EPITOME OF AGNOSIA, APRAXIA, AND APHASIA WITH PROPOSED PHYSIOLOGIC-ANATOMIC NOMENCLATURE

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The nomenclature of agnosia, apraxia, and aphasia has evolved in the manner of a straggling city and for the same reason, namely that independent workers have taken different viewpoints and have become interested in one phase of the subject at a time. The terminology is in some instances anatomic, in other physiologic, in still other psychologic. Certain terms have even arisen in the minds of pedants who have constructed hypothetical diagrams on the basis of which they have invented anticipated types of clinical manifestations which were named before they were observed.

Just as grammarians arise in a nation and systematize terminology to bring intellectual order out of the chaos of human expressions, so have organizers arisen in the study of aphasia. Their efforts have been laudable but unfortunately premature because of lack of anatomic-physiologic background for their conclusions. Kinnier Wilson stated in his excellent monograph of 1926 that not only was the subject not understood but the approach was not agreed upon. He envisioned an anatomic-physiologic nomenclature as the ideal to be striven for on the grounds that a psychologic terminology was too destructive to the subject of cerebral localization to be acceptable.

Through the great revival of interest in the subject of aphasia due largely to the enormous compilation and intensive study of Henschen and the immediately subsequent invaluable contributions of neurologic surgeons, notably Foerster, Penfield, McKenzie, Gardner, German and others, so much information has accumulated during the last 20 years that Wilson's ideal seems about to be attained.

It is with considerable trepidation and fear of being considered presumptuous that I offer the present system of nomenclature. However, a study of the system will immediately make it evident that I am presenting exceedingly little by way of original terminology; I am rather organizing present knowledge. The terms agnosia, apraxia, and aphasia can be retained with slightly modified definitions. The greatest single change suggested is one of the concept of the function of the minor cerebral hemisphere in language. Even this concept is far from new as it was suggested by that great master Hughlings Jackson and elaborated considerably by Henschen. Potzl's work in localization and Kleist's intensive studies are landmarks worthy of careful study.

The Functions of the Minor Cerebral Hemisphere

It is customary to speak of the two cerebral hemispheres as being dominant and recessive, respectively. The terms are based on the conception that the one ordinarily takes the lead and leaves little or nothing

for the other to do in the associative sphere of activity. It is tacitly understood that dominance does not apply to projection fiber systems or to the basal ganglia.

The term *dominance* or *dominant hemisphere* is expressive but inaccurate. The studies of many excellent authors on thousands of cases have shown that the relation of the so-called dominant hemisphere to the recessive is such that it can be much more accurately represented by the relationship of pilot and co-pilot on an airliner. In the beginning of their associated careers the pilot is slightly more expert than the co-pilot, but in case of sudden incapacity of the pilot the co-pilot can manage the ship almost equally well. The pilot does not dominate the co-pilot. I have suggested the use of the terms major and minor hemispheres as being more appropriate than dominant and recessive hemispheres because they suggest merely "more" and "less." The terms major and minor are already in wide use in industrial medicine relative to hands, feet and eyes.

There is, however, an important discrepancy between the two relationships in our simile (the pilot-co-pilot relationship and the two-hemisphere relationship) which must be clarified. The pilot ordinarily turns over the management of the ship to the co-pilot during at least a short portion of each flight and the co-pilot therefore remains proficient. The major hemisphere ordinarily never turns over its functions to the minor but instead becomes more and more proficient while the minor misses opportunity for further training. It is capable, however, of rapid training when the necessity arises if the patient (and the minor hemisphere) are not too old and if they are in good general condition.

So far as the *language* function of the brain is concerned, another simile is much more accurate. Consider a hypothetical situation of a group of men traveling together through a foreign country. Only one, the leader, has studied the language of the foreign country and is expert in it. The followers have not studied it. The group travels for some months, when suddenly the leader is killed. The proficiency of the followers to understand and speak for themselves will depend entirely on their individual independence and initiative. Some followers will have learned nothing, some will have acquired a short vocabulary and will have learned to understand a few simple phrases. Others will have read, listened, and spoken for practice with a view to preparedness. A very few will have become proficient.

That is the case with the major and minor cerebral hemispheres. They start through the world together almost equally incompetent. In some instances, when the major is destroyed the minor hemisphere will have developed its engrams so crudely that it does not function at all. In other instances it will be able to formulate language slightly, understand a little immediately and improve rapidly with training. In a very few instances it will have established its engrams so well that when the major hemisphere is suddenly destroyed, it will be able to assume the functions of language.

