completely speech deaf, possessed the capacity to grasp during the examination a few words correctly and others very nearly correctly. As he expressed it, "I hear

only the tone of the words, and I then must think what they can be." Perception of rhythm was well preserved, and he had good conception of the number of syllables in the uttered word and the intonation that was given to them. When asked to differentiate between the words "December" and "June," he did so with readiness, although he was often unable to differentiate between words that sound somewhat alike, such as December and September, or June and July.

The patient's intact, spontaneous speech confirmed the opinion that he was in possession of his auditory word images. Examination of his music-perception ability showed that he did not hear musical sounds so clearly as before his illness; for instance, he was unable to tune a violin, but sounds coming from the street, the beating of horses' hoofs, the lumbering roll of heavy wagons, the sprightly sound from light vehicles, the jingle of the tram-car bells were heard and differentiated. He could differentiate whistling, the snapping of fingers, the clucking of the tongue, the clapping of hands, etc., and he very seldom erred in naming the source of such sounds. He could likewise indicate the origin of tones from a violin, a trumpet, a piano, a mouth harmonica, etc. Entire tones he differentiated with greater readiness than half-tones, and for high tones there was striking impairment.

In response to the question whether he could hear himself talk, he remarked that he could hear his own words but not clearly and distinctly, and the more trouble he had in hearing them the louder he spoke. When eating he always heard (?) the grinding noise of the teeth.

The absolute deafness in one ear, the diminished bone conduction and loss of perception for high tones in the other, the normal condition on examination with the oto-

scope excluded absolutely middle-ear disease and bespoke a disease of the auditory percipient neural mechanism, due to bilateral labyrinth disease following epidemic cerebro-spinal meningitis.

I have quoted this case in detail because it may be said that no unsurmountable objection can be raised to the admission that word deafness similar clinically to that found in subcortical sensory aphasia was present. The only question is whether one is willing to admit that the peripheral auditory neuron can be normal for the conduction of ordinary sounds and diseased for the conduction of sounds having highly differentiated significance. Personally I see no objection to entering such cases as this in the category of subcortical sensory aphasia.

CHAPTER VII.

Total Aphasia.

Occasionally cases of aphasia are encountered in which there is a disturbance of all forms of intellectual expression, involving disturbance in the reception of stimuli that condition mental states preparatory to speech, and disturbance in the emission of such mental states. To such cases the name total aphasia is given because it includes the phenomena of both motor and sensory aphasia.

If the location and the relationships of the speech centres be kept in mind, the existence of such a condition will not be at all surprising. The speech area is dependent for its blood supply, and therefore for its functional integrity, upon the artery of the fissure of Sylvius, and a lesion of this artery that interferes with the circulation is apt to show its malign consequences in every part dependent upon the artery for nutrition. In individual cases the area supplied by one of its branches may be more profoundly affected than another. Thus in cases of total aphasia we sometimes see one or more components of language less wholly submerged than others, and, on the other hand, cases are seen in which the aphasia in the beginning is total, yet after a time the symptom complex becomes so modified that one of the modes of language may be partly recovered.

As an example of total aphasia, I may cite the following instance:

F. C——, thirty-eight years old, a native of Italy, by occupation a bank clerk. He is married and the father of two unhealthy children. His wife has had two miscarriages. There is no way of determining whether he has had syphilis. He has been a steady drinker for many years. After he had suffered from a condition which the family physician called influenza and which kept him in bed for about two weeks, he became very dizzy, and experienced the sensation of falling while passing from one room to another. Soon after it was noticed that the right upper extremity was very unwieldy and that the right side of the face was somewhat drawn, and that he rapidly became unable to articulate. In fact, save for complaint of a severe pain shooting through the head immediately after the occurrence of the vertiginous spell, he did not utter a word. His wife says that there was not the slightest loss of consciousness, but that the patient was "out of his head" up until a few days before I first saw him, which was six weeks after the onset of his sickness. The slight hemiplegia from which he had suffered disappeared in a few days. His wife, furthermore, states that he has not uttered a word that could be understood; that he has not understood what was said to him; and that he has not essayed to read since the beginning of his illness. She says that he is in his right mind, that he does nothing foolish, except to watch her or any other person who may be in the room with embarrassing persistency. Examination shows that voluntary speech is wholly lost, and that he makes not the slightest effort to repeat when he is instructed to. He never indicates wishes, desires, or other mental states by pantomime, and his wife tells us that he has not done so since the begin-

ning of the illness. When he is given a pencil and asked to write he makes a few up-and-down strokes, but frames no letters or words. On being requested to take off his coat he looks intently at the one who gives the command, watches his lips most carefully, and grunts (nothing approaching articulation), "Sih, sih." After the request is repeated a number of times, each succeeding time in a louder voice, it would seem that he gets some idea of what is wanted, for he grasps the lapel of the coat, but makes no further effort to remove it. When I suggest my request by aid of pantomime by throwing off my own coat and then pointing to his, he obeys. To the command, "Put out your tongue," a number of times repeated, he closes the eyes. When he is asked to take the doctor's hand, he does not apparently have any conception of what is desired. Under further questioning and commands, he becomes very restless, gives vent to inarticulate sounds, moves the lips as if counting or mumbling, twists the fingers, and then starts to go out of the room as if he wished to go home. To test for visual perception he was shown the following request, first in writing and then in print: "Take out your watch and show me the time." No response. On the contrary, after adjusting his glasses, he reaches forward, takes the pencil from my hand, and begins to copy the words in script. On endeavoring to interpret this action on the part of the patient, I learned that some days previously he had been given a letter addressed to his employer which had been prepared by his brother and which he was requested to copy. This letter set forth that the patient was not very ill, was not out of his head as it had been reported to his employer, and to show that his mental faculties were unimpaired, and that his position should be kept open to him, this letter written by his own hand was offered in evidence. The

deception, however, did not work, as the concocters of it had never been able to get the patient to do the part allotted to him. He could copy, however, and he copied script in script and print in print. To the written question, "How old are you?" first in printed and then in written characters, he takes a pencil and points to the individual words, at the same time making this inarticulate "Sih, sih, sih," and then he starts to copy it, without, however, giving the slightest intimation that he gets the purport of the question. It is impossible to say positively that there is no hemianopsia, but probably there is not, as thrusting the finger abruptly into the visual field causes prompt blinking.

There is no optic aphasia nor is there mind blindness. The patient fully recognizes the use of things. On being handed a watch, he opens it, looks at the time, compares it with the clock on the mantle, and returns it. On being given a tuning-fork, he sets it in vibration and holds it to the ear. He feeds himself, dresses himself, and goes out alone, and in a general way deports himself as does a person in the possession of his faculties.

Repeated examinations showed no considerable departure from this condition for some time, but after a number of weeks he became able to articulate "Yes" and "No," to write his name, that of his father, and of his wife, and to give evidence that he understood some things that were said to him, particularly if they were repeated a number of times. Voluntary speech, repetition of speech, still remained wholly submerged. Six weeks after the first examination, on being asked to put out the tongue, he did so; but when he was asked to go into the next room he deported himself as one unaware of any request. Still, from this time onward, it was noted that he took greater heed of commands and requests directed to him

through the hearing, and a general improvement of the word deafness followed. On being asked to read a simple sentence, he takes the pencil and points to each individual word, accompanying the indications with the utterance of the sounds eh, fa, eh, fa, eh, la, fe, eh, la, fe, and continues to do this throughout an entire page, pointing to each word. If the sentence embodies a simple request, he takes no notice of it. Frequently, after going through a sentence in this way he looks up at the examiner inquiringly, then shakes his head, assumes a look of distress, draws a line under the word, and goes on. He is now able to copy fairly well, but copies line for line, word for word, as in a drawing. In the same way he is able to copy simple figures and designs. He is totally unable to write from dictation or from written instructions. If he is given a pencil and left alone, he proceeds to write his name, then the surname of his father, and then that of his wife. On being asked where he lives, he writes his father's name; to a second request, he writes that of his wife. If the question is asked him in writing, he proceeds to copy the question. He is able to add up a column of figures, but the results are not usually correct; in fact, they are more often incorrect. He begins at the top of a column, points to the figures, and often says, "Feh, lah, feh, feh, lah," giving a variable intonation to each one of these utterances, then scratches his head, looks disturbed, goes again to the top of the column, and begins all over. His wife says that he is able to play cards, and that it tickles him very much to win. He apparently recognizes the numerals on banknotes, and he can tell time. He never essays to read a paper, and on being given a letter from his parents commiserating with him in his illness he takes no interest in the matter. To recapitulate briefly, this man has (1) loss of spontaneous speech; (2) inability to repeat

after dictation; (3) inability to indicate desires or feelings by means of mimicry; (4) inability to write spontaneously (except latterly he has regained the ability to write his own name and that of his wife and of his father); (5) inability to write from dictation; (6) letter blindness, word blindness, alexia; (7) partial (in the beginning complete) word deafness—he neither understands nor obeys spoken requests. He is able to copy, but copies exactly the words or letters that are put before him. He can interpret requests and commands made to him by pantomime; he can play cards and other games; he recognizes the use of things and the places and relations of objects; he can look at a watch and transfer to paper by means of figures the hour indicated; there is no trace of hemiplegia, unless it be a somewhat lessened activity in the muscles of the right side of the face.

This case shows, therefore, that there was disturbance of all the modes by which one person communicates with another, both receptively and emissively. The only interpretation to put upon it is that after the tenancy of an acute infectious disease a thrombosis formed in the left Sylvian artery, which robbed the speech area of the blood necessary for its functioning. That this pathological process did not cause a serious disturbance of other functions of the brain is shown by the fact that there was no loss of consciousness. That it did not involve the pyramidal projection is shown by the very slight and transitory hemiplegia which left no sequelæ, as organic hemiplegia invariably does. That there was some involvement of the motor cortex, however, is shown by the slight paresis of the right side of the face and the right arm, which, it would seem, was dependent upon disturbance of

function of the ascending frontal convolution adjacent to the frontal operculum. After the thrombosis or whatever lesion may have existed was partly removed, or at least after the circulatory disturbance which it produced was in part compensated for, some of the speech centres regained in a very slight way their functions, and this was particularly true of the auditory centre, for it was the word deafness more than anything else that showed improvement. To what extent improvement will still go on cannot be prophesied.

A very important and instructive case of complex aphasia has recently been reported (although not yet in detailed form) by Bastian.[1] A right-handed man had an attack of right hemiplegia with loss of speech three months before coming under observation. Eighteen years after the first apoplectic attack a thrombosis of the right middle cerebral artery caused death. When the patient was first seen he was incompletely paralyzed on the right side, and there was also incomplete hemianæsthesia on the same side. For six years after the first apoplectic stroke he had slight convulsive attacks from time to time. There was then an interval of twelve years, but during the last year of his life he had three severe fits, each of which was followed by a temporary aggravation of the paralysis of the right side. Within two months of his coming under observation his speech defects assumed the form which they maintained during the next eighteen years with remarkable constancy. The striking features of the man's condition were as follows:

1. Voluntary speech limited to a few words.

[1] Bastian: Lancet, vol. i., 1897.

2. Could repeat words that he heard.

3. Understood everything that was said to him.

4. Inability to read aloud. (The reporter says that he spent much of his time in reading, and undoubtedly understood what he read, yet many patients with sensory aphasia spend much of their time in reading, without comprehending a word.)

5. Inability to write a word from dictation.

6. Inability to name objects at sight.

As no mention is made of hemianopsia it is to be inferred that it was not present.

The autopsy showed a complete atrophy of the convolutions in the territory supplied by the left middle cerebral artery. The atrophy had extended inward so as to lay open the lateral ventricle, and the whole of this region was occupied by a large pseudo-cyst. The supramarginal and angular gyri, as well as the posterior two-thirds of the upper temporal convolution, were included in the parts that had completely disappeared.

The case is regarded by the author as "a very remarkable one; first, because of the complete destruction of the supramarginal and angular gyri and the posterior two-thirds of the upper temporal convolution without the occurrence of word blindness or word deafness." This would be very remarkable indeed if it were so, and would revolutionize in a measure the teachings not alone of aphasia but of cerebral localization. "It is presumed," says the author in attempting to explain these remarkable anomalies, "that the atrophy of these convolutions must have occurred at some unknown period during the patient's illness; that it must have occurred gradually, and that func-

tional compensation must also have been gradually brought about through the further development of the corresponding centres in the opposite hemisphere." The constancy of the speech defects during a long series of years and the very exact way in which the original defects were maintained after some of the functional activity of the lost parts had been transferred to the opposite hemisphere, the writer asserts, are worthy of attention.

Appreciating the great disadvantage that one is at in endeavoring to put interpretation upon a case from a very scant report of the leading symptoms, I hesitate to do more than to mention this case, which I believe will become an important one in the annals of aphasia when it is properly published (so far it has only been reported to a medical society). Nevertheless, it seems that a very different interpretation can be put upon some of the conditions than that mentioned by Bastian. In the first place, he says there was no word blindness, but the fact that the patient had inability to write from dictation (although he fully understood words), the fact that he could not read aloud, and the fact that he could not name objects at sight seem to me to indicate that he had loss of the visual images of words. If he could repeat words that he heard, there is no other lesion that will explain inability to read aloud except loss of the visual memories of words and naturally the inability to invoke such memories from seeing a word. The fact that he could utter words voluntarily and that he could repeat words after they were said to him shows that the auditory images were capable of being aroused, and that they in turn were able to react upon and evoke the articulatory images; otherwise the patient would not have

been able to say a word. But the most convincing feature in leading us to the assumption that the patient had loss of visual memories that are stored up in the angular gyrus of the left side is his total inability to write. Dejerine has shown that spontaneous writing is the result of arousing the visual images here stored and the copying of them by the motor apparatus that holds the pen, while writing from dictation is accomplished in exactly the same way, save that the visual images are evoked in this instance through the auditory. Furthermore, this patient retained the ability to copy, a possession which shows that the visual centre that reflects words, viz., the visual centre in the occipital lobe around the calcarine fissure, was intact; the patient saw the letter, the word, or the design, and copied just what he saw. The fact that he was unable to name objects at sight, although he saw them, recognized their uses, etc., is explained in exactly the same way—inability of the patient to resuscitate the visual memory of the name. Although willing to grant that the uneducated right hemisphere became in eighteen years somewhat educated and functioned vicariously for the part of the speech area that was destroyed on the left side, I am not inclined to believe that this vicarious functioning played a part of such paramount importance in the status of the patient's speech as Bastian would have us believe that it did. It seems to me the interpretation to put upon this case is as follows: The original apoplectic attack was due to a thrombosis of the artery of the fissure of Sylvius, the direct continuation of the internal carotid. The immediate result was a partial hemiplegia of a cortical nature, due to the deprivation of blood from the cortical branches

of the middle cerebral artery going to the Rolandic region; a partial hemianæsthesia, which is to be explained by the fact that the middle cerebral artery supplies in part the posterior limb of the internal capsule. The thrombus in the Sylvian artery caused defective activity or complete overthrow of functions in all the speech centres, and until I hear to the contrary I am prepared to believe that in this patient, before he came under Bastian's care, there were some word deafness, latent or manifest, loss of capacity to read mentally, and much more extensive aphemia than there was three months after the accident. After the substance occluding the calibre of the vessel was removed, or partly removed, some of the speech centres (let us say the auditory, for the temporal lobes are less dependent upon the integrity of the middle cerebral than is the parietal), the area in which are stored auditory images, nearly recovered itself, and there were no gross accompaniments of word deafness, but the loss of visual memories remained in part to the end as did the loss of articulatory images. The successive attacks of thrombosis expended themselves in causing softening and pseudo-cystic formation in the area from which blood had been deprived by the original thrombus.

Thus it seems to me that this is a good example of complex aphasia, in which the symptoms, although perhaps very nearly indicative of complete aphasia, or total aphasia, clear up in one or more modalities of speech as time elapses.

CHAPTER VIII.

Diagnosis of Aphasia.

To unravel the intricacies of aphasia is at no time an easy task, but it can be made immeasurably more difficult than it is in reality by approaching the examination of a case, and the analysis of the findings, in an improper or unmethodical way. Therefore, the first step in attempting the diagnosis is a simple method of eliciting and associating the different symptomatic constituents.

One who takes up the examination of a patient with aphasia should keep in mind that which has been said previously concerning the constitution of the speech faculty —that it consists of two parts, the receptive and the emissive, and that either of these two parts may manifest the predominance of the aphasic symptoms, but that in true aphasia, that is, aphasia dependent upon lesion of the speech centres, neither can be the sole medium of manifestation of the speech defects. It should further be remembered that emissive speech is manifest by articulation, by writing, and by pantomime, and that integrity of the receptive side of language is commensurate with the interpretation of visual and auditory stimuli.

It should not be forgotten that the attitude, the demeanor, the conduct, of the patient may be of the greatest service in orienting the physician, from the very beginning of the examination. Though recognition and inter-

pretation of information thus obtained is usually considered a quale of clinical insight, it is one of the cultivatable forms of this desirable qualification. The demeanor and expression of one with auditory aphasia are frequently those of a person who has lost all interest in his surroundings, and his attitude is that of a deaf person who is slightly demented. The same is true, though to a lesser degree, for the patient with visual aphasia. But the latter is more frequently of the restless, active kind, such as the case on page 265, who was so continuously moving and shifting that she gave the impression of one on the verge of acute mania. Moreover, patients who have this form of aphasia are often garrulous, and on the slightest provocation, or without provocation, emit a string of articulate or gibberish sounds that convey no meaning to those about them. This is especially true of cases of not very protracted duration. Patients with cortical motor aphasia and with subcortical motor aphasia, on the other hand, present a very different aspect. They are often absolutely silent but watchful, and the intensity with which they hold every move of the persons surrounding them is often very striking. This is well shown in the case of John Masterson (Case No. 2). His wife repeatedly said that the feature of his progressive recovery that impressed itself on her memory more than anything else was the assiduity with which he watched her every movement. This intentness of observation is particularly to be marked in cases of subcortical motor aphasia in which the patient is absolutely speechless yet capable of the fullest understanding of all that goes on about him and within his hearing and vision.

