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.

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 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.

Spontaneous speech, except occasionally the slight paraphrasia already referred to, and capacity to repeat are intact. The patient is able to write voluntarily and from dictation, but he cannot read what has been 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 accompanied 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.

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. 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, every constituent of internal language is intact. It is rarely if ever associated with paralysis of the extremities.

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 which 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.

Diagnosis.—The first step in attempting the diagnosis of aphasia is a simple method of eliciting and associating the different symptomatic constituents. It should be kept in mind that the speech faculty 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, by pantomime, and that integrity of the receptive side of language is commensurate with the interpretation of visual and auditory stimuli.

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. 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, of the patient with visual aphasia. 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 striking. This intensity 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.

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 mental 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 should 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 corresponding memories? This can be done ordinarily by asking some simple question, 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 and 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 noticing 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 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. As 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 aërial 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 made 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 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 is no such disturbance, the lesion is elsewhere than in the zone of language. In some patients the differentiation will be an easy one. 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 patients have not sufficient scholarship to know anything of syllables or word construction. 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 often 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 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 aphasia 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, easily, and correctly by writing and by pantomime, or, at least, he would be if it were not that the right half of the body is usually paralyzed, and he is obliged to portray mental states by the pantomime 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. 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, 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 Lichtheim, 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 motor and sensory aphasia, and if word deafness has been excluded there will be no difficulty in determining this inability, which is coexistent 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.

After having tested the patient's capacity to perceive and interpret words through the auditory apparatus, he should be examined with a 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 apparatus. This can be done by an ophthalmoscopic examination. Tests should 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 one 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.

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 uses or purposes, 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 the former, internal language will be preserved and spontaneous speech may be intact, although there is usually some paraphrasia 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 the aphasia be of the latter character and complete, the patient will be absolutely agraphic. This agraphia is to be considered a part of the disorder of internal language; there is inability to arouse the visual image of the word. In such a case, an arousal must precede the transmission to the part of the Rolandic cortex that innervates the member holding the pen; there is complete agraphia.

The physician then proceeds to examine whether the patient has word blindness; that is, whether the patient can read (1) print, (2) script, (3) figures and other forms of notation.

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 to receive any information from him.

Etiology.—Etiologically, aphasia may be classified into organic and dynamic. The principal organic forms are due to rupture of the blood-vessels and occlusion of their calibres, whether from embolus or from 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.

Under the dynamic forms may be included those in which no organic lesion is responsible for the development of aphasia 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 some 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 toxæmia, 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 which 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 such time of life.

Treatment.—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 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 in the subcortical speech tracts, and these can be diagnosticated as such, medicinal treatment is of the greatest value. 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 are, 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.

It would be a work of supererogation to repeat in detail the causal treatment applicable to the different forms 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, the treatment in the beginning of an uræmic attack 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 autochthonous 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 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.

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.

Taking it all in all, the question of the medicinal treatment of aphasia never comes up for consideration The question that does present itself 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 section 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. Despite this many of the dynamic or "functional" aphasias yield to appropriate medication for the conditions upon

which they are dependent, combined with fitting treatment addressed to the mental, moral, and physical sides of the individual.

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 becomes aphasic, even though the lesion be a very severe and extensive one, the faculty of speech may still be 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, has been completely destroyed, the patient may occasionally regain some capacity to speak single words or a number of words. Various hypotheses have been formulated to explain these occurrences, the most widely accepted apparently being that of J. Hughlings 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. 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 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 corresponding 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. In the process of functional compensation the portion of the speech centre that is not destroyed becomes connected with the other speech centres in previously unaccustomed ways, not by the development of new commissural fibres, but through their acquisition of functional activity. Secondly, the opposite hemisphere, the one that has the zone of language ontogenetically developed, is not an uneducated hemisphere at all, but 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 is 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 on it an impression similar to that which they do on the so-called "educated" hemisphere. These impressions are bilateral in reception but unilateral in interpretation. This unity 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 automatism, or an unfinished automatism, can be assumed by the opposite hemisphere, 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. A number of the cases that have been reported to show the assumption of speech function by the opposite hemisphere have 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 internal language and speech by forcing the educated opposite side to supply a centre similar to that which has been destroyed; or, if the damaged centre is not entirely destroyed, by re-educating the cells that remain, assisting them as it were in the acquisition of a function which they were intended to perform in unison with other cells. For example, if the articulatory kinæsthetic centre is destroyed, 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. 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 how to arrange the organs of articulation for the production of simple vowel and consonant sounds, a performance which he should be daily encouraged to do. After he has acquired the capacity to produce these sounds and has regained some control of the peripheral speech mechanism, he is to be tutored in the same way in the production and articulation of monosyllables and their combination in words. This process is a laborious one and requires great perseverance on the part of the physician. Whenever possible the task should be entrusted to a teacher. Naturally the greatest progress will be made with cases of subcortical motor aphasia, because these patients take a more intelligent interest in the matter and because they can aid themselves by reading and writing. Most of the published cases of marked functional compensation have been cases of subcortical motor aphasia, in which ability to read has been preserved.

When the auditory centre is diseased, then the object of teaching is either to get a primary revival of the idea of words in the visual or the articulatory centre, and then to throw into the circuit the component parts of the auditory that are not disorganized, or to favor the development of the auditory word centre in the opposite hemisphere. The patient must be taught to concentrate his attention on vowel sounds and then on words of one syllable, spelt letter by letter, while he tries to repeat them by the oral method. 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 left stranded, from a speech standpoint. The plan of education is in reality that which is used for deaf-mutes who are taught