When, in the above simile, minor cerebral hemispheres were compared with individuals they were actually compared with major cerebral hemispheres, because the individuals in the illustrations were using

their major hemispheres. While there is a hereditary determinant to make a certain hemisphere the major one, there is no essential difference between the functional capacity of major and minor hemispheres at birth. This has been shown repeatedly by instances of severe trauma to the major hemisphere early in life. In such cases the minor hemisphere invariably takes over the function of the major with such precision and dexterity that observers do not notice any disturbance of language. Handedness does not develop until the age of about nine months to a year. Even when it begins to manifest itself the superiority of the major does not appear great. This is proved by the fact that injury to the major hemisphere at the age of four or five years causes only transient aphasia. Whenever a minor hemisphere is called upon to take over a function it at first fatigues with astonishing rapidity. As it becomes trained it develops endurance rather than specific capacity.

"Majority" and Handedness

The common hereditary tendency to right-handedness is actually a tendency to left-brainedness; the brainedness comes first. The handedness can be shifted by training early in life but the brainedness, as a unit, does not shift with it. Well-meaning mentors frequently change left-handed individuals to right-handedness at so early a time in life that the subject may not be aware of it at all. This accounts for some instances of right-brainedness in "right-handed" persons. Aside from these occasions, however, there are instances of left-brainedness in left-handed persons. Ipsilaterality of handedness and brainedness occurs, according to the figures of Chesher, in about six per cent of all persons. Hereditary right-brainedness, as shown by the fallible index of left-handedness, occurs in about ten per cent of all persons. Because of these discrepancies between handedness and brainedness it is never safe to lateralize a cerebral lesion on the basis of aphasia alone. Unequivocal lateralizing signs based on projection fiber systems must be relied upon.

Up to this point, to facilitate clarity, the discussion of function of the minor cerebral hemisphere has centered about it as though it were a unit. Extensive research, however, has shown that the following subdivisions must be considered separately:

(1) Prefrontal lobes.
(2) The frontal writing center (foot of the second frontal convolution).
(3) Broca's convolution.
(4) Pars triangularis of the third frontal convolution.
(5) Anterior end of the superior temporal convolution.
(6) Wernicke's area (posterior third of the superior temporal convolution).
(7) Area 37 of Brodmann.
(8) Angular gyrus (area 39 of Brodmann).
(9) Area 18 of Brodmann.
(10) Area 19 of Brodmann.
(11) Convolutions of Gratiolet.

These eleven areas are all association areas, i.e. they are not areas of cortex concerned immediately with reception or transmission of impulses over the projection fiber tracts. The eleven areas occur in pairs, of course, but the degree to which the major in each case is superior to the minor is not the same for all pairs. Disregarding for the present the exceptional cases one may state the case for each area in the average person as follows:

The prefrontal lobes, so far as their intellectual functions are concerned, either establish no unilateral superiority or establish it to a small degree. They seem at times to divide the various functions of the lobes between them, this being manifest by the appearance of certain symptoms regardless of which frontal lobe is affected. But in any case either right or left prefrontal lobe seems capable of assuming the necessary function of both after a short period of training (1).

Area 18 of Brodmann (area parastriata) is concerned with recognition of objects and pictures. The evidence available at present indicates strongly that either the right or the left becomes the major during the first year of life and that the selection is purely a matter of chance. In most persons the degree to which either becomes the major is relatively slight and destruction of the major one leads to only temporary disability of recognition of objects. In a few persons (relatively speaking) the degree of superiority of one becomes so great that its destruction leads to visual agnosia for objects lasting for years. In most instances the major area 18 is ipsilateral to the major temporal lobe (when the major temporal lobe is determined by language function). In a few instances the right remains the major because of an early fixation even though the major temporal lobe becomes established on the left side (2).

Area 19 of Brodmann (area peristriata) is concerned with revisualization of former images. It follows the rules of lateral superiority described as governing area 18 (2).

The three groups described (prefrontal lobe, area 18 and area 19) are not concerned with language and this fact probably underlies the weakness of unilateral superiority. There is much evidence that the superiority of the occipital lobes actually shifts, when it does shift, with the establishment of language centers (second year of life). The lowest function which can be called language is music. It will be seen as we proceed that the areas concerned with vocal, instrumental, and auditory music perform their function almost equally well on the two sides.