If these facts be kept in mind, the difficulties encoun-

tered by the beginner in examining a patient with aphasia will be reduced and the chances of reaching warrantable and legitimate conclusions as the result of the examination will be greatly increased. Before a case can be recorded in such a way that the symptomatic findings can be used legitimately in establishing, or demonstrating, certain claims, it is necessary that the examination be conducted in a manner that searches every centre which must functionate for the production of normal speech as well as their connections. To do this it should be systematic, but it need not be done in conformation to any special formula or according to any hard-and-fast system. A number of schemes have been devised to facilitate the examination of aphasic patients, but I have found the following simple plan most serviceable: After securing a general history of the patient's life and of his previous illness from some member of the family, and in this way getting information of the character of the disease of which the aphasia is a symptom, the patient's ability to express ideas, to receive and interpret information should be inquired into. The mental processes apart from the manifestation of mental states and the capacity for the reception of sensory stimuli should then be examined. Although a number of these may be determined simultaneously, it is best to take each one separately.

In approaching a patient with aphasia it is natural that the endeavor be made to elicit information by speaking to him. It becomes necessary, therefore, to determine if the patient takes note of what is said to him orally, and, secondly, if he understands what is said. In other words, does spoken speech awaken in his auditory centre corre-

sponding memories? This can be done ordinarily by asking some simple question, such as, "How long have you been sick?" or by addressing to him some simple command, such as, "Give me your hand." Care must be taken not to employ too conventional questions or commands, such as, "What is your name?" "Put out the tongue," etc. The patient may have lost the auditory apperceptive faculty and still, oftentimes, make reasonable reply to such questions, merely from association or habit. Naturally the patient should get no information of what is being asked through any other avenues than those of hearing. Such patients are quick to grasp, particularly if they have been aphasic for some time, the significance of even slight emotional expression or pantomime on the part of the interlocutor. If the patient does not reply to such questions or commands, there may be trouble with the receptive or with the emissive speech faculties. If he is word deaf, that is, if the trouble is one that prevents the sound of the word from reaching the centre in which the memories of previous word sounds are stored up, the patient will not endeavor to respond by word or act, though in some instances he does so. Nor will the face show the slightest response or indication of comprehension. If he does respond the diagnostic feature is that his answer, even though it be made up of articulate words, has no pertinency or bearing on the question. If the patient is not word deaf, he will make some movement, be it of the head, hand, or features, to indicate that though he understands he cannot reply. Generally this gesture is very significant. It consists of a despairing expression of the countenance and a touching of the lips or the throat with the

fingers. Oftentimes the question can be decided very quickly, if there remains some doubt even yet, by asking some absurd or ludicrous question, and noting how the patient receives it. If, in reply to the question, "Are you one hundred years old?" he solemnly says, "Yes," or if he does not see the ludicrousness of a request to turn a somersault when he is so obviously paralyzed, it is rather convincing proof that such speeches do not awaken the proper responses in his mind; and if there be no dementia it is suggestive evidence that the patient is word deaf, and the examination should then proceed from that standpoint. Although other of the speech centres may be simultaneously disorganized, the symptoms attributable to the first one will dominate the character of the speech defect. If the examination so far seems to suggest the existence of word deafness as the leading feature of the sensory aphasia, it should then be determined to what degree of completeness this exists, and the extent and kind of disturbance that it causes in the externalization of language. The amount of diminution of the patient's vocabulary, the degree of inappropriate usage of words, the imperfections of sequence and rhythm, should all be noted. The patient should be tested for his power of recognition of simple words, short sentences, and long sentences. It has already been noted that, perhaps, he may react to conventional questions, such as, "Put out the tongue," etc. Uncommon requests, such as, "Touch the nose with the tip of the index finger," or, "Stand on the chair," should be made. The ability of the patient to interpret sounds should then be noted. Do sounds evoke previous memories of similar sounds and do they incite

the auditory centre to revive the name of the object from which such sounds proceed? When a bell is sounded, or a watch is held behind the ear and apart from the stimulation of any perceptual avenue other than hearing, can the patient say, "Bell" or "Watch"? Finally, the existence of any disturbance of bone or aerial conductivity should be demonstrated or excluded.

If word deafness can be excluded, and the patient still makes no reply, that is, if he remains completely speechless, the examination should be to determine whether or not internal language is defective, for it must be readily seen that the question has then narrowed itself to a determination of whether or not the aphasia is cortical motor (kinæsthetic word-image) aphasia, or whether it is subcortical motor aphasia. In other words, is the inability to speak due to a lesion of the storehouse of kinæsthetic memories of articulated words, Broca's area, or is it due to a lesion of the neurons that conduct the motor word impulses from the Rolandic area to the parts that externalize the word? The essential thing then is to determine if the patient is in the full possession of internal language. If internal language in any of its components is disordered, then the patient has true cortical motor aphasia. If, on the other hand, there be no such disturbance, the lesion is elsewhere than in the zone of language. In some patients the differentiation will be an easy one. They will show, as did Case No. 1, that they have the proper idea of words and that they can evocate them promptly, by the ease and rapidity with which they write, or by the exquisiteness of pantomime, as did the patient just referred to. On the other hand, however, the task is

oftentimes an extremely difficult one. It is particularly so because the test to determine if the legitimate idea of words can be evoked in the internal language, the test of Proust and of Lichtheim, is not one of universal application, because in the first place many of our hospital and dispensary patients are not of sufficient scholarship to know anything of syllables, or of counting the number of letters forming them, and word construction is an exercise that has never been indulged in. In the second place, there is very often associated with aphasia, and a concomitant of the disease giving rise to the latter, a degree of deficiency in the associative faculties that amounts to a slight degree of dementia. In such patients it is oftentimes extremely difficult to make them understand just what is meant by telling them to press the physician's hand as many times as there are syllables in the word Constantinople, or some other equally resonant and polysyllabic word. Nor is the substitute suggested by Dejerine, of asking the patient to make voluntary expiratory efforts as many times as there are syllables or letters in a word more applicable. But even when we cannot get the patient to respond to these tests, there is a general atmosphere about the patient with subcortical motor aphasia that one cannot be long in without recognizing that the patient is in full possession of his intellect and internal speech. The only shortcoming of the subcortical motor aphasic is inability to articulate. He understands everything that is said to him; he interprets information received through the visual sphere; he is capable of expressing his thoughts fully, facilely, and correctly, by writing and by pantomime, or, at least, he would be were it not that the right half of the body is

usually paralyzed and he is obliged to portray mental states by the pantomimic activity of the left, the less dextrous half of the body.

Physicians oftentimes find some difficulty in properly assigning cases of cortical motor (articulatory kinæsthetic) aphasia, because the patient is still able to articulate some words. I have often been made aware of this by conversation with my house physicians, who work apparently with the following formula: "If the patient can think of the word and is unable to say it, he has motor aphasia; but if he cannot think of the word, though he is able to say it, then he has sensory aphasia." If one had to choose between this formula, and nothing at all, it might be well to choose the formula, although it is only half the truth. If it be kept in mind that the patient with cortical motor aphasia (articulatory kinæsthetic) need not be absolutely deprived of the power to articulate words; that he frequently retains the ability to say one or several words, which he uses at all times and under all conditions, pertinent and impertinent alike; and that frequently these words take the form of recurring utterances; that there is always agraphia, which may be very evident or which may be difficult to bring out because the patient pleads paralysis of the right hand as an excuse for not making an effort to write; that the agraphia is usually proportionate to the aphasia; that it is manifest in voluntary writing and in writing from dictation, but not in writing from copy; and that the patient in copying copies print in script and script in script, showing that the copying is not a mechanical but an intellectual act; and that there is defective internal speech, as shown by the test of Proust and Licht-

heim — then the diagnosis of articulatory kinæsthetic aphasia will not be a difficult matter.

After voluntary speech has been satisfactorily examined, tests should be made to determine the patient's capacity to repeat. There is inability to repeat in both sensory and motor aphasia, and if word deafness has been excluded there will be no difficulty in interpreting this inability which is co-existent with loss of voluntary speech in articulatory kinæsthetic aphasia.

Particular attention should be given, in every case of aphasia in which the symptoms point to destruction of Broca's area, to the faculty of writing. Following Dejerine, it has been maintained, and I venture to hope consistently, that lesion of this area causes agraphia. Recently Bastian has reiterated the statement that the agraphia that sometimes accompanies articulatory kinæsthetic aphasia is not dependent upon lesion in Broca's area which prevents the patient from getting the correct notion of the word. He contends that it is an occasional phenomenon only, and when it occurs it is due (like alexia) to temporary or more or less permanent disablement of the visual centre. I readily admit that in some instances this does occur, but this in no way invalidates the explanation of the occurrence of agraphia, which, I believe, occurred in every case of cortical motor aphasia that has been given. The fact that such contradictory beliefs are held as to the occurrence of agraphia with motor aphasia demands the very careful examination of such cases in the future.

Of course, when a patient who has had articulatory kinæsthetic aphasia has partially recovered and has regained

quite an extensive vocabulary, it will require care and repeated examinations satisfactorily to establish the diagnosis. The one suggestion that I have to make in such cases is that there will always be found some degree of every one of the symptoms enumerated as occurring with this form of aphasia, if sufficiently careful and patient search be made for them, and if the physician is trained to recognize these slight defects they are of great service in orienting him.

After having tested the patient's capacity to perceive and interpret words through the auditory apparatus, he should be examined with the view of determining if there is any disability of acquiring and interpreting information through the visual apparatus. To do this requires patience and circumspection. In the first place it should be established that the patient has no trouble with the peripheral visual apparatus. This can be done by an ophthalmoscopic examination. Tests should then be made to determine the existence of hemianopsia. This is not an easy matter to do if the patient is aphemic or if he has word deafness; in fact, it is extremely difficult to do satisfactorily. With a patient who can understand what is said to him and who can indicate when he perceives the entrance of an object into the visual field, who can tell when the indicator of a perimeter passes beyond the range of vision, testing for hemianopsia is a very simple matter. If the patient is word deaf and if he has visual blindness, which of course he is apt to have if he has hemianopsia, one finds himself unable to convey to the patient by written or spoken word that which he wishes him to do or to observe. In such cases one must content himself with the

information that is to be derived from forcibly and suddenly thrusting some object into the visual fields, from the right side (for right-handed patients invariably have right lateral homonymous hemianopsia when they have any), and taking note whether or not the patient blinks, as he should do if the object be perceived. If he does not it is rather certain that he has hemianopsia. Each eye should be examined separately and the findings noted on a chart. The form of hemianopsia that may be found will be readily interpreted if one has clearly in mind the course of the optic nerve and the tract to the cortex (see Fig. 6). A destructive lesion of the cortex in the vicinity of one calcarine fissure, or of all the optic fibres leading to it, the radiations of Gratiolet, produces blindness on the opposite sides of the visual field. If the left one is destroyed there will be right hemianopsia, and as the hemianopsia is on relatively the same sides of the visual field, that is the right temporal and the right nasal, the hemianopsia is called homonymous. If the right cuneus is destroyed just the same condition will prevail, only it will be manifested on the other side; and if both cunei are destroyed there will be true cortical blindness. If these facts be kept in mind there can be no difficulty in properly interpreting the occurrence of hemianopsia. If there is destruction of the cortex in the vicinity of the calcarine fissure, that is, destruction of a half-vision centre, there will be lateral homonymous hemianopsia, but not word blindness, as the higher visual centre is still in intact connection with the half-vision centre of the other cuneus. If there is lesion of the bands of Gratiolet connecting both half-vision centres with the primary visual centre and one

cuneus with homonymous parts of both visual fields, then there will be hemianopsia and word blindness, complete alexia, merely because the patient cannot send impulses coming in from both half-vision centres to the angular gyrus where they are interpreted, although the latter is intact and shows this intactness by the undefectiveness of internal language and the ability to write spontaneously and from dictation. Such a patient copies mechanically, print in print, script in script, as one traces a drawing. This, and one other fragment of knowledge concerning hemianopsia, are all that is necessary properly to interpret its occurrence. Sometimes a lesion that destroys the angular gyrus extends sufficiently deep to sever the optic radiations of Gratiolet, which are immediately subjacent on their way to the occipital lobes; in such cases there will be true word blindness with all its entailment of disturbance of internal language, disturbance of intellection, agraphia, etc., plus hemianopsia, but the latter symptom is merely an accident, a superadded phenomenon, and it never occurs with destruction of the angular gyrus, except in some such way as I have indicated.

In testing the patient to determine the integrity of the visual mechanism one may begin by showing him familiar objects. If he does not recognize them, or show by act or deed that he comprehends their use or purpose, if he looks upon them as does one who sees them for the first time, then he has object aphasia and the lesion is of the occipital cortex. Such an individual may obtain information through the medium of other special senses, such as the tactile, gustatory, etc., that will enable him to recognize the object, the person, or the thing. If he is shown

familiar objects and he recognizes them, knows what they are for, but cannot name them, then he may have either an interruption in the pathway leading to the higher visual centre in the angular gyrus, or there may be lesion of the angular gyrus itself. If it be of the former, internal language will be preserved and spontaneous speech may be intact, although there is usually some paraphasia and possibly jargonaphasia, and this preservation is shown most conclusively by the retention of ability to write. He may write easily and moderately well, not only voluntarily but from dictation, but the patient is unable to read what he writes. If it be of the latter and complete, the patient will be absolutely agraphic. This agraphia is to be considered a part of the disorder of internal language, loss of the visual image, the visual idea of the word. There is inability to arouse the visual image of the word. In such a case, as arousal must precede the transmission to the part of the Rolandic cortex that innervates the member holding the pen, there is complete agraphia. There is no more strikingly illustrative case of this on record than one communicated to the Royal Medical and Chirurgical Society in 1872, which was one of the first, if not the first, in which subcortical word blindness was the striking symptom. Although this case was used by Broadbent then, and is still to-day, to bear evidence in favor of a naming centre, it must be apparent to him who interprets the genesis of speech in the light of our present knowledge that in reality it is a most recalcitrant witness in behalf of Broadbent's claim. The history of the case, if space permitted us to quote it in detail, would be very instructive to show the typical picture of subcor-

tical sensory aphasia. "The patient after an acute cerebral attack (?) showed absolute inability to read printed or written words (except his name), while he wrote correctly from dictation and spontaneously." There was inability to recall the name of the most familiar object presented to his sight, while he conversed intelligently, employing an extensive and varied vocabulary, making few mistakes, but occasionally forgetting names of streets, persons, and objects. There is no note of whether or not hemianopsia existed, but it may be taken for granted, I think, that it did, for in every published case, without exception, in which this condition was examined for it has been found. In regard to Broadbent's statement that it bears evidence in favor of a naming centre, it need only be said that the case shows that there was a lesion that severed the visual percipient centre from the visual interpreting centre, and if he prefers to call the latter a naming centre no adequate reason has ever been offered why he does so.

One then proceeds to examine to see if the patient has word blindness; that is, can the patient read (1) print, (2) script, (3) figures and other forms of notation. I have already described the significance of inability to do all or one of these. It can need no repetition to show that inability to read is not of itself an important localizing symptom; it may be produced by lesion in many parts of the optic projection and of the parts that the optic projection goes to. It is the association of the inability to read that facilitates localizing the lesion. If, for instance, a person's primary visual centres are intact, he sees the written or printed word, the figure or symbol, in all its details; it merely has no significance for him. This bears

testimony that the lesion is centralward to the primary visual area. He can decide at once whether it be far enough centralward to be in the angular gyrus, the higher visual centre, or not, by determining if there be disturbance of internal language. If there is not (and as a matter of fact one may say if there be no agraphia the lesion is not in the zone of language), then the word blindness is the result of a subcortical lesion. One word of caution must be sounded for the inexperienced in determining visual blindness. Some patients who have word blindness, particularly those in whom the symptom is dependent upon lesion of the higher visual centre, on being asked to read, take up a book or paper, or whatever is handed to them, and essay to read it understandingly. If they are made to read it aloud it will quickly be seen that they cannot read a word, that they "make up," as children do, as they go along. Oftentimes the assurance with which they take hold of a paper or letter and apparently read it deceives even an experienced examiner. It cannot do so, however, if the physician will show them writing or print embodying a request set forth in such a way that cannot escape their recognition if they read it, such as: "If you are able to read this then put your left hand in mine." Another test often employed is for the physician to read aloud the last lines of a page and request the patient to turn the page at the proper time. Naturally, he is unable to do so, as he cannot read, although if he is not at all word deaf, that is, if he can appreciate the quantity and amount read, he may very closely approximate the time when he should turn.

In cases of complete aphasia the examination is very

difficult and to one not accustomed to such a task it seems very unsatisfactory, as he is apparently unable to communicate with the patient or receive any information from him. Oftentimes it is thought that such patients have not recovered consciousness completely. I have such a patient at the present time under observation: A married man, fifty-six years old, of good habits, was seized one day with a feeling of numbness and beginning powerlessness in the right leg, which after a few minutes showed itself in the right upper extremity. In less than a quarter of an hour he became unconscious and completely hemiplegic. There had been no premonitory symptoms save recurring headache, which, his wife now recalls, had been complained of for several weeks previous to the apoplexy. On the third day after the attack signs of returning consciousness began to show themselves, and at the end of a week there was apparently complete restoration of consciousness. The hemiplegia continued complete, the face being least severely involved.

The following notes of the examination show the completeness of the aphasia in this case:

What is your name? No response; looks at me blankly and staringly.

Is your name ——? No reply, slight shifting or restlessness of the patient.

Are you seventy years old? No reply.

Would you like to go home with your wife? No reply, no interest.

Then these same questions were addressed to him in writing and in print, but he took no more heed of them when endeavor was made to bring them to his notice in this way than when they were spoken. It was impossible

to establish with any degree of certainty the existence of hemianopsia. The impression, as the result of repeated and careful testing, was that it did not exist. There was apparently no object, no mind blindness. The patient took a piece of candy that his wife brought, carried it to the mouth, and ate it with apparent relish. When given a pencil he grasped it and handled it in a familiar way, likewise a watch, a key, and other common objects. He recognized members of his family, and when his wife came to see him he would grasp her hand and carry it to his lips. He would try to detain her from leaving, and manifested appreciation of visits.

Persistent efforts to get him to write a word are wasted. He grasps the pencil as if he were about to write, but instead of proceeding to form a letter he scratches the paper, just as an infant does who is given a pencil for the first time. If I guide his left hand which holds the pencil to outline his name, or the names of familiar objects, and then show them to him, he does not understand the one or recognize the other. If I guide his hand over the letters of a word to trace it, he may trace the next word alone, but he does it mechanically and takes not the slightest interest in it. When he is given a number of bills of different denominations, it is impossible to say that he understands the difference in value of them, for he takes with the same readiness a twenty-dollar bill and a two-dollar bill. He does not utter a word or the semblance of a word. He does not indulge in any form of pantomime or mimetic action. There seems to be absolutely no way of communicating information to him or of receiving wishes or desires from him. In other words, he has total aphasia. In another connection, it may be recalled, it was stated that sometimes such cases of aphasia clear up on one side of language, the receptive or

the emissive, and the residue of the aphasic symptom complex constitutes sensory or motor aphasia.

