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. 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 look upon 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, 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.

Motor Aphasia.—(1) Cortical motor aphasia; articulatory kinæsthetic aphasia. (2) Subcortical motor aphasia.

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 coordination 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 movements 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.

There are two divisions of motor aphasia, cortical and subcortical. 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 works on the subject. Lichtheim suggested a 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 it was first suggested and utilized by Proust in 1879. To test this deficiency of internal language, Proust and Lichtheim showed that it sufficed to ask the patient who had heard some polysyllabic word, 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; 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.

The lesion that causes motor aphasia is in the great majority of 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 cooperation 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 coordinate 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 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.

Associated with this loss of spontaneous speech, due to the destruction of the sensory images of articulation stored up in the foot of the third frontal convolution, 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 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 the 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 supplies 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 articulatory images.

The capacity for articulate expression which a few (compared with the entire number) 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.

Another very striking variety of articulate speech which patients with motor aphasia show is that to which the term lalling or lallation is applied.

Although in cortical motor aphasia the power to make voluntary expression is usually entirely gone, the loss may be partial. When partial, the power of expression is limited, as a rule, to one or more monosyllabic words. 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 expressions 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. Such recurring utterances are distinctive features of cortical motor aphasia, and not of the subcortical variety.

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.

A patient with cortical motor aphasia is unable to express his thoughts in writing. His incapacity 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. 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. 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.

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, use the term "fascicular anarthria" to indicate the same condition.

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 is lost in cortical motor aphasia proportionately to the completeness of the aphasia; and, secondly, the patient responds to the Proust-Lichtheim test; thus proving 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.

When the lesion that causes subcortical motor aphasia is situated at a level as low as the internal capsule, then a diagnosis can often be made positively, not alone from the employment of the Proust-Lichtheim test, but from study of the difficulty 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 coordinate 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.

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 lesion 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 great 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. In the cortical form 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, abscesses, foci of inflammation, or other conditions which slowly destroy Broca's area.

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 is applied to those cases in which imperfections of language, disability or inability to speak, are due to interference with the reception 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 as such I shall describe it.

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.

Sensory aphasia possesses certain general features which 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 subsequent to 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 develops 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 lalling, and of echoing, down to absolute mutism as complete as that produced by total destruction of the articulatory kinæsthetic area.

Patients with sensory aphasia are 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.

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 farged into one, the syllables of which may be articulated, but the words have no similarity to words as usually spoken.

Not secondary in importance to the information that may be obtained of sensory aphasia 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 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, and with 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. If the patient has destruction of the angular gyrus, there will be practically total agraphia. 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.

The power to write 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, and makes an exact reproduction of what is before him.

In *auditory aphasia* 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 independently, being frequently associated with some degree of visual aphasia or motor aphasia.

The seat of the lesion which causes auditory aphasia is 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.

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 of 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.

In the milder forms of auditory aphasia it is often necessary, in order to estimate correctly the degree of word deafness, to test the patient carefully and repeatedly.

The concomitant accompaniments of word deafness are inability to write from dictation, defective comprehension of what is read, imperfect writing, paragraphia, etc. The first needs no explanation. The power of 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. He is quiet and observant; his glance betrays suspicion or fear, and his demeanor is often one of trouble and unrest. This change of demeanor and manner, 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.

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.

There must necessarily be as many forms of auditory aphasia as there are distinctive symbolic sounds. Spoken speech is the most highly symbolic, the next most differentiated to music. To the form of aphasia in which there is deafness for musical notes the designation tone deafness (musical deafness) is given. 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.

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 have some clinical independence.

Visual Aphasia; Verbal Blindness; Word Blindness.—This is a form of aphasia in which there is a loss of the significance of written or printed words, although the

words themselves can be seen with customary distinctness. In the form of aphasia described as 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 greatest 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 graphic 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, hieroglyphs, 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.

The visual area is in the posterior end of the brain. It is made up of two more or less distinct centres: a visual perceptive centre and a centre in which is stored the visual memory of words and other symbols. The former is situated on the mesial surface of the occipital lobes in the environmental 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 is 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. 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 there is no lesion of the peripheral visual apparatus, although the condition known as homonymous hemianopsia oftentimes exists.

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 into syllables (asyllabia).

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. 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. Occasionally cases are met with in which the verbal blindness is so very slight that it requires careful and persistent examination to reveal it.

If the angular gyrus is completely destroyed, the faculty of writing is lost with it. 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. 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.

Patients with word blindness are sometimes able to read written or printed words and sentences by tracing the word with the end of the index finger or with a pencil. 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 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.

There are two or three subdivisions of visual aphasia, to which I shall refer. The first is the 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, 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.

The lesion which produces such a condition is 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.

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. The lesion interrupts the pathways that unite the seat of cortical visual representation and the seat of cortical auditory memories.

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 blindness 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.

The condition known as apraxia—the inability to comprehend the usage of ordinary objects and things to which one has been accustomed—is analogous to this. The nature of the apraxia 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.

The subcortical forms of sensory aphasia, pure sensory aphasia of Dejerine, are analogous to the subcortical forms of motor aphasia. 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