The *pars triangularis of the third frontal convolution* (area 45 of Brodmann) is concerned with all of the functions of vocal music and the playing of musical instruments. Injury to it causes apraxia for these acts if it causes any symptom. But it has been abundantly verified that the minor side when called upon takes over the function with great ease and that in only rare instances is there any functional defect after injury to only one side (2c).

The *anterior extremity of the superior temporal convolution* (area 38 of Brodmann) is concerned with musical auditory recognition and interpretation. It receives impulses from the transverse gyri of Heschl which act as centers of primary auditory perception. Destruction of

area 38 of either side usually produces no disturbance of musical comprehension because the other side takes over the function. Destruction of the area bilaterally destroys the auditory musical sense (2c).

Wernicke's area (areas 41 and 42 of Brodmann) is concerned with interpretation of spoken language. Comprehension of spoken language is next in scale above comprehension of music and in harmony with this fact the degree to which the major side ranks above the minor is next in the scale. After removal of the major temporal lobe the average patient is able to comprehend about five or six questions addressed to him. He (his minor temporal lobe) then fatigues. After a year he usually understands a great deal of what is said and after two years one can as a rule hardly tell in ordinary conversation that anything has been removed. The right has assumed the function (3).

Area 37 of Brodmann is concerned with formulation of language. This function is really the one which has been called internal language. Destruction of area 37, or in some cases even relatively slight damage to it, results in inability of the patient to find his words and formulate his sentences. The degree to which the major side is superior is almost identical with that described for the area of Wernicke (4).

Formulation of language is a complex cerebral function dependent on several subsidiary functions. An area of cerebral cortex essentially identical in extent with area 37 of Brodmann is of the utmost importance for formulation of language, either written or spoken. Difficulty with word finding occurs in some cases of angular gyrus lesions, in perhaps one-half of the instances. The same difficulty occurs in a few instances of lesion of the posterior portion of the superior temporal convolution, i.e. when the lesion is behind Wernicke's area. Lesions of Wernicke's area itself interfere with formulation of language to such an extent (as a rule) that the patient does not speak.

These facts explain the views of Kleist that reauditorization of a word is necessary to formulation of language. He calls area 37 of Brodmann an acoustic-psychic sphere.

Kleist states that the word sound is not simply apperceived acoustically and then immediately connected with the concept of the object, but it first appears in consciousness. He says that something is meant with that speech sound, that it designates something without the individual's becoming conscious of what specific object is meant. While all this is true, there is nevertheless much more to the determination of the meaning of a word than the process described above. Some words cannot be distinguished by their sound alone or by their appearance alone. Some words alter their meaning according to context. The expression "they read" cannot be pronounced until context is added. We need to know whether the tense of the verb is present or past. And one cannot distinguish between "red" and "read" by the sound alone. The endless punning heard in entertainment is possible because of these peculiarities (imperfections) of language.

Because of these facts I cannot conceive of area 37 of Brodmann as being merely an acoustic-psychic sphere. It is an area in which knowledge is coordinated for significance of words and expressions and in which grammar, rhetoric, and syntax are considered in the formulation

of language. The area is in constant association with all of the other areas concerned with language and its separation from the other areas by organic lesions may give rise to defects of formulation of language.

Lesions of this area provoke as their most constant symptom amnesic aphasia. The area for formulation of language is chiefly area 37 of Brodmann but this area is so trained by association with Broca's convolution, Wernicke's area, and the angular gyrus that in some instances a lesion of one of these cripples the function of area 37 sufficiently to cause it to "resign" and turn over the function of formulation of language to the identical area of the minor side. The result is amnesic aphasia. I believe that this disturbance should be called formulation aphasia. (The term "formulation" need not be negative so long as it is followed by "aphasia.") Whether misuse of words, use of wrong words (commonly called paraphasia), or the more severe forms called jargon aphasia or agramatism result depends on the degree to which the minor side takes up the function. The minor side is usually capable of assuming to some degree the function of formulation of language, but, until it has had time for training, it functions imperfectly. The most pronounced defect is seen in loss of ability to associate objects and their names.