In this patient it would seem that the middle cerebral artery had ruptured and that the speech area has suffered so severely that restitution will not follow, as sufficient time has already elapsed to have it show itself if the vital forces were sufficient to do so. The fact that he has an advanced degree of chronic interstitial nephritis, and the fact that, despite most careful dietetic and vigorous medicinal treatment, he is continually emaciating, augurs ill for him.

Sufficient has already been said of dyslexia in the chapter on "Sensory Aphasia" to make reference to it here, as a factor in diagnosis, unnecessary. I am disposed only to reiterate, particularly since reading the first of Bastian's recent lectures on problems in aphasia and other speech defects, which appeared after this monograph was written, the necessity of the most painstaking examination to reveal latent defects of internal reading in every case of aphasia. After the condition of the functional state of the different speech centres and the immediate conducting tracts leading to and away from them have been inquired into, and a general survey has been taken of the patient, two things remain to be done; namely, an examination of the patient's capacity to externalize mental contents by pantomime or mimicry, and a study of the manifestations of emotion in melody, instrumental music, profanity, interjections, and gestures. It is believed that sufficient has already been said of these to make reconsideration of them unnecessary.

It is necessary to say one word concerning the time of

examination of patients with aphasia. The complexity of symptoms that may be determined one or two weeks after the restoration of consciousness following the apoplectic insult, or the confusion and delirium which may be the introductory symptoms of the aphasia, may be quite different from that found when the examination is made later. The morbid vascular changes that go on secondarily to the lesion, whatever it may be, will subside in part after the acute manifestations of the disease or accident have disappeared, and if they have not been of sufficient severity to cause destruction of the parts the area that was for a time obscured will again functionate with more or less integrity. It is well, therefore, to compare the results of the clinical status made early with those made after the symptoms have continued for a time, and thus to establish the permanent degree as well as the kind of aphasia.

CHAPTER IX.

Etiology.

Before enumerating the individual organic diseases or functional conditions of which the different varieties of aphasia may be a symptom, I wish to direct attention very

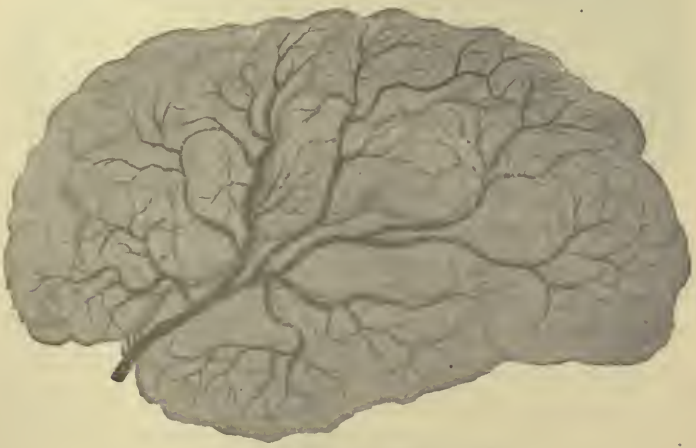

Fig. 19.—Vascular Supply of Cortex.

briefly to the boundaries and blood supply of the zone of language, which contains the centres of auditory, visual, and articulatory (kinæsthetic) images. The locations of these centres in this zone of language are now definitely assigned. Their relative position on the surface of the brain is shown in Fig. 7, page 99. Their boundaries and interrelations are considered in Chapter IV., "Conception of Aphasia."

A comprehensive knowledge of the vascular supply to this portion of the brain is necessary for a full understanding of the mode of development and the lesions of aphasia. Such knowledge can best be obtained by a study

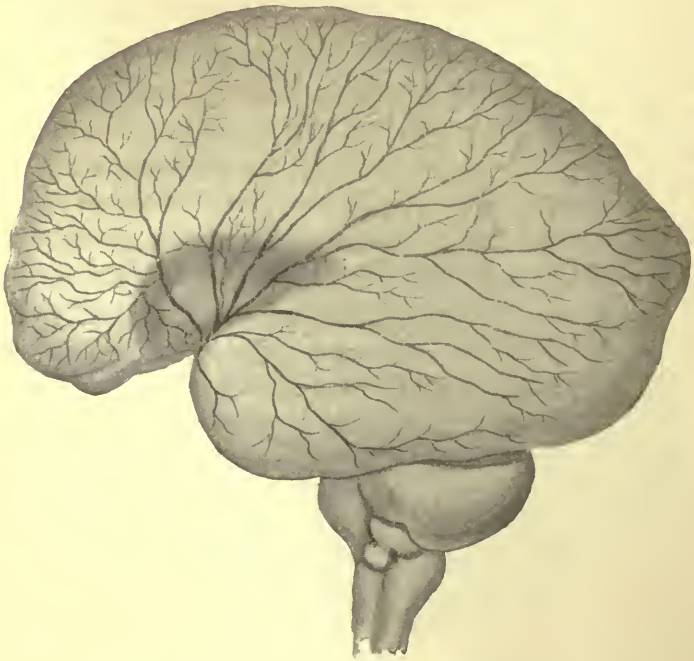

FIG. 20.—Cortical Blood-Vessels of Fœtal Brain.

of the fresh brain. The accompanying illustration (Fig. 19), taken from the well-known work of Duret, will convey an idea of the distribution of the blood-vessels to the external surface of the brain. It shows the middle cerebral artery, which supplies the side of the cortex in all its ramifications. It will be seen that this one artery supplies through its trunk, its principal branch, and its terminals

the zone of language and the three centres lying therein. Fig. 20 shows the cortical vessels of the fœtal brain particularly in their relation to the fossa of Sylvius. When one considers the importance of the middle cerebral artery to the zone of language, it is no longer surprising that we do not oftener encounter cases of aphasia whose symptoms can be interpreted as due to lesion of an individual centre. The middle cerebral artery, the largest and most important branch of the internal carotid, in fact, the continuation of the latter, supplies other parts than the zone of language, and enumeration of these parts facilitates interpretation of the accompanying symptoms in many cases of aphasia. The antero-lateral arteries which are given off from the middle cerebral immediately after leaving the carotid artery pass through the foramina of the anterior perforated space to the base of the corpus striatum and form the most important supply of that region. The lenticular branch supplies the inner and middle segments of the lenticular nucleus and the internal capsule. The lenticulo-striate arteries supply the outer segment of the lenticular nucleus and external capsule and the caudate nucleus, and the lenticular-optic arteries supply the outer part of the optic thalamus. The vessel then passes to the cortex.

The cortical branches of the middle cerebral artery, or the Sylvian artery, the continuation of the main trunk of the internal carotid, are the interior external frontal, distributed to the outer part of the orbital surface of the hemisphere and adjacent frontal convolutions; the ascending frontal, distributed to the convolution of the same name and to the root of the middle frontal convolution; the ascending parietal to the parietal convolution and to

the forepart of the superior parietal lobule; and the parieto-temporal, which runs backward in the posterior limb of the fissure of Sylvius and ramifies upward over the angular gyrus and downward over the superior and upper part of the middle temporal convolution.

Contrasting the distribution of this artery on the cortex with the relative position of the different speech centres, it will be seen that a lesion of the trunk at its entrance into the fossa of Sylvius, and particularly a lesion of the first branch, will be very apt to destroy Broca's convolution, in which are stored the kinæsthetic memories of articulation. If the lesion is not of sufficient severity to destroy the centre it may materially pervert its functions, and this perversion may be transient or lasting. Simultaneously with the occurrence of such a lesion the integrity of the circulation in the posterior parts of the vessel, in the terminal branches, the one bending up over the angular gyrus and the other down over the temporal convolution, may be disturbed, though to a very insignificant degree compared with the disturbance of Broca's centre, but yet sufficient to add a sensory element to the aphasic symptom complex. Thus, in the beginning of some cases of aphasia, the symptoms may indicate a mixed form, but the slight sensory or motor element, as the case may be, may disappear, leaving the other dominant to constitute the form of aphasia. It is not in place here to point out that the aphasic symptoms vary not alone with the seat of the lesion, but with the intensity of the lesion, the rapidity of its progress and development, or, in other words, with its nature. This may be inferred from what has just been said.

With the exception of destruction of the speech areas that are the result of injury and new growth, organic disease of the zone of language is almost always the result of vascular lesion. These vascular lesions are rupture of the blood-vessels and occlusion of their calibres, whether from embolus or thrombus, and the consecutive changes dependent thereon. The lesions of the blood-vessels may, however, be due to inflammatory conditions of the vessels, but even then it is not at all improbable that the pathogenesis of the lesion is the direct result of a septic or infectious process that causes infectious emboli and thrombi. The traumatic conditions that may produce aphasia are bullet and stab wounds, depressed fractures of the skull, and injuries producing meningeal hemorrhage.

Etiologically, aphasia may be classified into organic and dynamic. The principal organic forms have just been enumerated. Under the dynamic forms may be included those in which no organic lesion is responsible for the development of aphasic symptoms. The term dynamic is used merely as a convenience in preference to the conventional "functional." The dynamic variety includes aphasia occurring with neuroses and psychoses which are not yet proven to be dependent upon recognizable brain lesion, of which epilepsy, neurasthenia, and hysteria may be taken as examples. It also embraces most of the cases of aphasia occurring with toxihæmia, such as uræmia, diabetes, and gout; although aphasia in some of these cases, especially aphasia occurring with uræmia, is often dependent upon organic vascular lesion of the cerebral blood-vessels. Aphasia caused by the vegetable poisons, santonin, belladonna, tobacco, etc., is almost invariably of the dynamic

form. The aphasia that sometimes occurs in individuals who have been poisoned by lead, copper, etc., may be of the dynamic variety, or it may be a focal manifestation of the encephalopathy that these poisons occasionally cause. The dynamic aphasias also include the aphasic speech disturbances occurring with neuralgic affections of a migrainous order, those occurring with forms of insanity that have no known anatomical basis, and, finally, the comparatively insignificant number which are attributed to fright, anger, so-called reflex causes, such as intestinal worms, and the transitory aphasias from loss of blood.

Ordinary etiological factors, such as age, sex, occupation, etc., have no bearing on the causation of aphasia, because it is itself a symptom, and it results only when the diseases of which it is a symptom occur or are prone to occur; but as aphasia is so often associated with cerebral apoplexy, and as cerebral apoplexy occurs usually in late maturity and advanced age, it follows that aphasia is seen oftener in people beyond fifty years of age. Nevertheless, it would be misleading to leave this statement unmodified, for the reason that three diseases which not infrequently have aphasia as a symptom, namely, uræmia, acute hemorrhagic encephalitis, and tuberculous meningitis, are particularly liable to occur in the young. Moreover, aphasia sometimes develops in the wake of the infectious diseases, typhoid, diphtheria, and pertussis, and, as these occur more frequently in youth than at any other time, it follows that the aphasias of this variety will be seen oftenest at this time of life.

It is my intention to enumerate the more prominent diseases of which aphasia is an important symptom, and

to dwell upon a few of these only. In addition to those already mentioned, it may be said that aphasia of any kind may be referable to some such disease of the skull as exostosis and bony tumor; in short, to any condition of the skull bones or the meninges, such as pachymeningitis, that produces pressure on or irritation of the speech area. It is an occasional symptom of thrombosis of the dural sinuses, particularly thrombosis of the lateral sinuses.

Aphasia is one of the most important and constant symptoms of acute non-purulent encephalitis. A case recently reported by Leva[1] is a good illustrative example. The patient had pronounced sensory aphasia, total alexia, and agraphia, but no articulatory aphasia. At the autopsy there was found diffuse encephalitis, with softening in the first and second insular gyri of the left hemisphere, in the adjacent convolutions of the inferior hemispherical convolutions, and some disintegration of the first temporal gyrus adjacent to the insula. In the right hemisphere similar areas of softening were found in the right temporal gyrus.

Depending upon the locality of the abscess, a variety of aphasia is frequently an important symptom of purulent circumscribed encephalitis, and especially the form complicating purulent disease of the middle ear. In fact, in cases of brain abscess it is oftentimes a localizing symptom of most exquisite value. This is well illustrated by a case recently published by Zaufal:[2]

[1] Leva: Virchow's Archiv, vol. cxxxii., part ii.

[2] Zaufal and Pick: "Otitischer Gehirnabscess im linken Temporallappen, optische Aphasie, Eröffnung durch Trepanation. Heilung." Prager med. Wochenschr., Nos. 5, 6, 8, 9, 1897.

A young woman, twenty-five years old, complained of headache, pain in the left ear, nausea, and vomiting. Eight days later there was a discharge of pus from the left ear. Examination revealed acute suppuration of the middle ear, inequality of the pupils, progressive stupor, and vomiting. Shortly after this, the patient was unable to name objects shown to her, although she knew them well and could describe their appearance; that is, there was optic aphasia, pointing to a lesion severing the connections of the primary visual area with the area of word memories; in other words, a pure, or subcortical visual aphasia. The lesion was localized by Pick, and an operation revealed an abscess of the size of a hen's egg in the region indicated, namely, in the left second and third temporal convolutions far back and in the white substance. The patient made a complete recovery. In this case it is to be noted that there was no hemianopsia, which showed that the primary visual area around the calcarine fissure, as well as the radiations of Gratiolet were not affected.

Another most instructive instance of the same kind, but in which the outcome was not so gratifying, is that of a case related by Lannois and Jaobulay.[1] In this case there were, in addition to symptoms of ear disease and brain abscess, alexia, agraphia, word blindness, right-side hemianopsia, and slight facial paralysis. The patient was operated on but no pus was found. A second operation was made three weeks later, and a collection of pus was evacuated. Eleven days later the patient died, and on autopsical examination a large abscess was found in the centre of the left occipital lobe. There were also a number of foci of diffuse encephalitis, some of them purulent, proba-

[1] Lannois and Jaboulay: Revue de Médecine, August, 1896, p. 659.

bly metastatic, in other parts of the left hemisphere. In this instance, the symptoms were of such absolute localizing significance, pointing to destruction of the left occipital lobe and of the radiations of Gratiolet, that it would have been fully justifiable to lay open that part of the brain instead of endeavoring to draw off the pus by puncture.

Of the other intracranial inflammatory conditions, that of meningitis of the convexities and meningeal tuberculosis are the two diseases sometimes accompanied by aphasia. In purulent leptomeningitis secondary to optic disease aphasia often develops, and, were it not for the teachings of Hugenin, it is probable that aphasia would not be considered of infrequent occurrence in tuberculous meningitis. To show the extent and complexity of the aphasic symptoms in tuberculous meningitis, I need only refer to a case communicated by Carrière,[1] in which a tuberculous meningitis (the lesion predominating in the posterior part of the fissure of Sylvius) was accompanied by verbal blindness, ageusia, anosmia, and, finally, total blindness. This case is interesting also for the reason that a spot of recent softening was found in the hippocampal gyrus. The anosmia was probably dependent upon this lesion, as this would tend to corroborate the claims of Jackson, Beevor, Carbonieri, *et al.*, who place the cortical centre of smell in the hippocampal gyrus.

Aphasia may occur as a symptom of direct injury to the brain, and in times of war and riot such cases are of common occurrence. A remarkable instance of destruction of

[1] Carrière, G.: Archives Cliniques de Bordeaux, 1896, p. 135.

Broca's convolution by a foreign body is shown by a case of Simon.[1] The patient had been injured by a fall from a horse. After death a splinter of bone, which had been detached from the inner table of the skull, was found in the left third frontal convolution. Aside from the cases in which there is solution of continuity of the skull, it may result from injury to the head, such as from a blow, a fall, a kick, unaccompanied by fracture of the skull. Whether or not the lesion in some of these cases is a dynamic one cannot be said. Usually the aphasic symptoms in such cases are neither so complete nor so continuous as is aphasia depending upon vascular lesion. This is shown by an instance published by Cameron.[2] A young man fell a distance of fifteen feet and struck on the head. He was unconscious for a time and later very restless. On the seventh day after the accident he had a series of mild convulsions, manifest on both sides of the body, but with special involvement of the mouth and eyes. After this the stupor became greater, but from the eighteenth day there was a gradual clearing up of consciousness. On the nineteenth day he was able to say "Yes" and "No." On the twentieth day there were aphasia, alexia, and agraphia. These symptoms disappeared gradually, and about six weeks after the accident the patient was quite well. In a case of this kind, it is much more likely that there was no considerable organic lesion, and that the anatomical condition may be compared to that which is supposed to exist in some cases of traumatic neurosis.

[1] Simon: "A Case of Aphasia." Johns Hopkins Hospital Bulletin, Baltimore, 1889–90, i., p. 48.

[2] Cameron: "Notes of a Case of Traumatic Aphasia." Glasgow Medical Journal, August, 1896, p. 126.

Therefore the aphasia is properly considered to be of the dynamic variety.

The dynamic aphasias may be dependent on functional disorders of the brain, that term being used in its widest sense to cover not alone states of nervous exhaustion, but to include such diseases as epilepsy, hysteria, and migraine. They, as well as the aphasias of toxihæmia, are characterized by the variability of their manifestations, the intermittency of their course, the transitoriness of their duration, but especially by their favorable outcome. The form of aphasia that occurs with the neuroses varies, and we are not in position to state the conditions governing such variation. In some instances, the aphasia will be typical articulatory kinæsthetic aphasia, while in others the sensory elements will predominate. The most common symptom of the aphasic speech disturbance attending migraine would seem to be paraphasia and inability to name objects. Not long ago, a hard-working physician, about forty years of age, who had been for a time under my treatment for epilepsy, developed under the auspices of an acute infectious influenza a profound status epilepticus, which lasted nearly sixty hours. He then gradually recovered consciousness, but for the next five days had aphasic symptoms characterized particularly by loss of articulatory motor memories, as he has since then decided with me. After he recovered consciousness he was wholly unable to speak, although there was no word deafness, no word blindness, nor was there the slightest trace of disturbance of motility in any part of the body. He was at this time agraphic, although able to copy. I labored with him for some time to get him to pronounce the simple words "Yes" or "No"

in response to questions which he apparently understood; showed him how to say them by fixing his lips and by example, but all to no purpose. He regained ability to speak fluently and correctly within a week.