Broca's convolution contains engrams representing the physiologic basis of memory of how to use the vocal organs for the production of words. These engrams are formed simultaneously with, or very shortly after, those of Wernicke's area. The major is superior to the minor in about the same degree as already stated for Wernicke's area, i.e. far superior. Destruction of it in adulthood leaves the patient, in the great majority of cases, able to say only a few words, which he uses for all responses. Usually several years are required for training of the minor side; but as a rule, if the general cerebral function is of good quality, the minor side becomes nearly as proficient as the major had been (8).

The convolutions of Gratiolet, located between the angular gyrus and the second occipital convolution (zone between areas 19 and 39 of Brodmann) are concerned with the body scheme. As the naming of parts of the body, especially naming of fingers and designation of laterality, includes phases of language, disturbances of the body scheme follow the rules for language areas rather than those for the non-language areas regarding laterality. The major is superior to about the degree described for the area of Wernicke, i.e. greatly superior, as the syndrome occurs with great regularity from lesions of the major side (5).

The frontal writing center and the *angular gyrus* are concerned with the most highly specialized of language functions, emission and reception, respectively, of written and printed language. The writing center at the foot of the second frontal convolution is essential to the memory of handwriting movements. In the majority of cases it cannot function without close association with the convolution of Broca. It cannot, of course, function in writing unless the language formulation area (area 37 of Brodmann) and the area of revisualization of the written word (angular gyrus) are intact and in communication with it. The major writing center is far superior to the minor in most cases. A long period of training is usually necessary for the minor to function

at all well. Unless the destruction of the major occurs early in life, the minor rarely attains a degree of perfection comparable to that which the major had enjoyed. Conversion of a left-handed person to right-handedness for writing establishes a writing center on the minor side (6).

The *angular gyrus* has a double function. Its cortex contains patterns of two types, one for recognition, the other for revisualization of the written and printed word. The patterns of both types are spread diffusely over the cortex and the superiority of the major is great. The minor angular gyrus in most instances has its engrams so crudely made that many years are required, after destruction of the major one in adulthood, for it to perform at all well. Unless the major is destroyed early in life, the minor rarely attains a degree of perfection comparable to that of the major.

The angular gyrus is trained in very different ways in different persons and consequently its degree of ultimate independence varies enormously. When a child learns to read he at first associates very intimately the engrams for the enunciated word (for the word spoken by himself) with the auditory memory engram of the word which he reads. This is obvious when one recalls that the child reads aloud; he *says* the word and he *hears* himself say it. During the formative period this association of the engrams of the angular gyrus with those of Broca's convolution is so intimate that a lesion of Broca's convolution prevents the child from even *recognizing* the written or printed word. If his training in reading does not advance beyond the point of enunciating (or at least retaining a habit of lip movements while reading) the angular gyrus never becomes independent of Broca's convolution. If the child is taught to read by simultaneous writing of the word the angular gryus may become and may remain dependent on the frontal writing center also. In such cases alexia results later in life from a frontal lesion. However, in the majority of educated persons the angular gryus develops, by silent reading, an essential independence of the centers in the frontal lobe.

The situation is somewhat different regarding the continued dependence of the angular gyrus function on the area of Wernicke. In the area of Wernicke one "auditorizes" the word one is reading and this habit in most instances continues throughout life. For this reason, at any time of life,, in the majority of cases, destruction of Wernicke's area, or destruction of the short cortical area between the angular gyrus and Wernicke's area, produces alexia by preventing not only interpretation but also recognition of the written or printed word. It is because of the elaborateness of the angular gyrus connections that the major becomes so strongly superior to the minor and that training of the minor, when the necessity arises, is extremely difficult (7).

In addition to these important facts about the functional capacity of the various cortical areas of the minor side there is another principle which has not been enunciated, but which is becoming more and more evident and which is of the utmost importance to the understanding of localization in agnosia, apraxia, and aphasia. This is the principle that a given area of cortex concerned with language may, after a

minor lesion in it, either attempt to carry on its function, though crippled, or resign immediately. The question whether it continues to function after it is crippled or resigns depends on the facility with which the corresponding minor area can assume the function. Thus a strongly superior language formulation area (area 37 of Brodmann) may continue to function, though imperfectly, after a minor lesion, while the same area in another case with a similar lesion may resign and turn over the function to the minor area. There is no absolute criterion by which an examiner can determine which has happened in a given case. In the majority of cases the damaged area resigns its job. It is for this reason that the brain of a person who has been unable to formulate his sentences may show a tiny lesion in the area concerned or it may show a complete destruction of the area. The whole matter resolves itself into this question, "Can the crippled major area perform with greater ease than the normal but untrained minor area?" The individual in question will take the easiest course. With these facts before us we can see that a positive agnostic or aphasic symptom has a positive value in diagnosis but a negative one has little if any value. If the projection fiber signs and symptoms indicate that an area concerned with aphasia is destroyed, but there is no aphasia, the negative finding of absence of aphasia has little if any weight in determining the extent of the lesion.