Another case of epilepsy, but in which the aphasic symptoms were the result of bromide intoxication, has been for me an instructive example. The patient is a young woman of social position, who desires above everything else not only to remain free from the convulsive attacks of her infirmity but to keep her friends ignorant of it, and to do this she is willing to take very large doses of bromide. Occasionally if hydriatic and other restorative procedures necessary to keep up the tone of the nervous system are at all neglected, she develops symptoms of sensory aphasia, characterized especially by verbal amnesia and mild degrees of word blindness and object blindness. I have had her maid make notes on numerous occasions of the mistakes of utterance that are noticed at these times, and I quote here a few of them. Wishing to say, "I am going to my room," she said, "I thick, think, that's the stick thick." For "I am going down stairs," she said, "I am going down town." When she desired the maid to hand her a cup, she said, "Will you give me that window;" and at table, desirous of living up to the mandates of her physician, in refusing dessert she said, "I do not care for interest." She says that oftentimes she finds herself conning a printed page or a letter trying to make out what it means, to decipher the words, and then, all at once, after looking at them for a time, it will quickly dawn upon her that they are symbols, letters, and words with which she is familiar. It has also been noted

that at such times, on preparing to go for a walk or a drive, she will insist that she cannot go without her hat, while all the time the hat rests on her head and she may be apparently looking at it in a mirror. There is no dementia or trace of insanity; her conduct and her actions are in keeping with her breeding; and when the bromides are diminished in amount and restorative measures applied vigorously the aphasic symptoms disappear.

The genesis of aphasia occurring with epilepsy is not an easy matter to interpret, but it should not be forgotten that some cases of epilepsy of which it is a symptom are dependent upon organic disease, such as a tumor, and vascular lesion, and in every case of epilepsy in which aphasia occurs a very careful examination is demanded. A case recently reported by Hay prompts me to this statement. A man, thirty-nine years old, free from syphilis, suffered three attacks of influenza in rapid succession, and immediately afterward complained of great weakness, headache, depression, which brought him to a very distracted state. Shortly afterward a condition of status epilepticus developed, and when this terminated he had aphasia of a sensory type and agraphia. Six months after the beginning of the symptoms status epilepticus developed a second time, and from this he did not recover. The autopsy showed a spot of softening in the left temporal convolution.

It does not need the recitation of individual examples to show that aphasia in some of its forms, and particularly sensory aphasia, characterized by verbal amnesia and paraphasia, is a common attendant upon states of mental exhaustion, especially when associated with physical fatigue,

and upon preoccupation. Almost every one who has been overcome by the former is in possession of a personal example. Naturally I do not mean to say that preoccupation produces aphasia. What I mean is that a person's cognitive areas may be so intent upon subjects that engross him that the zone of language is temporarily ungeared. A very good illustration of this is related of Emerson. It is well known that this immortal transcendentalist was wont to accompany himself with the traditional New England umbrella in his walks. On occasions when more engrossed in absorbing thought than usual, he would hesitate on going out, search various corners, the hatrack, etc., where the cotton rain guard was usually to be found, and not finding it he would stand, solemnly tap the temple or the brow, and say, half to himself and half to a bystander, if there happened to be one, "Oh, where—is—, where—is,—where—is, my, oh—where is that thing that honest people take or borrow and never think it necessary to return?" When the word umbrella was suggested, "Yes, my umbrella." Although this has been utilized as a contribution to "Umbrella Pleasantries," it reminds one of the patient described by Trousseau, who, desiring the same article, was wont to say, "Where is my—u—u—u—*sacré matin!*" "Your umbrella?" "Yes, my umbrella."

Genuine aphasia is a very rare accompaniment of hysteria, as rare correspondingly as mutism is frequent. Why this should be so, I am at loss to understand. It appears to me that the most reasonable interpretation of most, if not all, hysterical phenomena is one that posits the partial abolition or perversion of function of one or more of the cortical areas. When the involved cortical

area is the somæsthetic or Rolandic area, there result perversions of sensibility and motility; when of the primary visual areas, we have hysterical blindness; when of the auditory area, we have hysterical deafness; and when of the frontal lobes, we have diverse and protean psychical manifestations. Thus it would seem to me that a dynamic perversion of the zone of language analogous to the condition that forms the basis of the above-mentioned phenomena might produce aphasia; but as a matter of fact there are very few examples of hysterical aphasia on record.

Hysterical aphasia is usually less transitory than are other forms of dynamic aphasia, but, unlike hysterical aphonia, it does not extend over months and years. It may be the only major symptom of hysteria or it may be associated with hysterical hemiplegia and contracture or with contracture in other parts of the body. It is more frequently seen accompanying traumatic hysteria, than with the so-called idiopathic form. Aphasia analogous to the hysterical form has been artificially produced in persons by hypnotism.

In studying the aphasic speech disturbances which sometimes accompany neuralgic affections of the migrainous order, it is well to keep in mind the genetic relationship existing between migraine and epilepsy. Usually the aphasic disturbances of migraine are of a sensory character and very transitory. In discussing the relation of amusia to aphasia, I have cited one or two examples of the aphasia of migraine, and shall here refer only to one example recently published by Pick.[1] A young physician

[1] Pick: Berliner klinische Wochenschrift, 1894, No. 47.

who had lived a very irregular life developed suddenly symptoms of ophthalmic migraine, with which were associated motor aphasia, word deafness, and echolalia, all of which disappeared with the attack.

It has been suggested that the genesis of migrainous aphasia is in reality a vascular one, a contraction of the cortical blood-vessels of the left hemisphere, which causes a perversion of function of the zone of language. This is a very plausible supposition, considering the very striking evidences of vasomotor instability in other parts of the body that occur with migraine, but it is easily understood that there are insurmountable difficulties in the way of adducing proof of this hypothesis.

There is an amount of very convincing evidence on record to show that certain drugs, such as santonin, belladonna, tobacco, etc., of the vegetable poisons, sometimes produce aphasia when given in toxic doses. There is nothing especially characteristic of such aphasia to aid us in differentiating it from other dynamic forms, and the aphasia can be suspected to be of such origin only when it is known that the patient has exposed himself to one of these poisons. If the cause can be discovered and removed, the aphasic symptoms soon disappear, and this, more than anything else, stamps their origin.

This leads me to speak of other toxic conditions that may cause aphasia, particularly uræmia, diabetes, and gout, as well as more uncommon forms of poisoning, such as snake bite, etc., and of the aphasia which sometimes occurs with Raynaud's disease. Aphasia is an extremely uncommon complication or coincident symptom of Raynaud's disease, symmetrical gangrene. I have been able

to find but two references to it in the literature, one by Weiss;[1] another by Osler.[2] Personally I have seen one case. A young man was brought into the hospital suffering from symmetrical gangrene of both feet, slight cyanosis of the tip of the nose and the upper part of both ears, and with the general-collapse symptoms attending the advanced stage of this disease. He was thought to be demented or amented by some members of the house staff who saw him, because it was impossible to extract any information from him. He could not tell his name, age, residence, occupation, or anything about his illness, neither could he be got to write or read. Ability to articulate words was preserved, for he would occasionally use words, but they could not be understood or interpreted, *i.e.*, they were without sense. Under tonic and stimulating treatment, including large doses of nitroglycerin, he improved slowly, and eventually the sensory aphasia disappeared.

The patient with Raynaud's disease described by Osler had three attacks of transitory right-side hemiplegia and aphasia, and died six months after the third attack. In my patient there was no trace of hemiplegia. It is not at all unlikely that the aphasia in these cases is dependent upon vascular conditions in the brain similar to those in the extremities that become blanched and then cyanosed. Unless the disease is very severe the aphasia is not complete, and may manifest itself only in paraphasia and difficulty in interpreting spoken and written language. When the affection of the cerebral blood-vessels is so profound

[1] Wiener Klinik, 1882.

[2] American Journal of the Medical Sciences, vol. cxii., 1896.

as to cause hemiplegia the aphasia is usually total. In my patient the disturbance of speech was mainly on the receptive side of language, and there was no disturbance of locomotion except that conditioned by the peripheral manifestations of the disease and the asthenia which is always such a profound symptom.

Of the toxic aphasias, that produced by uræmia is by far the most common. It may be a mistake to include it in the dynamic aphasias, for it must be classed with the cerebral symptoms of a paralytic order, provoked by a toxic substance in the blood. Uræmic aphasia is seen oftener in the old than in those of middle age, and oftener in children than at any other time of life. The frequency of uræmic aphasia in children is accounted for by the fact that post-scarlatinal nephritis and nephritis following other infectious diseases are more common at this time of life. During the years of fullest maturity, uræmic symptoms are more liable to be dependent upon cirrhotic kidney than upon any other form of kidney disease. Clinically, uræmic aphasia is more commonly of a mixed form than it is visual, auditory, or articulatory, but of course this depends, as does the determination of the variety of aphasia in any given case, upon the part of the zone of language that is predominantly affected.

In cases of transitory aphasia, accompanying grippe, pneumonia, etc., the aphasic symptoms frequently come on after the patient has been "flighty" or delirious, and usually the motor form predominates. A case of this kind is reported by Isager.[1] A child ceased to speak after

[1] Isager: "Et Tilfälde af Afasi i Tilslutning til en krupös Pneumoni." Hosp. Tid., 1894, p. 42.

the crisis of a croupous pneumonia. He understood fully what was said, he could not speak voluntarily, he could not repeat, nor could he read aloud. There was no paralysis, and after a week he began to use individual words, and in the course of a few days the faculty of speech was quite restored.

It is not at all unlikely that cases of aphasia occurring with lead poisoning are in reality dependent upon a lead encephalopathy, and the aphasic symptom complex may be looked upon as a focal or localizing symptom. This leads me to say another word anent the aphasic speech disturbances of the specific fevers. A great deal of attention has been devoted to this subject, and it is by no means settled that the majority of cases belong to the one form of aphasia, organic, or to the other, dynamic. It is likely that some are of the first kind and others are of the second. It is known that the infectious diseases predispose very materially to septic states of the blood, to phlebitis, to endocarditis, and to other conditions directly causative of embolism and thrombosis, and thus indirectly to organic aphasia by producing an obstruction of the middle cerebral artery and its branches which supply the zone of language, and which leads to softening.

The aphasic speech disturbances that are sometimes, though rarely, associated with chorea, primary degenerative tic, and with different forms of peripheral irritation, usually called reflex, such as from intestinal worms, phimosis, etc., are very uncommon, although the existence of cases of this kind seems to be well substantiated. They do not call for particular discussion. The aphasia is to

be looked upon as the result of reflex inhibitory phenomena and purely dynamical in constitution.

Aphasic symptoms occasionally develop after a considerable loss of blood, such as from epistaxis (Berthold), rupture of varix (Storp), post-partum hemorrhages, or similar conditions causing acute anæmia. Hallervorden[1] deplores that so little attention is given by writers on the etiology of aphasia to acute anæmia. As a matter of fact, unless the loss of blood causes a marantic thrombus in some cortical branch of the left Sylvian artery, genuine aphasia is an excessively rare symptom of such hemorrhages. Sudden partial exsanguination is often accompanied by dysarthria, sometimes by anarthria, but these are bulbar symptoms, just as the syncopal attacks that often occur from the same cause are bulbar manifestations. True aphasia, the result of loss of blood, is usually partial, and may be manifest through both the reception and the emission of speech. It is usually transitory and is one of the first exhaustion symptoms to disappear.

There remain to be discussed in the etiology of aphasia two of the most important organic diseases of which aphasia is a symptom. These are tumor of the brain and cerebral apoplexy. Aphasia as a localizing symptom of new growth in the brain is one of the most important and trustworthy guides. As a rule, tumor presents the ideal lesion to cause a strictly confined destruction of one of the centres in the zone of language. At least, I mean to say, that it is so at the beginning of the tumor. Tumors begin in the great majority of cases in one of two ways: by a proliferation of the tissue of the part which is pathologi-

[1] Hallervorden: Deutsche med. Wochenschrift, vol. xxii., No. 5, 1895.

cal from the beginning, or by the appearance of a tissue that is foreign to the part. In the beginning the abnormality is small, perhaps microscopical. It may be strictly confined to an area that has such highly individualized function as the angular gyrus, the first temporal convolution, or to a definite part of the motor area, and at such time it will produce symptoms of almost mathematical precision. These symptoms may occur before the symptoms of brain tumor, that are so consecrated by time that they are called cardinal, develop. If the physician takes proper recognition of them and gives proper interpretation to them, such perspicuity on his part may be rewarded by the life of the patient. It is not necessary to point out in detail what these varicus symptoms are. If one has in mind the topography of the surface of the cortical substance, and the functions allotted to it, as well as an understanding of the connecting pathways of these cortical areas, it is unnecessary to make explicit recitation of such symptoms.

Aphasia may be a prominent symptom of a recoverable form of tumor, gumma, and therefore the importance of recognizing it and applying appropriate treatment is apparent. A most instructive case, showing not alone the amenability to treatment of very profound forms of sensory aphasia when due to syphilitic lesion, but also the accuracy with which such a lesion can be localized from the clinical data, is one recently reported by Bramwell.[1] A young widow, who had had several miscarriages and abortions, complained after repeated exposure to the sun's rays, while working in the fields, of severe headache, worse at

[1] Bramwell: "Illustrative Cases of Aphasia." Lancet, 1897.

night; mental stupidity and loss of memory (she stated that she felt as if she was in a mist); and dimness of vision. On examination there were found slight optic neuritis, right-sided homonymous hemianopsia, almost complete word blindness, complete agraphia. She could not write spontaneously, from dictation, or from copy. There was inability to name objects correctly, and when she tried to name she nearly always applied the wrong name, although she recognized when others called things by the wrong name. The auditory speech centre was practically normal, as was likewise the articulatory speech centre. Soon after iodide of potassium was prescribed the symptoms began to be ameliorated, the headache was relieved, the mental condition cleared up, the word blindness and the agraphia disappeared, and the fields of vision gradually enlarged until at the date of her discharge they were quite normal. She regained possession of the modes of speech in the following order: First, ability to name objects; then ability to read letters and words, but at this stage she was completely unable to write spontaneously, to copy letters, or to write from dictation; then she gradually found herself able to read a little more each day, and in the course of a few days to write a few letters. It is worthy of remark that before she regained the ability to write letters she seemed to have an idea of the letters that she wished to write; in other words, she could evoke them in her mind. This is shown by the fact that when she was asked if she did not know the letter *a* she said that she did, that it was an *o* with a crook. Finally, she regained the power of writing fully, and at the time she was discharged from the hospital every one of the aphasic

symptoms, without exception, had yielded to antisyphilitic treatment.

I have recently had a very similar experience. A young married woman whose manifestations of constitutional syphilis were repeated abortions and exfoliating necrosis of the palatal region of the upper jaw began to complain of headache, nervousness, flightiness, depressed states of mind with frequent crying-spells, and of trouble in seeing. Later, she complained of a subjective feeling of numbness and unwieldiness in the right hand. This was followed after a few weeks by a convulsive attack beginning in the right hand and involving the entire extremity, without loss of consciousness. After the convulsion ceased she was unable to speak and to write for several hours. There was no hemianopsia, apparently no disturbance on the receptive side of language, and she could understand what was said to her. She had three attacks similar to this, and after the last of them I saw her. The twitching of the right upper extremity had ceased and there was no evidence of hemiplegia, but the patient had well-marked symptoms of cortical motor aphasia, which gradually disappeared after several hours. She made a complete recovery under treatment by mercury and the iodides. All the symptoms, including a slight degree of choked discs, had disappeared at the end of two months' energetic antisyphilitic and restorative treatment.

This, then, shows clearly that the etiology of aphasia, and that, too, of organic aphasia, has a most important bearing on the course and outcome of the symptom and on determining the treatment. In Bramwell's case the lesion, although it did not destroy the higher visual centre

in the angular gyrus, made such inroads upon the latter that it determined its functional overthrow, as indicates the agraphia, writing in all of its modes having been lost, while the homonymous hemianopsia and the alexia, the inability to apply the proper names to objects, showed that the way from the primary visual centres in the gray matter of the occipital lobes around the calcarine fissure to the higher visual centre was severely encroached upon.

The efficaciousness of prompt and energetic treatment directed against the luetic meningitis or the gummatous encroachment upon the cortex is shown very strikingly by the disappearance of the symptoms in my own patient.

Of the large number of examples of cases of brain tumor whose existence was directly pointed by the presence of aphasic symptoms, I shall cite but one example, and that recorded by Walton. The patient was a man, forty years old, who suffered from gradually increasing attacks of headache, at first of brief duration, later continuous, together with difficulty in speech ("ataxic aphasia"). He complained of red spots in the field of vision, and examination showed word deafness, word blindness, and right-side hemianopsia, in addition to double optic neuritis and paralysis of the left abducens. The patient was trepanned, but after the skull was opened it was decided that the tumor was too deeply seated and too extensive to warrant interference. The patient died two months after the operation. The autopsy showed a more or less lobulated pear-shaped mass, occupying the region of the posterior parietal and anterior part of the occipital lobe on the left side, loosely attached in some places to the brain substance, infiltrating it in others. The temporal lobe was

pushed downward, the occipital backward. The growth reached practically to the mesial surface, and on microscopic examination it was found to be a glioma.

In this case, therefore, the symptoms were most pathognomonic and suggested with great accuracy the locus of the growth. A lesion that will produce word blindness, left-side homonymous hemianopsia, and word deafness can be situated only in the angular gyrus, extending far enough centrally to sever the radiations of Gratiolet, and downward to impinge upon the auditory centre in the first temporal convolution of the left side. This case furnishes opportunity to say another word concerning the laxity in the use of terms by even those most worthy of the name of cultured neurologists. In this case it was said that there was ataxic aphasia. Now, in addition to the fact that no designation of medical terminology is more indefinite than ataxic aphasia, it may also be said that for the reader who would judge of the speech defects in the case from the published report there was nothing to point the existence of articulatory motor aphasia; nor was there any lesion found on autopsy that would point to the existence of such a condition. Moreover, it is difficult to say from the autopsy report alone whether or not the cortical substance of the angular gyrus was completely destroyed. One infers that it was not, because the tumor was apparently out of the reach of the surgeon and could not have, therefore, forced its way to the surface; otherwise a part, at least, of it would have been removed. I wish hence to reiterate that, in the report both of the clinical side of a case and of the anatomical side of a case, those who would contribute to the real study of aphasia and thus to the

tomes of physiology, psychology, and biology, should be mindful ever of the need of accurate statements and of detailed description.

So frequently is aphasia an accompaniment of apoplexy, a term which I use to include rupture of a blood-vessel, obliteration of its calibre, and acute softening, that in the minds of many physicians the word aphasia suggests apoplexy. The genetic and anatomical relationship existing between the zone of language and the middle cerebral artery has already been mentioned, and, as the subject will be considered *in extenso* in the chapter on "Morbid Anatomy of Aphasia," it is referred to here in the briefest manner. Nor do I think it necessary in a chapter on the etiology of aphasia to remark on the causative factors of cerebral apoplexy.

Of the organic mental diseases that aphasia may be a symptom of, general paresis is the most important. In the beginning of this disease, which is pathologically a widespread degeneration of the cortex, the most prominent symptom may be aphasia. This probably coincides with a beginning degeneration in some part of the zone of language. Usually the aphasia is of the sensory type.

There remains but one form of aphasia to be mentioned, and this chapter is finished, and that is the form known as congenital aphasia, or congenital limitation of speech development. These cases are not very uncommon. Kussmaul described a number of instances more than a quarter of a century ago, and recently Gutzmann has studied the subject very carefully. Congenital aphasia may be partial or it may be complete. I do not include in this category lack of speech development dependent upon porencephalia

or other gross lesions of the cerebral hemisphere. Aside from the disturbances in the organs of perception which are a part of idiocy, there is a not inconsiderable number of cases in which, without any disease of the peripheral percipient sensory apparatuses, or of the central sensory perceptual areas, there is a condition of speechlessness, and that, too, entirely divorced from any apparent disturbance of intelligence. It is to these cases that Coen[1] gives the name of auditory dumbness in contradistinction to deafness and dumbness. Although these children do not present gross mental or psychical defect, usually careful examination shows that there are always a certain amount of abulia, tardiness in learning to walk, slowness in acquiring skill of any kind, and various other manifestations of psychic and somatic degeneration. A very remarkable feature, and one that should be borne in mind when in the presence of such cases, is that in about one-third of them there have been found adenoid vegetations. The only other etiological factors that can be enumerated are that this condition has been met with more frequently in males than in females, and that there is almost always a neuropathic heritage, particularly from the father's side, and that, although a great many of these patients acquire considerable speech facility, it is often later in life associated with stammering.

[1] Coen: Wiener klinische Rundschau, 1893, No. 6

CHAPTER X.

Morbid Anatomy of Aphasia.

Were one to write the morbid anatomy of aphasia in detail, he must needs consider the various disease processes forming the basis of all the organic and functional diseases, enumerated in the chapter on etiology, with which aphasia may be associated symptomatically. Such description would carry us far beyond the limits put upon this work. Indeed, I doubt if such consideration could be made to serve any useful purpose in elucidating the intricacies of aphasia. Never for a moment should we forget that aphasia is a symptom; and, although it is the most comprehensive symptom that a physician has to deal with, it nevertheless always remains a symptom. It has already been said that one associates in his mind almost involuntarily the symptom aphasia with the disease apoplexy, because it is with apoplexy, using this term generically to include hemorrhage, occlusion of the cerebral blood-vessels, and acute softening, that aphasia most frequently occurs. Notwithstanding this, a discussion of the various forms and stages of cerebral hemorrhage and cerebral softening cannot be attempted here.

In reality aphasia due to lesion of the speech centres, true aphasia, is not often dependent upon cerebral hemorrhage, and this for the reason that cerebral hemorrhage limited to the cortex is an extremely rare condition. On

the other hand, acute softening following the occlusion of one of the cortical vessels is not so rare. Aphasia is rarely dependent upon hemorrhage in the basal region, for rupture of the trunk of the middle cerebral artery before it gives off the ganglionic arteries is such a serious condition that its occurrence is rarely consistent with the continuance of life. In young persons, however, the main trunk of the Sylvian artery may be occluded at any level beyond the branching ganglionic branches, and yet no softening take place. This accounts for the transientness of aphasia in young people and for the rapidity and completeness of their recovery. The first of the author's cases cited in this monograph is an excellent example of such speedy recovery, even in a very severe form of intracerebral vascular disease. A more convincing example, because accompanied by an autopsical report, has been published by Ross.[1] A young girl, while suffering from endocarditis, had, first, embolus of the femoral artery, and in rapid succession embolus of the kidneys, spleen, and left Sylvian artery, occlusion of the latter being evinced by an attack of right-side hemiplegia and combined motor and sensory aphasia. The patient died from exhaustion about a fortnight after the attack of aphasia. There was found in the main trunk of the Sylvian artery, a little outside the anterior perforated space, but proximally to any of the cortical branches, a very firm occlusion. Not the slightest amount of softening could be discovered, although Ross states that it is very likely that the nerve cells supplied by the Sylvian

[1] Ross: "Aphasia." Wood's "Medical and Surgical Monographs," vol. vi., No. 1, 1890.

artery had undergone fatty degeneration. There can be little doubt, judging from the outcome of analogous cases, that, had the patient lived, the nutritive and functional activity of the tissues of this area would have been gradually restored. Such an outcome should not be at all surprising when we consider that the cortical branches of this artery, unlike the ganglionic branches, are not terminal ones.

Although the study of the morbid anatomy of aphasia really began with Broca when he made an autopsy on his first aphasic patient, Leborgne, previous writers on the subject of speech and its defects were not without anatomical data to fortify their contentions, and of these may be mentioned Bouillaud[1] and Dax,[2] who based their inferences on not an inconsiderable pathological experience. Broca's first case was one that is usually looked upon as a typical example of cortical motor aphasia, but to-day it would, I believe, be considered one of subcortical motor aphasia, as the lesions found were widely distributed on the surface and in the substance of the left hemisphere. Although one of his cases showed extensive involvement of the auditory area, the father of aphasia persisted in the belief that the seat of lesion causing aphasia was in every instance in the posterior part of the third frontal convolution on the left side.

It would be supererogatory to enumerate even a part of the great number of autopsies to show that destruction of Broca's convolution is attended by symptoms constituting the symptom complex of cortical motor or articulatory

[1] Bouillaud : "Traité Clinique et Physiologique de l'Encéphalite," 1823.
[2] Dax : Gazette Hebdomadaire, April, 1895, No. 17. Republished.

kinæsthetic aphasia. This is something that every one admits. In the chapter on etiology of aphasia the relation of the area of Broca to the first cortical branch of the middle cerebral artery has been alluded to, and likewise the liability of this part of the cortex to be diseased. When it is diseased, a complexity of symptoms is produced that varies with the completeness of destruction. The lesions that are found there, constituting the organic basis of cortical motor aphasia, circumscribed encephalomalacia, localized gummatous meningitis, tumors, abscess, purulent leptomeningitis, penetrating wounds, were also discussed in the chapter on etiology and need no further enumeration here.

Future study of morbid changes accompanying motor aphasia should be particularly with two ends in view: first, to separate closely subcortical or pure motor aphasia from motor image aphasia, due to destruction of the area of Broca; and second, to show that when the disease process is limited to the latter area there is absolutely no secondary degeneration in any projection tract such as has been described by Pitres, Raymond, Brissaud, and many others as occurring in the inferior pediculo-frontal fascicle. To do this microscopical study of every case is necessary. Investigation of this kind can do for the motor side of aphasia what the microscopical studies instituted by Dejerine have done for the sensory side of aphasia. One cannot overestimate the importance of such microscopical study until he examines the literature and finds how easy it is to put different interpretations on the same case, if only the gross lesion is described. Incomplete anatomical investigation must account in some instances for the in-

terpretations of some of the cases that have been cited by authors to prove the existence of a graphic-motor centre, such as that of Henschen,[1] for example. The patient suffered from word blindness and agraphia, and after death there was found not only destruction of the foot of the second frontal but also a softening of the angular gyrus.[2]

[1] Henschen: "Klin. u. anatom. Beiträge zur Pathologie des Gehirns," Upsala, 1890–94.

[2] I am constrained to mention again, at the risk of wearying the reader beyond justification, the fact that just so long as writers on aphasia refuse closely to differentiate cortical from subcortical forms of motor aphasia, just so much longer will it be before the question, "Does destruction of Broca's convolution entail agraphia?" is settled to the satisfaction of every one. I am prompted to these remarks from an examination of the first of Bastian's recent lectures, which are now appearing in *The Lancet*. No one can be more profoundly cognizant of the fact, that, as a writer on speech disturbances, Dr. Bastian should rank with the Fathers of Aphasia, with Broca and Wernicke. It is not hazarding the truth to say that he has contributed more to the elucidation of the genesis of speech than has any English writer. My astonishment is therefore the greater that the following case, quoted from Wadham, should be offered in evidence to negative the claim that cortical motor aphasia always entails agraphia. I venture to state that if there has ever been a case published which is a mirror held up to the symptom complex and morbid anatomy of subcortical motor aphasia, it is the one just mentioned and which I now proceed to quote.

"A youth, aged eighteen, left-handed and ambidexterous, became partly hemiplegic on the left side and completely speechless after long exposure to cold. Twelve days later, on being given a slate and pencil, he wrote readily the word 'orange,' and when asked his name wrote it correctly with the right hand, although his mother asserted that she had never previously seen him do so. He and four of his brothers were left-handed. About a week after this, being still absolutely speechless, when asked whether he tried to speak and was unable, he wrote, 'Yes.' Asked if when well he wrote with his left or right hand, he wrote 'Both,' and then added, 'Fight with left.' In six weeks' time the hemiplegia had much diminished, but he still never had spoken a word, and continued to write all his wishes on a slate. His manner gave the impression of a very intelligent and rather facetious young man. At the expiration of three months he left the hospital, and when seen at his home later it was noted that the boy repeated after his mother's dictation various words and sentences with the intonation of one who endeavors to speak without moving the tongue. This power gradually increased until he was able to talk with sufficient distinctness to

The clinical history of Bastian's case stamps it as one of subcortical motor aphasia. Personally I should have been willing to make the diagnosis on one fact alone, viz., the patient's ability to write. I appreciate, however, that many physicians doubt the momentousness of this possession as a diagnostic indicator, and to those it might be said that a patient with cortical motor aphasia of such severity that it causes absolute speechlessness, and who is "very intelligent and rather facetious," and who passes through the stammering stage in learning to talk, and finally talks like one whose tongue is fixed in the mouth, has yet to be recorded, if the present writer's excursions afield into the literature of aphasia have been properly interpreted. A person to be facetious must be in the possession of internal speech at least, and if one of his speech centres is destroyed the integrity of internal speech is destroyed with it. The autopsy record puts the case in the right light, "*a lesion in the white substance beneath the Rolandic area.*" That is what the writer of these pages means by the term subcortical; therefore his astonishment that the case has been cited by Bastian in support of the statement that cortical motor aphasia does not entail agraphia.

We are in the early stages of positive knowledge concerning the exact limitations and seat of the lesions that produce aphasia, and of the conclusions that can be drawn from such lesions. If any one thing is needed to make the data of aphasia more reliable in the future and more

be perfectly understood by those accustomed to him. He subsequently suffered from necrosis of the jaw and died. At the necropsy a large area of softening was found in the right hemisphere, involving part of the white substance beneath the Rolandic area and the island of Reil. The left hemisphere was normal."

utilizable as scientific evidence in support of, or in behalf of, certain theories of the genesis of speech and its localization, it is that the patient be studied methodically and carefully, and when the case comes to autopsy that the findings be recorded in definite, conventional, and scientific language, particularly the seat, extent, and nature of the lesions that are found, and that there be depicted on a chart which represents the usual convolutional relations and fissuration of the brain the seat and extent of the lesion as it manifests itself on the cortex. A study of the subcortical lesions, to be of any considerable value, must be done with the microscope after the tissues are properly hardened and stained.

In the conduct of an autopsy of this kind a number of points are to be noted. After the removal of the calvarium, the condition of the pia should be particularly remarked, as knowledge to be obtained from its color, its adherence, and its consistence may be of much help in interpreting the nature as well as the duration of the lesion in the brain, especially if the lesion be of the cortex. Not infrequently aphasic patients will have a complexity of symptoms that indicates the destruction of one or more of the speech centres, and yet before death such a patient may develop symptoms that are to be explained only by positing disease of other parts of the brain. Observation and study of the macroscopic appearances of the lesions in such a case, their color, consistence, etc., will allow us to say with much positiveness that one lesion is so recent that it could not have been the cause of symptoms of long standing, and that another lesion bears the imprint of ancientness, and therefore must account

for the symptoms of similar duration. To be noted, then, are the color of the softening, the consistence, the exact location, particularly as contrasted with the undiseased hemisphere of the other side, the extent and number of the lesions, and the state of the blood-vessels. After this the consistence of the parts surrounding the area of softening should be carefully determined in order that one may estimate how extensive the secondary changes are which, though not sufficiently advanced to produce softening, may yet have reached a pathological state that robs the area of its function.

It should be particularly noted if the lesion limits itself to areas of the brain to which have been allocated by physiologists and clinicists special centres for language, or if a number of such areas are involved, thus constituting the anatomical foundation of mixed or compound aphasia. After the surfaces of the hemispheres have been carefully examined and any lesions found there specifically noted, then the degree to which such cortical lesions have extended into the substance of the brain and the subcortical location and extent of the lesions should be determined. This is by far the most difficult part of the autopsy. It is especially these subcortical lesions that should be most carefully studied, both in their extent and in their relation to, and separation from, the superambient cortex. Several cases of subcortical motor aphasia have been carefully studied, but the authors of such studies have interpreted them on the basis that the symptoms were due to the destruction of fibres of projection going from the foot of the third frontal convolution of the left side; that is, lesion of the fibres which are described as constituting the

inferior pediculo-frontal fascicle. Now, if the existence of this fascicle is denied, that is, if the third frontal convolution has no projection fibres, then the aphasic symptoms resulting from destruction of the parts of the cortex subjacent to it must be explained in another way. I believe they can be more convincingly interpreted in a way that has already been indicated.

The differentiation of subcortical motor aphasia from cortical motor aphasia has been most serviceable in widening our conception and in expanding our knowledge of the entire subject of aphasia, but much that is desirable can still be done in this direction. The lesions accompanying subcortical motor aphasia have been considered to some extent in the chapter on motor aphasia, so that it is unnecessary to enter very fully into the subject here. To show the distance that such lesions may be from the cortex in cases of clinically typical subcortical motor aphasia, I may refer to an instance published by Banti.[1] A man, sixty-two years old, who had never been able to read or to write, developed immediately after an attack of apoplexy hemiplegia of the right side and inability to talk. He understood questions that were asked him, but when he endeavored to respond the only sound that came forth was a confused unintelligible sound resembling "ti—ti—ti—ti—ti." He died five years after the first attack, there being in the mean time no essential change in his condition. At the autopsy there was found an apoplectic cicatrix of a brownish-yellow color at the level of the internal capsule between the lenticular nucleus and the thalamus; that is, in the anterior part of the posterior segment of the

[1] Banti: *Loc. cit.*

capsule. Dejerine[1] has published an observation very similar to this. The patient was a man sixty-seven years old, who had right-side hemiplegia and aphasia of a number of years' standing, and who was absolutely unable to speak aloud. He could, however, whisper some words that were recognizable. There was no agraphia or paragraphia. He died eleven years after the beginning of the illness, and on autopsy there were found three small foci of softening situated in the interior of the hemisphere, one in the middle of the internal capsule, another in the caudate nucleus, and a third in the fibres of the white substance subjacent but some distance removed from the foot of the third frontal convolution. Although some writers, such as Brissaud, are unwilling to admit this case to the category of true subcortical motor aphasia, there would not have been any objection to its admission if all fibres coming from the articulatory areas of the cortex had been involved.

The plan suggested by Dejerine for the conduct of the autopsy after the superficies of the cortex has been studied is probably the most useful one, although no hard-and-fast rule can be laid down for one's guidance in this matter. Dejerine's plan is the one that was followed by Vialet and Mirallié, whose labors have done so much to put sensory aphasia, cortical and subcortical, on a satisfactory foundation. The exact position and extent of the lesions having been noted, a division of the brain should be effected that leaves the diseased region intact. The usual method of division is that recommended by Flechsig, which consists of a horizontal cut passing through the head of the cau-

[1] Dejerine : *Loc. cit.*

date nucleus and the median part of the thalamus. It is done in the following way: After the skull cap has been sawed in the usual way it is left in place, and a knife introduced between the borders of the severed bone cuts the brain horizontally, *en bloc*, from without inward. This method of section facilitates particularly study of the central masses and of the internal capsule. The brain is then hardened and a plaster cast taken of the two pieces of the hemisphere. This gives an exact representation of the convolutions and is of the greatest service in orienting one later when the microscopical sections are made. The further handling of the tissues to render them susceptible for coloration is the same as that for ordinary brain tissue, and does not call for special description. Naturally, a special microtome is necessary to make the sections, which are cut horizontally through the length of the hemisphere, and particular care is called for in handling these large sections. Mirallié has pointed out that, to use the method systematically and regularly and to get the greatest service from it, it is necessary to take every twentieth or thirtieth section and make a counter-drawing of the surface of the section on a piece of polished glass. This counter-drawing, used in connection with casts of the cortex that have been taken, makes it very easy to tell just what levels and what parts of the cortex the various numbered sections represent.

The investigations that have been carried out in this manner by Dejerine, by Vialet, Mirallié, Redlich, Wyllie, and others have been the means of advancing our knowledge of sensory aphasia, and especially of subcortical sensory aphasia, to a degree that is not easy to overesti-

mate. In fact, it must be said that the reliable anatomical data of sensory aphasia have come from the investigators whose names have just been mentioned, although we do not mean to say that those of other pathologists and clinicians, such as Bastian, Seguin, Henschen, Pick, Wilbrand, and others, are not of the greatest importance.

The monograph of Vialet which appeared in 1893, embodying the report of several cases of sensory aphasia which the author had studied personally and with Dejerine, marked an epoch in the knowledge not only of the cerebral centres of vision and the intracerebral visual mechanism, but also of sensory aphasia. These cases and a few others which I shall presently mention are of such importance in exposing the pathological anatomy of sensory aphasia that every writer who essays to describe the morbid anatomy of aphasia must cite them.

I shall first refer to the findings in one of Vialet's patients, in whom the symptom was left homonymous hemianopsia, pure cortical hemianopsia. On removal of the brain an ancient spot of softening was found in the anterior one-fourth of the cuneus. Serial sections, made in the manner described above, showed that destruction of tissue was very much more extensive than the appearance of the lesion on the cortex indicated. The softening involved the anterior two-thirds of the cuneus, the anterior part of the calcarine fissure and the tissue in which the parieto-occipital fissure pushes its way, and the foot of the cuneus reaching as far as the foot of the hippocampus. In short, there was softening of the entire area supplied by the anterior branch of the occipital artery.

The consequent secondary degeneration involved the optic radiations of Gratiolet and the interhemispherical association fibres of the fibræ callosæ. The internal geniculate body, the thalamus with the exception of the outer part of the pulvinar, the anterior quadrigeminal tubercle, and the entire internal capsule, as well as the foot of the peduncle, were entirely spared.