We now come to the question, "How complete must the defect be to constitute agnosia or aphasia?" If, following a lesion of the convolution of Broca, a person is able to speak but with stammering and hesitancy, some students might say that he does not have Broca's aphasia. But we know that from the standpoint of cerebral localization he does have a lesion and that the reason for his being able to talk at all is either that the crippled area is performing or the imperfectly trained minor convolution of Broca is doing the work which we observe. We should therefore say that the patient does have aphasia (apraxia of speech). If the patient, after such a lesion, speaks as well as he did before he either had two equally capable convolutions of Broca or it was the minor one which was destroyed, the minor one merely having been located on the side where we expected the major one to be. Our misinterpretation is due to the erroneous assumption that handedness is an infallible guide to brainedness.

We thus arrive at the principle that a disturbed function of language has the same weight in cerebral localization as a complete loss of the function. A corollary of this is that in the study of human cerebral function in the associative field (by the use of clinicopathologic material to which we are limited) it is impossible to determine the function of any area unless we confine our studies to *bilateral* lesions of the area in question. If this principle had been recognized earlier, millions of words could have been omitted from the scientific literature on aphasia and allied branches. If a given area is bilaterally destroyed we are certain of a functional defect. The determination of the functional defect by a clinical study is not difficult in the light of present knowledge.

Apraxia

The function of the minor cerebral hemisphere in acts not pertaining to language is far greater than in language functions because handedness and brainedness are largely governed by the development of language. It is for this reason that apraxia is far rarer in the non-language sphere than in the apractic aphasias.

In the precentral gyrus are located patterns of movement, physiologic patterns for coordinated motor performance. Physiologically speaking, on a par with these patterns are many others. Loss of function of these patterns has been called kinetic apraxia of the limb (limb-kinetic apraxia of Liepmann). I have suggested the term cortical motor pattern apraxia because in many instances the limbs are not involved. Illustrations of this type of apraxia are: Broca's aphasia, dysarthria and dysphagia resulting from a lesion of the precentral gyrus of the mouth area, paralysis of voluntary lateral gaze with preserved reflex lateral gaze resulting from a lesion of the foot of the second frontal convolution, apractic agraphia resulting from a lesion of the frontal writing center, loss of finger movement with preservation of use of the upper limb as a whole resulting from a lesion of the precentral gyrus, avocalia (of Henschen) from a lesion of the pars triangularis of the third frontal convolution, and probably a few other instances of disorganization of movement resulting from lesions of areas 6-a and 5-a. In all forms pertaining to language unilateral dominance is relatively strong; in the others dominance in most persons plays a minor role.

These various centers of performance of *movements* are in turn governed for the performance of *acts*. An arbitrary differentiation must be made between movements and acts, the latter consisting of a series of the former coordinated to the carrying out of a plan of action. There is no "center" for carrying out an act. Loss of the power to do so (in the absence of paralysis, ataxia, or dementia) is ideokinetic apraxia. Lesions of the corpus callosum or deep lesions of the major supramarginal gyrus are most potent but in any case we are dealing with a disturbance of associative function and not a cortical lesion.

There is relatively slight dominance of one hemisphere over the other in ideokinetic apraxia for which reason the condition is rare relative to aphasia and usually transient. Surgical severance of the corpus callosum does not cause apraxia. The ideational plan of action can usually be formed in either hemisphere for both lateral sets of limbs. Ideokinetic apraxia represents physiologically an interruption between the ideational plan and the patterns for execution of an act.

When the ideational plan itself is disturbed, i.e. when an ideational plan cannot be kept in consciousness long enough for its execution, the condition is called ideational apraxia. This condition is always due to a diffuse cerebral lesion, quite often a toxemia.