In a case of this kind there was naturally no aphasia, and no disturbance of reading except that conditioned by loss of vision. It is for this reason that I refer to the case here. The disability of such a patient to read is entirely the same as that which might result from destruction of one-half of the retina or other peripheral defect, and it is grossly misleading to speak of it as "disturbance of reading."

One of the cases described by Dejerine and Vialet was one of pure word blindness. The patient had right homonymous hemianopsia, associated with inability to read letters and words. He could write voluntarily and from dictation, but he could not copy correctly. The autopsy showed a focus of softening evidently of long standing, situated in the base of the cuneus and the posterior portion of the lingual and the fusiform gyri. Examination of microscopical sections of the brain showed that the softening did not confine itself to the cortex, but that it could be traced in the depths of the white substance from the calcarine fissure to the ependyma of the ventricle, where there was complete destruction of the tapetum, the optic radiations of Gratiolet, and the inferior longitudinal fascicle at the level of the lower wall of the occipital horn. In addition, there was found secondary degeneration of

the radiations of Gratiolet, that pass beneath the field of Wernicke in the auditory area.

Another case was one of complete sensory aphasia, the patient being both word blind and word deaf. The area of softening involved the posterior parts of the first and second temporal gyri, the angular gyrus, the larger part of the external surface of the occipital lobe, the pole of the lobe being spared. In this case microscopical examination showed that not only the cortex but the white substance of the parietal and occipital lobes was affected. The three layers of fibres which border the external surface of the lateral ventricle were degenerated. The inferior longitudinal fascicle and the optic radiations were only partially destroyed. In fact, the inferior longitudinal fascicle was remarkably well preserved in all its internal parts and in the interior walls of the ventricles. The lesion destroyed the cortex of the posterior part of the first temporal and all the inferior parietal, reaching as far forward as the foot of the ascending parietal and the posterior part of the island. The posterior part of the internal capsule from its beginning in the lower part of the ovale, as far as the inferior part, was degenerated. The optic radiations were matted by the first lesion to the upper part of the thalamus, and this seemingly had served to preserve the integrity of these fibres at lower levels. In contradiction to case three, this case showed that the anterior part of the field of Wernicke was degenerated.

The fifth case was one of true sensory aphasia, word blindness. On autopsy a spot of softening the size of a silver dollar was found in the angular gyrus. Microscopically it was seen that the lesion began at the point of the

ventricle which penetrated the softened mass, and the degeneration involved the bundle of the white sagittal substance, the radiations of Gratiolet in varying degrees of severity, the fibræ callosæ, and the long occipito-temporal association fibres. The two forceps were intact. The fibres situated on the external border of the ventricle suffered particularly. In the upper part of the thalamus there were two kinds of lesion: the posterior one very marked, extended into the zone of the optic radiations; the anterior occupied the corona radiata at the level of the posterior part of the thalamus, the secondary degeneration following along the course of the inferior longitudinal fascicle and the optic radiations, although the tapetum was preserved.

A communication concerning a case of pure word blindness, reported by Hoisholt [1] as a case of word blindness and music blindness without agraphia, is accompanied by a report of the autopsy, which shows how a lesion beginning in the posterior pole of the brain and producing symptoms of subcortical or pure sensory aphasia may extend forward until it implicates some of the speech centres themselves.

Hoisholt's patient was a musician, sixty-three years old, addicted to alcohol. Entirely cognizant of time and place, he was somewhat confused and his memory of recent events was impaired. His language was coherent, and speech was normal, both in form and arrangement. The intelligence of the man, and the comprehension of what was spoken to him, likewise seemed to be normal. He

[1] Hoisholt: "A Case of Pure Word Blindness." Occidental Medical Times, vol. vii., p. 483, 1893.

was able to spell words correctly, and also to write properly from dictation his name and a number of short English words, but there was an inability to read what had been written, even his own name. He would generally read the letters of the alphabet correctly, but was unable to read the smallest words. The ability to see and to recognize objects at a distance was preserved. He played from memory the most difficult passages without a fault, but when requested to play by note he tried to do so and failed, hesitating and playing something not before him. He was unable to name correctly any written notes. There was no trace of paralysis. Hearing was impaired and there was complaint of imperfect vision. Careful examination showed that there was left homonymous hemianopsia. For a time there was some improvement in his general condition, but finally the visual defect became more pronounced, the fields of vision becoming more and more contracted until there was total blindness, while the pupils grew larger and failed to react to light. Death resulted from cystitis. Upon post-mortem examination the whole occipital lobe of the left side of the brain presented a yellowish-green color, and, viewed from above, appeared to be depressed below the general level. The convolutions of this area were reduced in size. These changes extended forward and upward as far as the angular and supramarginal gyri, upon which they impinged, and inward along the median surface of the occipital lobe, the tip of which was considerably softened. The posterior part of the right hemisphere was of a yellowish-red color, from the occipital lobe upward and forward, a little beyond the limits of the change of color upon the left side. The convolutions were flattened, but not so narrow or contracted as those of the opposite side. The cortical substance around the posterior extremity of the temporo-sphenoidal

convolution (angular gyrus) was somewhat depressed below the level of the surrounding surface, and presented several hemorrhagic spots varying in size from that of a pinhead to a pea, some of them extending through the whole thickness of the cortex. Smaller hemorrhages were also visible on the median surface of the occipital lobe in the fusiform lobule.

Although it is out of place to discuss here the clinical features of this case in relation to the anatomical lesion, it is appropriate to call attention to the fact that in the beginning the lesion of the posterior cerebral artery manifested its destructive effect in the interior of the occipital lobe and as the lesion extended posteriorly and anteriorly it caused respectively true cortical anopsia and true word blindness.

A case of sensory aphasia reported by Dejerine and Mirallié is accompanied by the results of a most carefully conducted autopsy and microscopical examination of the diseased focus and its consequent secondary degeneration. The patient had presented typical symptoms of sensory aphasia, associated in the beginning with right hemiparesis, which soon disappeared. The course of the disease was attended with considerable amelioration of the word deafness, but the complete alexia, total agraphia, paraphasia, and jargonaphasia were very pronounced and remained until the end. There was no optic aphasia or mind blindness, but on account of the difficulty of communicating with the patient it had been impossible to determine the absence of hemianopsia.

The autopsy showed a lesion of the supramarginal and of the angular gyrus, and also of the part immediately ad-

jacent to the latter in the inferior parietal lobule of the left side. On microscopical examination a large focus of softening was found surrounding the marginal fissure of the island, and destruction of that part of the inferior parietal lobule known as the angular gyrus. In the white substance of the ascending frontal convolution there were three foci of degeneration. There was also descending, retrograde degeneration in the optic radiations, the inferior longitudinal fascicle, the pulvinar, the external geniculate body, and of the internal capsule and the foot of the peduncle.

In this case the three distinct and primitive foci were: 1. A focus, by far the most important, situated in the posterior marginal fissure of the island, which had severed the base of the inferior parietal convolution just at the point of junction with the island. This focus comprised all the supramarginal gyrus, and destroyed the white fibres of this convolution and the cortex as far forward as the fissure of Sylvius. 2. A focus of softening occupying the base of the Rolandic operculum at a point where it is continuous with the island. 3. A focus measuring only a few millimetres in diameter, just at the crest of the angular gyrus, and which caused a degeneration very strictly confined in the middle part of the white substance of this convolution. These foci had each caused secondary degenerations, the principal one of which had severed completely the retrolenticular segment of the internal capsule, the radiations of the thalamus, and the inferior longitudinal fascicle. Behind this point the degeneration followed the external face of the lateral ventricle and reached to the occipital lobe. The fibres of the corpus

callosum which turn about the posterior extremity of the lateral ventricle were degenerated, forming at this level a zone of translucent fibres, standing out in contrast to the normal fibres of the corpus callosum. Anteriorly the degeneration penetrated the posterior part of the thalamus. The pulvinar was much atrophied, and its radiating fibres had disappeared. The external geniculate body and the white fibres surrounding it showed slight departures from normal.

In this case, the original lesion occupying the posterior marginal fissure of the island had separated the angular gyrus and all its connections from the zone of language. Although the visual images of words which are stored in the angular gyrus were in a measure preserved, they were no longer accessible to the zone of language, and this isolation of the angular gyrus had caused from the standpoint of its function bearing on internal language the same results as a direct lesion of the centre for the visual images and words would have. Mirallié, in reporting the case, says very truthfully that this is the first time that a case of this kind had been studied microscopically, and the scientific accuracy with which the lesion and its subsequent secondary degeneration were depicted allows of true interpretation of the symptoms of the case, for without such microscopic examination the totality of the symptoms would have been much less intelligible. The anatomical findings in a case of sensory aphasia under personal observation have been detailed in connection with the history of the case, and need not be repeated here.

The morbid anatomy of a case of subcortical sensory aphasia of the verbal type (*pure* verbal blindness of De-

jerine, subcortical alexia of many writers) has recently been studied with great care by Redlich. The patient, a sixty-four-year-old man, had had for a considerable time loss of visual acuteness, dependent upon optic atrophy, but this did not prevent him from discharging the duties of a scrivener. Later he developed a right-side motor and sensory hemiparesis, right-side hemianopsia, and word blindness. The hemiparesis was transitory. After the first fleeting disturbances following the shock there was total literal and verbal alexia, but no mind blindness. Writing was undisturbed, both voluntarily and from dictation.

At the autopsy there was found a spot of softening in the left occipital lobe immediately around the calcarine fissure in the lingual and fusiform lobules, extending as far forward in the medullary substance as the posterior horn of the ventricle. The splenium of the corpus callosum, the posterior part of the thalamus, and the tail of the caudate nucleus were softened. Microscopical examination showed destruction of all the optic radiations; and the forceps major as well as a part of the forceps minor were degenerated. The inferior longitudinal fascicle was also the seat of degeneration. The cortex of the angular gyrus was entirely intact. The left fornix, the anterior portion of the cornu ammonis, and the tapetum showed some spots of softening, which, according to Redlich, had interrupted the connection between the right visual area and the left visual centre, the author believing with H. Sachs that this pathway is through the tapetum. On account of the interruption of the inferior longitudinal fascicle, the connection between the left visual centre and

the zone of language was interrupted. This case is in reality one of the most important that have yet been contributed to the fund of exact knowledge concerning the pathology of aphasia, and although its reporter draws what I believe to be some unwarrantable inferences, which are criticised in the chapter on "Sensory Aphasia," particularly concerning the neural basis of writing, it nevertheless puts beyond doubt the existence of this form of aphasia. Redlich is of the opinion that cases of this kind would be better designated by the term intercortical aphasia than subcortical.

There are a number of other observations that have been of great importance in corroborating the conclusions of Dejerine and his pupils in regard to sensory aphasia. Wyllie has published the report and autopsy findings of a patient with subcortical visual aphasia, the two prominent symptoms having been word blindness and homonymous hemianopsia. The lesion was found to be a softening of the white matter in the floor of the posterior horn of the left lateral ventricle. It was due apparently to occlusion of one of the branches of the posterior cerebral artery. The softening confined itself strictly to the white matter, and, though the gray substance of the convolutions immediately over it was in a somewhat sunken and atrophied state, there could be no doubt that this was merely a condition consequent to the destruction of tissues underneath.

Examination of the sections of the brain showed that the atrophy extended from the tip of the under surface of the occipital lobe, where the dilated posterior horn of the ventricle reached to within an eighth of an inch of the sur-

face forward as far as the middle of the crus cerebri. That is to say, the convolutions affected were the lingual and fusiform, together with the posterior half of the gyrus hippocampus. There was no involvement of the angular gyrus. In this case the disease of the white matter of the occipital lobe had involved the fibres connecting the angular convolution with the right and left primary visual areas, thus causing word blindness; and on account of the interruptions of the radiations of Gratiolet from the primary centre for vision in the left occipital lobe there resulted hemianopsia.

A second case recorded by the same writer is no less interesting. A man seventy years old developed a slight motor and sensory hemiplegia with well-marked right lateral homonymous hemianopsia. The hemiplegia soon almost completely disappeared, but the hemianopsia and total word blindness continued. There were a slight degree of word deafness and amnesia of nouns. At the autopsy the zone of language macroscopically was entirely normal. The chief morbid appearances were found on the under surface of the occipital lobe, involving principally the fourth temporal lobe, bordering the occipital convolution, the hippocampal convolution, the lingual convolution, the anterior part of the cuneus, and the calcarine fissure. On section of the brain, it was seen that beneath the thin cortex there was such marked atrophy of the white matter that the cortex was in direct connection with the ependyma of the ventricular horn. At this region of greatest atrophy, *i.e.*, the under surface of the occipital lobe, about the middle of the inferior temporal occipital or the fourth temporal convolution, there was a marked

depression on the surface of the brain about two inches in length and an inch in breadth. This depression marked the situation of a cyst-like cavity where the gray matter and the subjacent white matter had been almost entirely destroyed.

Serieux has recorded a case of word blindness with agraphia caused by destruction of the angular gyrus, and, although the report is not accompanied by the details of a microscopical examination, the strictness with which the lesion was confined to the inferior parietal lobule makes it a very important and valuable case, more, however, as substantiating the allotted function of the angular gyrus than as a contribution to the morbid anatomy of aphasia.

The same writer has also published a detailed account of a case of mind blindness associated with word blindness. The patient, a woman, sixty-two years old, had a stroke followed by transient paralysis, word blindness, and agraphia, mind blindness, word deafness, and paraphasia. The patient's condition had bettered somewhat, when she died suddenly from an intercurrent pneumonia. Unfortunately, the lesions in the brain were multiple, there being on the left side a softening in the inferior parietal and also a limited focus of softening in the posterior extremity of the first and second temporal convolutions. On the right side of the brain a softening, somewhat more extensive, was found in practically the same areas.

A very similar observation to this has been communicated by Bruns,[1] except in this case the clinical phenomena

[1] Bruns: Neurologisches Centralblatt, Nos. 17 and 18, 1888; Nos. 1 and 2, 1894.

were those characteristic of complete sensory aphasia, with right homonymous hemianopsia. The autopsy showed that the greater part of the white substance of the first temporal convolution and the adjacent parietal convolution were in a state of advanced softening.

There are few better cases to illustrate the lesions of sensory aphasia, type of word deafness, than a case published by Leva.[1] The symptoms pointed most unerringly to a lesion of the auditory centre, and on autopsy there was found a yellowish, fluctuating sunken-in area of softening in the middle segment of the left upper temporal convolution and almost exactly two millimetres from the upper border of the second temporal convolution. The softening was found to be a cyst filled with milky, cloudy fluid. It measured in every diameter about three centimetres. Anteriorly it extended to one centimetre from the tip of the first frontal gyrus and posteriorly to two centimetres from the posterior end of the same gyrus. Internally it approached the two external segments of the lenticular nucleus. In the second frontal convolution of the left side there was another small focus of softening.

Although there are on record many cases of aphasia accompanied with more or less autopsical details, I shall have to content myself with the relation of one more case taken from the literature, and that a case of subcortical word deafness, published by Pick. The patient was a typical case clinically. She could understand neither spoken

[1] Leva: "Localisation der Aphasien." Arch. f. path. Anat. und Phys. und f. klin. Med., Berlin, Mai, Bd. cxxxii. (Folge xiii., Bd. ii.), H. 2, p. 333, 1893.

speech nor melodies. On removal of the brain, no departure from normal could be made out save that the convolutions seemed to be a little sunken. Examination of the superior gyri of the temporal lobes showed that they were somewhat abnormal in consistence and color. On the right side the first temporal and a large part of the second temporal, the island of Reil, and the adjacent parts of the anterior central convolution and of the inferior frontal convolution were transformed into a whitish-yellow firm mass. The environment of these softened parts was more dense. The lateral ventricle of this side was slightly distended and contained some yellow serum. Section of this hemisphere made after the plan of Pitres showed that the cortex of the affected convolutions and the white substance were the seat of yellow softening. This softening comprised, in a section made through the ascending frontal convolution, the region just in front of the external capsule and most external part of the lenticular nucleus. The globus pallidus and the internal capsule were intact. In the left hemisphere the posterior part of the first temporal convolution and the supramarginal gyrus were softened, the same as on the right side. Frontal sections of this hemisphere showed that the softening was superficial and affected no part of the external capsule or the central nuclei. The island of Reil was intact. The softened areas in this hemisphere had a more gelatinous aspect and the substance of the adjoining convolutions was firmer. On account of the meagreness of the anatomical details in this case, and on account of the extensiveness of the lesion, it bears only indirect testimony in behalf of the exact localization of the aphasia lesions.

In a word, the pathology of true aphasia is the pathology of a lesion that injures the zone of language, and of sub-cortical aphasia a lesion of the immediate incoming and outgoing pathways by virtue of whose integrity the speech centres manifest their function.

CHAPTER XI.

Remarks on the Treatment of Aphasia.

I shall discuss the treatment of aphasia very briefly, from the standpoint of the physician and surgeon, and from the pedagogue's point of view.

Unhappily neither the physician nor the pedagogue can be of considerable assistance to the vast majority of aphasic patients. The medicinal treatment depends entirely upon the nature of the lesion that causes the aphasic symptom complex. If the lesion be a focus of encephalomalacia, then all that can be expected of medicinal treatment is to assist nature to prevent further destruction of tissue, and particularly to assist in preventing a repetition of the immediate exciting cause of the softening. On the other hand if the lesion be a gummatous meningitis, or an isolated gummatous formation, in the zone of language or the subcortical speech tracts, and these can be diagnosticated as such, medicinal treatment is of the greatest value. A case of sensory aphasia recorded by Bramwell and cited in another chapter is in evidence. This patient had the profoundest symptoms of sensory aphasia, yet she fully recovered under the influence of antisyphilitic medication. Another case in which the results of antisyphilitic treatment were most gratifying, even though the symptoms did not completely yield to medication, has very

recently been published by Mantle.[1] The difficulty in cases of this kind is oftenest with the etiological diagnosis. Usually the patient is not in condition to vouchsafe any information concerning himself, and as his family is, as a rule, ignorant of such matters, the physician is compelled often, if he has not been familiar with the patient's history, to make a diagnosis of previous syphilitic infection on less satisfactory data than are ordinarily considered essential. Personally I am inclined to suspect a luetic origin in every case of aphasia coming on abruptly that occurs before the fifth decade of life, when valvular trouble of the heart, the recent possession of acute disease, and injury can be excluded.

It would be a work of supererogation to repeat in detail the treatment applicable to the different forms of aphasia, for it will occur to every one who has in mind the various causes of aphasia. The treatment for aphasia in one patient may be just as different from the treatment applicable to the next one as the causes are different. For instance, in the beginning the treatment of a uræmic patient is venesection if the patient has not an organic form of renal disease; yet this kind of treatment would be fatal to a patient whose aphasia was dependent upon autochthonic thrombosis.

When aphasic symptoms develop slowly without fever and with symptoms of increasing intracranial irritation and pressure, then tumor and abscess must be thought of. In making the diagnosis and the differentiating diagnosis one must be guided by the general rules applicable to the solution of these problems. When there are grounds for

[1] Mantle : British Medical Journal, February 6th, 1897, p. 325.

the belief that the lesion is of a luetic nature, then the administration of mercury and iodide of potassium cannot be carried out with too great promptness and attention. Syphilitic lesions that develop some years after the primary infection are, it is universally conceded, more amenable to the iodide of potassium than to all other measures combined. If, however, the date of the primary lesion is not very remote, then the administration of iodide should be simultaneous with the mercury, or the one should follow the other in the shape of a course of the one and then of the other.

The treatment of aphasia dependent upon organic disease such as tumor, abscess, purulent meningitis, and focal disease of any nature, does not differ from the treatment of these conditions when aphasia is not present. When their presence is attended by symptoms which seem to indicate that they are amenable to surgical treatment their removal should not be delayed. In fact, the aphasia is oftentimes the localizing symptom that makes diagnosis positive and operation possible. The case which I have cited of Zaufal and Pick, an abscess of the brain successfully treated by operation, is in evidence.

To enumerate the symptoms caused by focal, cortical, or subcortical disease that may cause aphasia would be a repetition of much that has been said in the chapter on "Diagnosis," and elsewhere. The seemingly widespread belief that aphasia is almost exclusively an on-hanger of the apoplectic state seems to necessitate, however, emphasizing the fact that some manifestations of the complexity of symptoms constituting aphasia are of great diagnostic im-

portance in nearly every disease affecting the brain. It is a common symptom in the recently recognized and described disease acute hemorrhagic encephalitis; it is perhaps the most constant symptom of abscess of the brain, on account of the pathogenetic relationship of disease of the middle ear and cerebral abscess, and it is not uncommon at some stage in the career of general paresis and of multiple, insular, and diffuse cerebral sclerosis; while its occurrence after injury which may cause localized inflammation of the meninges or of the brain itself, hemorrhage, depression of bone and spicules, is not unusual. Naturally, in order to produce aphasia, these factors must manifest their injurious activity on the speech centres, their interconnections, projections, or the immediate pathways leading to them; to be less specific, on the left hemisphere in right-handed persons and vice versa. The form of aphasia that any of these diseases and accidents may cause will depend upon the location of the lesion and not on its nature. The only variety dependent upon any of the above-enumerated conditions that is very uncommon is the subcortical form of motor aphasia; while the subcortical sensory variety is correspondingly frequent. This is readily understood, if we recall that the part of the brain which must be diseased to cause subcortical motor aphasia is well protected from injury and has no particular relationship to the important factors that condition abscess of the brain.

Oftentimes a careful consideration of the symptom aphasia in these diseases will be the most important factor in determining whether or not an operation shall be done, *i.e.*, whether the lesion is sufficiently localizable to warrant

advising the surgeon to trepan the skull and attempt to remove the materies morbi.

Taking it all in all, the question of the medicinal treatment of aphasia never comes up for consideration. The question that does present is: How shall we treat the condition of which aphasia is the symptom? To answer that question satisfactorily requires an intimate knowledge of the therapeusis of all the diseases, functional and organic, that have been enumerated in the chapter on "Etiology" with which aphasia may be associated. Treatment may consist of such a simple matter as the interdiction of alcohol in a case of toxic dyslexia, or it may require the combined skill of the physician and surgeon to diagnosticate and remove an abscess or tumor. The treatment of the dynamic aphasias is a different matter from the treatment of the organic aphasias. In the former all that is necessary is to remove the cause and the symptom will disappear, while in the latter the cause may be removed and the pathological condition which it has excited still continues and with it the aphasia.

The pedagogical treatment of aphasia is a matter of recent development. It has been the legitimate result of an inquiry into the physiological and psychological antecedents of articulate speech, and of clinical observations that when a young person became aphasic, even though the lesion was a very severe and extensive one, the faculty of speech was restored to him. Moreover, almost from the very beginning of the history of aphasia, it has been recognized that, even when the so-called "speech centre," meaning Broca's area, was completely destroyed, the patient regained occasionally some capacity to speak individual

words or a number of words. Various hypotheses have been formulated to explain these occurrences, the most widely accepted apparently being that of Jackson, who suggested twenty years ago that the "uneducated centre" of the opposite side is in a way related to conventional, emotional, and other forms of what he terms "degraded" speech, in contradistinction to intellectual speech. This is the theory accepted by many writers to-day. Recently Wyllie has framed a theory along somewhat the same lines on the "overflow of education into the opposite hemisphere;" the hemisphere that contains the zone of language takes up all that it can in the way of education, and that which it is not equal to taking up flows over into the other hemisphere. As I have said in a previous chapter, the entire subject of the repossession of the speech faculty in patients in whom it has been lost must needs be looked at to-day from another standpoint than it was a few years ago, when the various forms of subcortical aphasia had not been satisfactorily differentiated. It seems to me that in the light of our present knowledge of aphasia it must be granted that not only do the areas of the opposite hemisphere sometimes under the stress of education undertake, in a very incomplete way, the speech function of the destroyed area of the hemisphere phylogenetically and ontogenetically prepared to carry on the speech faculty, but that the immediate environmental areas of the speech centres of the left hemisphere may take up the function in part; secondly, that the opposite hemisphere, the one that has the zone of language ontogenetically developed, is not an uneducated hemisphere at all, but that it is in one sense just as much educated as the hemisphere in which the

zone of language is situated. It must needs be admitted that there are a general auditory area, a general visual area, and a general kinæsthetic area in the right hemisphere as well as in the left hemisphere, and that in-coming stimuli make a similar impression on it as they do on the so-called "educated" hemisphere. These impressions are bilateral in reception but unilateral in interpretation. This unilaterality of interpretation is determined by commissural fibres of the corpus callosum. Now the same factors that determine right-handedness determine also that the left hemisphere shall be the executive speech side, but the elementary work is done on both sides. It seems to me that so far every one who is willing to accept the suggestions of experimental physiology must go. How many are willing to admit that the execution of speech is an automatic act and requires no conscious preparation, if process of anatomical completion is not considered "preparation," is another matter. Those who believe that the execution of speech is an automatic act find it easier to explain how an approach to or an unfinished automatism can be assumed by the opposite hemipshere which is educated but which is not intended to be automatic, and especially in young children, in whom the habit of automatic activity has not become fixed by continued practice. I do not think it at all improbable that if a healthy child should be kept mute until it was from five to six years of age, that is, until such a time as the neuro-muscular apparatus subserving speech was fully developed, he would go through the lalling and other stages of speech imperfections as do children who begin "to learn" to talk before the executive parts are fully developed. The words

that such a child used (which would, of course, depend upon the words that he had heard) might, I believe, be perfectly formed. In other words, the execution of speech would be as automatic as breathing, and that in mankind speech is an endowment more than an acquirement. Furthermore, the factors that determine the seat of this automatic activity are the conditions that we have heretofore supposed determined the education of the left hemisphere.

A most remarkable case bearing on this matter has recently been published by Bastian. The patient was a boy, twelve years old, who had been subject to epileptic fits at intervals. The first of these occurred in infancy, when the patient was about nine months old. Toward the end of the second year the fits seemed to have ceased. The hearing was good and the child appeared to be of average intelligence—to be well, in fact, in all respects, except that he did not talk. When nearly five years old the little fellow had not spoken a single word, and about this time two eminent physicians were consulted in regard to his "dumbness." But before the expiration of another twelve months, on the occasion of an accident happening to a favorite toy, he suddenly exclaimed, "What a pity!" although he had never previously spoken a word. The same words could not be repeated, nor were others spoken, notwithstanding all entreaties, for a period of two weeks. Thereafter the boy progressed rapidly and speedily became most talkative, and spoke without the least sign of impediment or defect.

One other point that has previously been mentioned. A number of the cases that have been reported to show the assumption of speech function by the opposite hemisphere

have, I hope, been conclusively shown to be dependent upon a subcortical lesion and not upon destruction of a speech centre, and the partial or complete recovery of speech was commensurate with a disappearance of the conditions that had determined the partial interruption of the conducting fibres. In these cases recovery of speech has gone on *pari passu* with disappearance of other symptoms, such as hemiplegia, for instance. In other cases in which the lesion has been of the speech centres the partial repossession of speech has been due to the fact that the entire speech centre, which in the beginning of an aphasic attack was completely overthrown, has in a slight measure righted itself after the exudative and occlusive conditions have subsided. Then the patient finds himself in possession, to a very insignificant degree, of his previous speech endowment. In other cases there can be no question that the educated areas of the other hemisphere develop some executive capacity. This is determined artificially, *i.e.*, by education, and not ontogenetically as it is normally, except to the very slightest degree.

In brief, then, the education of an aphasic patient should consist in endeavoring to cause the centre or centres in the left side of the brain that are not destroyed by the lesion which causes the aphasia to take the initiative in the primary recall of words, and complete the "circuit" necessary for speech by forcing the educated opposite side to supply a centre similar to that which has been destroyed. For example, if the articulatory kinæsthetic centre is detroyed, the primary revival of the word that should be spoken is through the auditory centre, and this calls up in temporal coincidence or succession the

visual and the articulatory centres. The articulatory centre, being destroyed, the speech impulse of the formed word cannot be completed, and the kinæsthetic articulatory centre of the opposite side is acted upon through commissural fibres in just the same way as the articulatory centre of the left side was through intercentral fibres, in the beginning. The process of education is very slow and must be given artificial aid in the way of showing the patient what movements to make in order to get the variety of kinæsthesis of which it is desired to store up memories. The utilization of this suggestion is in reality at the bottom of educating patients with cortical motor aphasia to speak.

In these cases in which it is desired to supply the articulatory kinæsthetic memories, everything is to be gained by the use of the physiological alphabet, educating the patient to master the letter sounds. Even the briefest consideration of the physiological alphabet would require more space than can be given, and I prefer to say nothing in the way of explanation of it rather than give a faulty and imperfect exposition of the subject. To those who would have a most readable article on the subject, the first chapter of Wyllie's "The Disorders of Speech" is recommended.

When the auditory centre is diseased, then the task is to get a primary revival of the idea of words in the visual or the articulatory centre. This is a very much more difficult matter, because in the vast majority of peoples the primary revival takes place in the auditory centre, and when this is destroyed the patient is stranded, from a speech standpoint. The plan of education is in

reality that which is used for deaf-mutes, who are taught to think by the revival of the word impulse by the visual centres, the revivification of visual symbols prompted by hand or lip movements. In case of those born deaf and blind the primary revival is in the articulatory kinæsthetic centre, which, in cases like that of Laura Bridgman, is conditioned by the tactile sense. In fact, it is in all those defectives who learn to read aloud by the use of raised type.[1]

Patients with the auditory form of sensory aphasia should be patiently taught to repeat words the meaning of which is conveyed to them through other senses, the visual, tactual, and olfactory. In this way it is believed that generally the auditory area of the *same* side that is not destroyed or of the opposite side may develop some executive capacity.

The treatment of sensory aphasia conditioned by destruction of the visual centres is most unsatisfactory, and very little can be done to ameliorate the condition of such patients, even though all modes of education be assiduously employed. An effort should be made to teach the patient the recognition of forgotten symbols in connection with the arousal of other memories of them, the auditory and the articulatory.

In short, the pedagogical treatment of aphasia embraces the methods of the kindergarten and the methods for the instruction of those defective in one or more of the special senses. Even with their aid but little can be done.

[1] I am aware that these cases are somewhat opposed to the contention that the primary revival of words is never in the articulatory centre, and possibly the position I have taken may have to be modified or altered by further investigation.

CHAPTER XII.

Remarks on the Medico-Legal Aspects of Aphasia.

Aphasia is so frequently a symptom of mortal disease, and it is of so much more frequent occurrence in the aged than in the young, that its presence and occurrence often give rise to exigencies necessitating disposition of possessions in such a way as to satisfy later tribunals of justice. On the other hand, patients with aphasia are oftentimes so changed in demeanor, in conduct, and in appearance, and they respond to environmental conditions in a manner so different from that habitual to them when in health, that they are adjudged insane by the laity, and, unfortunately, occasionally by physicians as well. These two facts necessitate a discussion of the testamentary capacity and the mental status of patients with aphasia.

The literature bearing on the testamentary capacity of aphasic patients is not very extensive, and, by way of introduction, it may be said that much of it is valueless. A quarter of a century back the subject was discussed by Legrand du Saulle,[1] by Gallard,[2] by Bateman[3] and others, but, as these discussions were held before the subcortical

[1] Gazette des Hôpitaux, June and July, 1868, and *idem*, vol. lv., 1882.

[2] Clinique Médicale de la Pitié, reviewed in Le Journal de Médecine et de Chirurgie Pratiques, vol. xlviii., pp. 377–380.

[3] *Loc. cit.*

forms of sensory and motor aphasia were separated clinically and established on a firm anatomical basis, they are of comparatively slight value. For example, a case reported by the last-named of the trio mentioned above: An elderly man, about to be married, was stricken with right hemiplegia attended with aphasia. He, in anticipation of death, desired to make a will for the benefit of his *fiancée*. The instrument was prepared by his physician, with whom the testator communicated by means of signs which the former understood. The testator put his corroboration on communications to the physician by making an affirmative or negative gesture when the latter repeated them after having written down what he understood the deponent to say. For instance, he held up the hand and extended five fingers; then closed the fingers and extended them again; and repeated this performance three times to indicate thirty. On being interrogated if he meant "thousand" he nodded affirmatively. He was then asked if he wished his *fiancée* to have £30,000; he again nodded assent. When asked if it was his wish that she should have this sum absolutely, he made a nod of negation. When asked if she was to have it during her life and then to have it revert to his own family, he signified assent. He signed the document by making his mark, but owing to the non-satisfaction of a technicality of the law the will was not admitted to probate, although it must be evident to every one that the mental faculties of the patient were intact and that he had subcortical motor aphasia. As I have already hinted, it is around the differentiation of the subcortical and cortical forms of aphasia that this entire question of testamentary capacity revolves. If it were neces-

sary to define the status of the aphasic patient's testamentary capacity dogmatically and in a few words, I should say that, although every case is a law unto itself, no one is of sound and disposing mind who has true aphasia—aphasia due to lesion of the zone of language;—while a person who has any form of subcortical aphasia, be it motor or sensory, may be, and usually is, capable of indulging in civil transactions, although there are exceptions to the rule. If the integrity of the primary speech centres constituting the zone of language is necessary for the full and legitimate genesis and notion of the word, then disease of any of them must be attended by disorder in the conception and in the idea of the word. And as it is necessary to employ words or their motor equivalent in the making of a will, it will readily be seen that the patient with true aphasia cannot do this in a way to satisfy the law. Yet a person may have had true aphasia, and have recovered sufficiently, either by the education of the opposite side of the brain, or by the assumption of function by undestroyed portions of the diseased centre, to know the correct application of words and to use them rationally and intellectually. It is in these cases that the observations of the physician and the completeness with which he has examined and studied the case should have the greatest weight in deciding as to the patient's testamentary capacity.

Given a person with subcortical aphasia, the matter is very different. In a case of uncomplicated subcortical motor aphasia, that is, not associated with lesion of other parts of the brain that might interfere with intellectual functioning of the brain, the individual may be in full-

est possession of his faculties, including internal speech, and have simply a supreme inability to externalize his mental content. For purpose of contrast he may indeed be compared with a man who is bound and gagged. Probably no one would contend that the latter is incapable of making a will, although the inability to do so without outside aid must be very apparent. The testamentary capacity of patients with subcortical motor aphasia may, however, be impaired by coincident lesion of a nature similar to that causing the speech defect, occurring in other parts of the brain. For instance, a thrombus of luetic origin located subjacent to the executive motor speech centre may cause symptoms of subcortical motor aphasia, while coincident luetic disease of the blood-vessels in other parts of the brain may cause a degree of dementia inconsistent with the making of civil contract. But, as I have said, such superadded deficiencies must be detected or eliminated by the examination of the physician.

Subcortical sensory aphasia, be it of the auditory or visual kind, offers a more serious obstacle to the testamentary capacity of the patient than does the subcortical motor form, because in the former the indifferent attitude and the inattentive and unnoticing demeanor, which these patients so often have, lead those about them, and others with whom they may come in contact, to look upon them as insane. The testimony of these of the laity before a Surrogate, or before twelve of their peers, often has great influence even though it is contradicted by testimony which has the misfortune to be called "expert." But if the aphasic symptoms be wholly of a subcortical nature and uncomplicated, the patient has no defect of internal

speech, and because of the shortcomings of internal speech he should not be forced into the category of those of unsound mind because he is obliged to borrow another's eyes or ears. It would be just as legitimate to contend that a highly myopic or deaf person is incapable of making a will because he cannot lay hands on his glasses or his trumpet. As has been said before, the analogy is overdrawn, because uncomplicated forms of subcortical sensory aphasia are extremely uncommon, and it is the complications or coincident symptoms that add to the complexity of the question.

Thus it will be seen that my position in this matter is materially different from that of many writers on this subject. For instance, I cannot agree with Gowers, who says: "Word deafness is incompatible with will-making, because it is impossible to know whether the testator really understands what is said to him." If the auditory pathways were the only ones through which a patient could be communicated with, this would be true; but word deafness may be complete without involvement of the higher auditory centre and with the central visual mechanism intact, and therefore the patient can be communicated with by writing. Even in a case of word deafness and word blindness there might still be no optic aphasia and the patient could be communicated with by pantomime, sign language. Neither can I agree with Diller,[1] who says by way of introduction to a brief discussion on the medico-legal aspects of aphasia: "In such a study neither the site of the lesion nor the particular division or subdivision of aphasia present need be considered." For my part, I be-

[1] Journal of Mental and Nervous Disease, May, 1894.

lieve that the determination of the variety of aphasia that the patient has is the most important thing in determining the patient's capacity to make contracts, wills, checks, and indulge in other civil matters. No more can I subscribe to the statement that "Motor aphasia does not of necessity incapacitate the patient in will-making, etc." If by motor aphasia is meant aphasia due to lesion of the centre in which are stored the memories of articulation, then such aphasia does incapacitate the patient, for in every case of this kind there is some disturbance of internal speech, not to speak of external speech. In fact the majority of cases that have become famous in this country on account of the litigation connected with them, in which physicians have given testimony of the testamentary capacity of the patient, such as the Beven case, analyzed by Hughes;[1] the Parrish case, referred to by Ray;[2] and a case recorded by Clark,[3] will be found on close scrutiny to be cases of subcortical aphasia, and it was because of the symptoms of the latter that such testimony could be given, although at the time when some of the cases occurred the subcortical forms of aphasia had not been separated.