Selection of Terms

The reasons for the choice of each term will be much clarified by development of the physiology in brief and simple language. The function of area striata has been designated as *primary perception*. While it is true that there is no perception in the usual sense until the impulse

reaches consciousness, and since the visual impression does not reach consciousness until impulses reach the diencephalon, the area striata does not really perform the function of perception. However, common usage has dignified the term primary perception, and it seems wise to keep it.

So far as the cortex is concerned, the next physiologic step above the level of primary perception is recognition and for this function area 18 of Brodmann is essential. Loss of the function of recognition resulting from an organic cerebral lesion and through one sense organ only is *agnosia,* and a distinction must be made between agnosias for each of three senses; olfactory and gustatory agnosias are not definitely known. Loss of the function of visual recognition is *visual agnosia.* From the standpoint of cerebral localization it is necessary to distinguish between recognition of animate and inanimate objects because one function is not infrequently lost without the other. And as objects have many attributes such as form, color, distance (dimension), and direction and as each of these attributes has been known to be lost separately through an organic lesion of area 18 (parastriata) we are compelled to acknowledge *general visual object agnosia, visual animate object agnosia, visual inanimate object agnosia, visual form agnosia, color agnosia, visual distance agnosia, and visual direction agnosia.* In the realm of animate objects, organic cerebral lesions cause agnosia separately for certain parts of the body, especially the fingers. We thus have *visual finger agnosia.* Visual agnosia for the entire body has occurred from a cerebral lesion and this has been described as autotopagnosia (referring to agnosia for topography of oneself). The term, however, should be qualified by the adjective visual, making *visual autotopagnosia.* The lesion causing this condition is found in the occipital lobe, areas 18 and 19 of Brodmann of the major side, or both sides. All visual agnosias not concerned with language result from occipital lesions, but in order to have a consistent nomenclature an anatomic term must be added to each.

The localization of the lesions causing agnosias *in the sphere of language* is not so simple. The area of cortex essential to visual recognition of letters, syllables, words, and musical notes is in the angular gyrus. The similar area for mathematical figures is made up of the borders of the interparietal sulcus. (One little exception to these statements which must be made because of its importance in treatment of certain cases is this: when the infant first learns letters they are to it not symbols but objects. They are therefore stored as engrams in area 18 of Brodmann at that time. They may remain there ready for use and this explains the fact that adults who have had their angular gyri destroyed and who consequently are totally unable to recognize syllables or words, sometimes recognize letters and are thus able to spell words aloud and recognize them by their sounds.) Visual verbal agnosia, visual literal agnosia, visual numeral agnosia, and visual musical agnosia all result from cortical destruction of the corresponding cortical areas. But this is not the only possible site for the causative lesions because subcortical destruction in the same area or between the cortical areas in question and the occipital pole may cause it also.

Moreover, in the cases in which the individual affected has established strong associations between the angular gyrus and Wernicke's area (which is the rule) or between the angular gyrus and the convolution of Broca (the exception) destruction of these associated areas may cause visual agnosia for letters, words, etc. In a given case one cannot tell by the agnosia alone where the lesion is. Other findings in the case give the clue. Thus hemianopia would indicate temporal, lower parietal, or occipital location; hemisparesis would indicate a capsular or frontal cortical lesion, etc. Because of these possibilities anatomic terms in the form of adjectives must be added to the physiologic descriptive term in designating the agnosia.

We thus have *visual parietal numeral agnosia, visual occipital numeral agnosia, visual angular literal agnosia, visual occipital literal agnosia, visual temporal literal agnosia, visual insular literal agnosia (rare), visual frontal literal agnosia (rare), visual angular verbal agnosia, visual occipital verbal agnosia, visual temporal verbal agnosia, visual insular verbal agnosia, visual frontal verbal agnosia, visual subcortical angular musical agnosia, visual musical angular agnosia, visual musical occipital agnosia, and visual subcortical angular verbal agnosia.* A corresponding series with the word *syllabic* substituted for *verbal* could be made but is hardly necessary. The distinctions between cortical and subcortical lesions of the angular gyrus with reference to agnosia are made clinically by the occurrence of simultaneous agraphia in the former (cortical) and its absence in the latter.

The auditory agnosias all result from temporal lesions. The reason that temporal lesions can cause visual verbal agnosia while angular gyrus lesions do not cause auditory verbal agnosia rests on the fact that the temporal lobe is trained in the infant and child for four to five years before the angular gyrus comes into function. The angular gyrus is usually dependent on the area of Wernicke but the area of Wernicke is independent of the angular gyrus.