The need of making such differentiation in every case of aphasia when it is necessary to determine the testamentary capacity, or to make a determination of the mental status for other purpose, is well illustrated by the pertinent words of Hughlings Jackson on the subject: "Such a question as 'Can an aphasic make a will?' cannot be answered any more than the question, 'Will a piece of

[1] American Journal of Insanity, January, 1879, p. 410.
[2] "Medical Jurisprudence of Insanity."
[3] American Journal of Insanity, 1892–93, p. 291.

string reach across this room?' The question should be, can this or that person make a will?" And the determination of the variety of aphasia that he has will do more to answer this question than will anything else.

Physicians are rarely called upon to decide the question of the responsibility of an aphasic in criminal processes; but when they are, the same precepts should guide them in estimating the patient's mental responsibility as were laid down to determine his testamentary capacity. It should never be forgotten that the majority of cases of organic aphasia occur with diseases of the brain that put great inhibition upon the unfortunate victim's passions and emotions, and for the indulgence and manifestations of these he should not be held responsible in the same way as a normal man is. The physicial infirmities of patients with aphasia alone, especially the motor forms, usually spare them from crimes against person and State; while the speech shortcomings of sensory aphasia are of such a nature that patients must be cared for by others, and are therefore kept from indulging any such tendencies that might be prompted. Personally, I believe that a patient with subcortical aphasia, it matters not of what form, is as capable of determining between right and wrong, *meum* and *tuum*, as a normal person, coincident disease of the brain that might impair his faculties being excluded. The same cannot be said for a patient who has lesion of the zone of language; but in cases of this sort the eventual determination must be reached from personal study of the case and not from its conforming to any hypothetical conditions.

APPENDIX I.

Conduction Aphasia.

In the body of this work I have limited myself to a discussion of the varieties of aphasia that have been substantiated by morbid anatomical changes found post mortem. No one doubts the reality of the forms of aphasia discussed in the text, although different interpretations have been put upon the symptomatic accompaniments of each of them by different writers, but all are in accord as to their occurrence. A number of writers, prompted mainly by theoretical considerations, have described several varieties of aphasia dependent upon lesions situated between the different speech centres. To these varieties they have given different names, according to the posited seat of the lesion. The most plausible of these subdivisions is one described originally by Wernicke, to which he gave the name "Leitungsaphasie," conduction aphasia (intercentral aphasia, connection aphasia).[1] Personally, I believe that theoretically there are very good grounds for the differentiation of this form of sensory aphasia, but as

[1] Bramwell distinguishes two varieties of conduction aphasia: (1) connection aphasia due to interruption of the connection between the different cortical speech centres; and (2) commissural aphasia, due to interruption of the commissural connections between corresponding speech centres in the opposite hemispheres. He remarks that in the present state of our knowledge it is impossible to distinguish these commissural forms. The second distinction seems to me scarcely warranted.

yet there is no convincing anatomical evidence to prove its existence. Therefore it is referred to in an appendix, instead of in the body of the work. I shall cite briefly, and without critical comment, some cases that have been contributed to establish the reality of this form of aphasia. Even the most casual reader of the chapter "Conception of Aphasia" will recognize that the explanation of the occurrence of the one symptom of conduction aphasia, viz., paraphasia, given by those who have written on the subject is not in harmony with what has been contended for in that chapter. The cases of conduction aphasia that have been reported can all be explained as symptoms of sensory aphasia, and the variations in the clinical picture depend on the different locations of the lesion in the zone of language.

Every one must admit the possibility of the existence of a lesion in the zone of language between the auditory centre and the articulatory kinæsthetic centre, without destruction of either of these centres. On the other hand, the proximity of the visual and auditory centres and subjacency of the radiations of Gratiolet make it very improbable that a lesion could exist between these two centres and not implicate either of them.

A number of the most reliable writers on the subject of speech disturbances have given this form of aphasia extensive consideration. Wernicke, Lichtheim, Pick, and other writers have recorded examples in support of its occurrence and differentiation. Personally the writer is of the opinion that conduction aphasia can rarely be differentiated from sensory aphasia, of which it is a part. It is possible that such differentiation may be made when the

lesion is of the island, but careful observations are needed before this can be decided. When the lesion is of the island, the connection between the auditory centre and Broca's convolution may be only partially interrupted and some impulses sent from the former reach the latter. All of them do not. This remaining partial anatomical connection has been taken by some writers (Ziehen) to explain the occurrence of paraphasia in conduction aphasia. Before reciting the symptomatology that has been attributed to conduction aphasia, it should be stated that if the articulatory centre is dependent absolutely upon the excitatory influence of the auditory centre, as so many physiologists believe it to be, then an intercentral lesion (a Leitungs lesion of Wernicke) should produce as complete inability to speak spontaneously or on repetition as does destruction of the area of Broca itself.

In the conduction aphasia of Wernicke the lesion which is posited as the cause of the aphasia is one that interrupts the conducting fibres that unite the centre for auditory and articulatory word images. Anatomically such a lesion is generally in the floor of the fissure of Sylvius or in the island of Reil. In this form of aphasia the speech centres themselves are intact. It is only their connections that are interfered with. Thus neither will such a patient have true word deafness nor will there be inability properly to speak out the words; but, as the connection between these two centres is interfered with, the controlling influence which the one, the auditory, has upon the other, the articulatory speech centre, will be lost, and therefore there will be defect in the proper use of words. Wernicke assumes that the images stored in Broca's centre are re-

vived directly by the centre in which primary revival takes place from the object representation. The speech disturbance which such a lesion gives rise to is technically known as paraphasia and occurs in repeating as well as in spontaneous speaking. Patients with this form of aphasia understand and appreciate everything that is said to them. In fact they are responsive to all forms of auditory stimulation, and such auditory stimulation calls up in the natural way the proper auditory image and helps to give rise to the proper percept. On the other hand, the patient is not prevented from attempting to communicate his ideas and thoughts, and the thoughts which are formed for communication are, it is said, the correct ones. When, however, he endeavors to embody these in words the primary revival of the auditory centre cannot send a stimulus to the articulatory centre, because the conduction is interrupted. This being impossible, the motor centre endeavors to do the work unguided and the result is a misuse of words. The patient may himself be cognizant of the shortcomings in his speech and endeavor to correct them, and to convey his meaning by pantomime. These cases are very rare, and when they occur they are to be diagnosed principally by the absence of symptoms pointing to lesions of the individual speech centres. Occasionally paragraphia has been noted in this form of aphasic speech disturbance. When the latter occurs, the explanation that has been suggested is that such patients have been accustomed in writing to transcribe the word which has been revived primarily by the articulatory centre and passed on by the auditory. Thus anything which prevents the association between the last two

causes disturbances in writing analogous to those of paraphasia.

The diagnosis of these cases is led up to more by the absence than by the presence of symptoms. If there are paraphasia and absence of word deafness, and nothing more, the diagnosis of conduction aphasia is said to be justified.

An abstract of the case of Lichtheim, which was reported as one of conduction aphasia, is as follows:

A man, forty-six years old, with incomplete right-side hemiplegia. No history could be obtained. Examination showed that the patient understood spoken, written, and printed speech. The most remarkable feature of the case was paraphasia, which was so great that spoken speech was quite unintelligible. He was aware of the mistakes in his production and tried to assist himself by pantomime. Writing was very imperfect; he disarranged the order of the letters and words, and it was difficult to get him to make efforts of writing. The same defects were manifested in attempting to repeat as when he endeavored to speak voluntarily. He retained the ability to copy. The autopsy showed extensive lesions, the chief one, according to the writer, being of the island and of the floor of the Sylvian fissure.

The extensiveness of the lesion robs this case of the weight it might otherwise have in contributing to a substantiation of the diagnosis of conduction aphasia.

The following is, in brief, the history of a case that Pick has recently recorded as a contribution to the study of insular aphasia:

A woman, Franciska Dillkins, sixty-six years old, had suffered from infancy with epilepsy. Otherwise she

had been well until her sixty-fourth year, when she developed symptoms of insanity; spoke falsely; would not work; was very restless at night, and during the day was absolutely uncommunicative. On admission to the hospital she answered the general questions correctly, in monosyllables, but often made use of the same answer. The countenance was staring and unexpressive, and she made the impression that she did not understand questions and commands addressed to her. After being taken into the hospital, she remained most of the time in bed, and when things were shown to her she took no notice of them or held them unobservingly in the hand. At other times she seemed to recognize things and to name them correctly, *i.e.*, objects presented to the visual and tactile senses. Once she designated money, "motarische Sprache" (the words "motorische Sprache" had been used a short time before). Physical examination showed slight pupillary inequality, incomplete right-side hemiplegia without facial involvement, increased patellar reflex, but no ankle clonus.

Examination.—What is your name? Franziska. Your last name? Franziska. Are you not a Dillkins? Yes. How old are you? Answered correctly and also the question as to her last residence, where she had lived for four years. In response to the question if she had suffered a stroke, she raised the right arm and said: "There is where the stroke affected me." In response to the question if she could read she said: "Formerly I could read but not now." Shown some money and asked what it was, she said: "That is a watch." Is it not money? No. Shown paper money. "That is two gulden." Shown a glass. "That is two gulden." Then she took it in the hand and said: "From that one drinks water." What is it called? "From that one drinks water." Shown a watch. Looked

at it for a long time and then said slowly: "I do not know what that is." When the name was said in her hearing, she said: "Yes, that is a watch." Shown a ring. That is a watch." For what purpose is it? "A watch." Is it a ring? "Yes, that is a ring." Although she was shown the ring immediately afterward she again responded "That is a watch. Shown a knife. "That is a watch." Then she took it in her hand and said in response to the repeated question: "Yes, I know what one does with that," but she gave no evidence that she understood what it is for. A burning match. "That is ein Mäsche (a mesh?)." She did not concern herself when it was brought close to her face. Afterward she made several inarticulate sounds which had no significance for those who heard them. Ability of repetition was markedly impaired, and what she said in endeavoring to repeat could not be understood. Oftentimes she would harp on the last word that was spoken.

Tests for writing and reading were not made, as she said that she could not read (formerly she could). When requested to point to the scissors, she pointed to a key. Almost all of the objects surrounding her she called "A watch." When her attention was called to pictures on the wall she called them "A watch," but apparently was not satisfied and said "Watches."

Soon after this she was seized with general convulsions, which began with conjugate deviation of the head and eyes toward the right. The pupils were widely dilated and reactionless. Immediately after the attack there was deviation of the eyeballs toward the left, and the knee-jerks were not elicitable. Later the knee-jerks were exaggerated. After the convulsive attacks the patient was very mute. The defectiveness of speech became more apparent. The hemiparesis was followed by profound

hemicontracture. After another convulsive attack she developed an extensive bronchitis and soon died.

Autopsy.—The left hemisphere (opened after the method of Pitres) showed in the lenticular nucleus, extending so as to involve the claustrum and the internal capsule, an old focus of softening of the size of a walnut. The microscopical examination showed that the entire island in its total transverse diameter was interrupted by the focus.

In conclusion Pick remarks that he does not offer this as a pure case of insular aphasia, because of the fact there were other changes, including a universal atrophy of the brain. In spite of this he says the case may be cited as a "negative case," particularly in reference to the patient's capacity to repeat. These cases are both so incompletely reported clinically and anatomically that it is unwise to attempt criticism of them.

APPENDIX II.

A Case of Articulatory-Kinæsthetic Aphasia.

In the text only as many cases were cited as was thought absolutely necessary to illustrate different types of aphasia. The clinical findings in the patient whose case I am about to relate mirror so completely, however, the exact conditions found in true cortical motor aphasia, articulatory-kinæsthetic aphasia, that I am prompted to make brief note of them here. Moreover, I believe that the case is of no small importance in substantiating the claim made in the text, that Broca's convolution sends no projection fibres directly to the internal capsule or to the capsular irradiation, and that it is in reality a sensory area.

Mrs. X——, a widow, sixty-three years old, the mother of eight children, has had a vigorous, active life, free from ill health, save that twelve years ago she suffered severely from attacks of renal calculi. During the past year or two she has complained of indigestion and more recently of a dull, aching sensation in the back of the head and neck, with occasional attacks of very severe pain in the left temple. For a few weeks previous to the beginning of her present symptoms she suffered from insomnia, from irritability, nervousness, and forgetfulness. Her son, a physician, gives the following account of the onset of her aphasic symptoms. One week before consulting me she discovered, while making a call, that her speech had be-

come, without warning, very much embarrassed. She could not finish the sentence that she had started to speak. She forgot what she wanted to say. She chafed under this impotence and got very much excited. She returned home in a street car, and was much astonished to discover on looking at the signs with which the cars are lined that she was quite unable to comprehend their signification. She could see the letters and words, she knew that they were letters and words, but they conveyed no meaning to her. When she got home she tried to tell her family about her disability, but was able to say only a few words, and these were entirely disconnected. After trying to speak for a time she became excited and began to cry. On the following day, when she awakened, she could say only "Yes" or "No," but as the day wore on her vocabulary became somewhat larger. It was particularly remarked that when she was excited or very emotional sometimes words would flow out of her mouth in an astonishing manner. From that time until I saw her there had not been very much change in her capacity for speech production.

The following is a stenographic report of the examination to determine disorder of voluntary speech. In response to the question to tell me all that she could concerning the onset and course of her symptoms, she said:

"Well, mem-mem—three weeks, m-m-em—feel-m-em-em—sometimes [prolonged pause, seems to be thinking] couldn't thought—no thought—forget—but—eh—last Friday [another prolonged pause] am—no—noticed they—I couldn't—eh—I—[prolonged pause] I couldn't tell, am, I don't, I can't, can't express [explosively] I can't tell—I cannot [points to her head and looks weary]. It seems, I can't, last Monday, con-con-nects—sentence, two or three

words—gone. Was—gone, blank, didn't know. Can't think, was gone, forget—forget everything. Couldn't, couldn't, can't."

To test her capacity to repeat, I asked her to say after me: "I stood on the bridge at midnight." Her reply was:

"I stood—the—night," said with great effort, and with apparent endeavor to repeat each word as quickly as they fell from my lips.

"Still sits the schoolhouse by the road?"

"Forget—yes—the—the—s' s' s' forget—road."

"Waterloo was a battle of the first class, won by a captain of the second."

"The bat-tle, ah, me, ah me, ah."

It is particularly noticeable that when I speak she endeavors to say the words after me very rapidly one word after another, but it is quite imposible for her to repeat more than a word or two. The patient is an English lady who formerly was able to speak German very fluently, but when I recite the first verse of Schiller's "Bell," beginning, "Fest gemauert in der Erden," etc., she is not able to repeat a word of it. I then ask her to repeat the Lord's Prayer. She assures me by nod of the head that she cannot do so, but when encouraged to try she says: "Fa' a' ther—our fa——" [gets excited and I believe tries to convey to me that she was unable to repeat it last evening]. I then ask her to say it to herself. She again indicates that it is entirely impossible. I ask her if it is impossible, and she says "Yes." It is interesing to note that when I encourage her to say it to herself after I have told her that I am going to say it in my internal language to determine if we reach the end simultaneously, she adopts the conventional attitude and manner, probably thinking that they will prompt the recalcitrant words, but

all to no purpose. She is quite unable to read, either in a loud voice or to herself, although she can say a word of what she reads here and there, but words and sentences convey no meaning to her. She takes up the newspaper, cons it carefully, then puts it down with an expression of dissatisfaction and disgust. In other words, there are manifest verbal blindness and profound alteration of mental images. There is no trace of hemianopsia.

When I request the patient to write her name, she does so promptly. When I ask her to write the name of her son, she does so; likewise the street and number where she resides. She is absolutely unable to write spontaneously. Her capacity to write from dictation is tested by asking her to write, "When in the course of human events," but she is absolutely unable to do so. The only word that is produced after numerous attempts and repetitions of the sentence is the word "When."

Writing from copy is done without trace of hesitation or error; and when she is asked to copy printed letters in writing she does so with great readiness.

She comprehends spoken speech, but oftentimes it is necessary to repeat before the meaning of what is said fully dawns upon her. In other words, although there is no word deafness, there seems to be some difficulty in calling up auditory images quickly and readily.

She has no trace of hemiplegia, unless we call a slight asymmetry of the angles of the mouth an indication of defective cortical innervation, as the right angle of the mouth seems to be a trifle lower than the left. There is no ataxia or inco-ordination of the extremities; the knee-jerks are lively and of equal intensity on both sides; the pupils react to light and shadow; there is no tone deafness or object blindness. The urine contains albumin

and casts; the pulse is regular, the arteries are hard and incompressible, and the second sound of the heart is very much accentuated. In other words, she has extensive arterio-capillary fibrosis.

The interpretation which I put upon the case is as follows: Pathological diagnosis, general arterio-capillary fibrosis, with consequent encephalomalacia of Broca's convolution. Clinical diagnosis, true cortical motor aphasia, articulatory-kinæsthetic aphasia. I need not repeat that the elicitable symptoms parallelize in every detail those which have been proven to be typical of this form of aphasia. The chief deficiency of internal language seems to be an inability to evoke the articulatory-kinæsthetic images of the word, and this constitutes a gap in the circuit of internal-speech impulses. No more illustrative case could be cited to show that spontaneous writing and writing from dictation are disordered commensurately with voluntary speech in this form of aphasia. Moreover, the case shows with uncommon clearness that a striking degree of verbal blindness occurs with cortical motor aphasia. It is probable that in time this at present manifest verbal blindness will become latent, and, if the pathological process on which the symptoms are dependent does not progress, that a future careless examination might fail to reveal it.

In the text I have stated that hemiplegia is almost constantly associated with cortical motor aphasia, and that it is dependent upon extension of the lesion to the Rolandic cortex. This case is an exception to the rule, and I venture to believe that it is an important exception, going to demonstrate that Broca's convolution has no direct repre-

sentation in the capsular irradiation, and finally that the products of its activity are not sent directly to be externalized, but are sent to the Rolandic representation of the speech musculature and to the area of representation for other modes of language communication.

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