Of auditory agnosias we have *general auditory temporal agnosia, auditory temporal verbal agnosia,* and *auditory temporal musical agnosia.* Auditory verbal agnosia results from a lesion of Wernicke's area and occasionally from lesions in its immediate vicinity within the temporal lobe because in some cases the pressure or edema from a neighboring lesion causes the area of Wernicke to "resign." Auditory musical agnosia is rare because the minor side assumes the function easily. When it does occur it results from a lesion of the anterior extremity of the superior temporal convolution (area 38 of Brodmann). It is entirely useless to consider the site of the causative lesion in transcortical or subcortical sensory aphasia because the lesion is located in a position identical with that for ordinary auditory verbal agnosia. The same statement applies to paraphasia. Paraphasia or senseless repetition of sounds heard is simply due to imperfect performance of the minor side.

Olfactory and gustatory agnosias are still impossible of diagnosis either clinically or pathologically. It is doubtful whether they occur.

Tactile agnosia (astereognosis) may result from a lesion of the parietal lobe (usual) and very rarely from an occipital lesion. In the

former case the *tactile parietal agnosia* is unilateral only and contralateral to a lesion of the postcentral gyrus, superior parietal lobule, or supramarginal gyrus. It is probable that the postcentral gyrus is the essential area concerned and that lesions in the other two areas cause tactile agnosia by edema or pressure. When tactile agnosia results from an occipital lesion it is invariably associated with a lesion of area 19 of Brodmann. The patient is unable to form visual concepts of objects by either the visual or tactile route and hence the tactile agnosia (astereognosis) is bilateral even from a unilateral occipital lesion. Such a clinical condition is now known as apperceptive blindness of the senile (Pick) but should be called *occipital tactile and visual agnosias.* This term indicates that the area for revisualization of former images is out of function and its removal is causative of the tactile agnosia (there are two simultaneous agnosias).

When the body scheme is disturbed (by a lesion of the gyri of Gratiolet) the patient may have separate tactile agnosia for the fingers. This should be called *tactile finger parieto-occipital agnosia.* If the entire body scheme is disturbed, *visual autotopagnosia* results.

In the use of the term agnosia, there is only one slight modification of its past usage that needs to be agreed upon, namely that agnosia should apply in any case to recognition from one sense organ only. Its misuse in the past has resulted from inaccurate thinking. Several unacceptable terms have thus arisen. We cannot logically speak of ideational agnosia because ideation goes far beyond recognition. Neither can we speak of loss of finger recognition as finger agnosia unless we mean either visual recognition or tactile recognition. If both are present we have two simultatneous agnosias before us.

The third step in sensory cortical function is that of reactivation of former engrams (images in the visual sphere, sounds in the auditory sphere, tactile reconstructions in the tactile sphere). This third step consists in bringing into consciousness memory from the past. We have been accustomed to speak of it as revisualization in the visual sphere and as recall in the other two spheres. The term recall is distinctly English and not at all specific for the function of cerebral function. The term amnesia is in universal use for forgetting, but forgetting is often a positive process, not merely a failure to remember but a relegation of something unpleasant to the subconscious. Moreover, all agnosias, apraxias, and aphasias are forms of amnesia. There is an international term which means the process of reliving a former memory, namely reminiscence. The failure to effect *reminiscence* could be termed *irreminiscence* and the verb, now only colloquial in English, *reminisce,* can be dignified with usage. I therefore suggest that the function of the tertiary sensory cortex be called reminiscence. We can then speak of *visual, auditory, tactile, olfactory,* and *gustatory reminiscence* and *irreminiscence.*

The function of the tertiary visual cortex (area 19 of Brodmann) is, then, that of visual reminiscence. By means of area 19 (area peristriata) one relives former visual memories. It must be recalled that area 18 is the servant of area 17 and functions only through it (area 18 serves the purpose of recognizing images primarily perceived by

area 17) and one cannot voluntarily stimulate it without allowing area 17 to function again by the patient's voluntary act of looking again. But area 19 is the card index to former memories in the service of all the rest of the brain. One can revive a former image by hearing an associated sound, by feeling an object which is associated in memory, by smelling or tasting a substance which can revive the memory or by seeing an object which has associated memories. But if one reactivates the engrams of area 19 by seeing, one works through area 18. The other four senses reactivate area 19 directly. Its reactivation is reminiscence.

For verbal or musical visual symbols the angular gyrus serves for reminiscence. For numerals the borders of the interparietal sulcus serve the purpose. For reminiscence of the body scheme the gyri of Gratiolet are essential.

Visual irreminiscence is manifest clinically by loss of power to revisualize objects and all attributes of objects such as form, color, direction, dimension and relative distances between objects or parts of objects. Loss of power to revisualize cardinal directions, street scenes, one's home, one's friends or one's body have all been observed following lesions of area 19 of Brodmann. We thus have: *form irreminiscence, visual occipital; color irreminiscence, occipital; distance irreminiscence, occipital; direction irreminiscence, occipital; visual verbal irreminiscence, angular; visual musical irreminiscence, angular; visual literal irreminiscence, angular; visual numeral irreminiscence, parietal; visual finger irreminiscence, parieto-occipital;* and when the body scheme is affected, *visual body irreminiscence, parieto-occipital.*

In the auditory sphere cortical areas concerned with reminiscence cannot be sharply separated from the areas for recognition. It seems that the portion of temporal cortex juxtaposed to the musical and general auditory cortices for recognition serve for reminiscence. It is probable that the area essential to the function varies considerably in different individuals. Clinically it is well known that *auditory musical irreminiscence, temporal; auditory verbal irreminiscence, temporal;* and *general auditory irreminiscence, temporal* occur. One may be unable, after a temporal lesion, to recall a tune but able to recognize it when heard, or one may lose the exact wording of a quotation while retaining a concept of the idea expressed and recognize the words when they are spoken.

Aphasia has been defined in the past as loss of the power to comprehend or express signs and symbols by which man communicates with his peers. It has been agreed that dementia, psychotic disturbances and dysarthria are excluded as causes and that aphasia is always due to an organic lesion of the brain. We can smplify this definition somewhat by saying that *aphasia consists in organic disease of the cerebral memory engrams for language.* Either of these definitions makes aphasia embrace those agnosias and apraxias which have to do with language. I suggest that in any compound term embodying the word agnosia or apraxia the added term of aphasia is a redundancy and not advisable. We can reserve the use of the term aphasia in our nomenclature for designating "the loss of language association with." Thus *visual finger*

aphasia means loss of power to associate the written terms for the fingers with the *object* (finger), *color aphasia* loss of power to associate the language symbol with the color. When one wishes to distinguish between the loss of power to associate the symbol and the loss of power to determine the *significance* of the symbol in terms of language, the adjective *semantic* introduced by Head serves nicely.

There are, of course, all degrees of semantic aphasia even within the present use of the term. So simple a term as the verb *to read* may have a great variety of significances. It may mean:

a. To pronounce the sound indicated by the written symbol without even the faintest understanding, just as I may "read" Welsh (of which I am ignorant).

b. To render the symbol in another language, as to say that this P is an R because the language is Greek.

c. To interpret the word symbol in its most elementary way as to say that that word (cat) means an animal like the one we have in the house.

d. To interpret the symbol in any degree of complexity as to say that "cat" means a domesticated, carnivorous quadruped of the feline family, or to interpret many pages of a legal citation as blackmail, or to interpret the scriptures. To read has even wider meanings as in "to read between the lines" or to read a latent content into a dream.

However, if we are to establish a practical nomenclature such a range of semantics must be discounted. We must be willing to accept an arbitrary distinction between simple recognition and recognition plus interpretation.

It will be necessary to distinguish carefully between the object and its symbol. Loss of *recognition* of the object or its symbol is agnosia for the object or the symbol. Loss of *significance* of the symbol is aphasia.

Inasmuch as all of the old motor aphasias are forms of apraxia it will be an improvement to call them apraxias, or apractic aphasias.

With these explanations we can proceed to elucidation of terms. It is practical to work out from the known agnosias to the aphasias, both anatomically and clinically.

An object may be recognized and comprehended without the patient's being able to establish the symbolic association by seeing or feeling the object. The patient says, "I know what it is but I can't name it." This is the classic amnesic aphasia. Inasmuch as the difficulty is one of language with reference to the object, this is properly called visual object aphasia and tactile object aphasia. In either case the lesion affects the association between the occipital area for visual recognition and the temporo-angular-formulation area or the association between the parietal lobe and the temporo-angular-formulation area. This area is in the posterior part of the temporal lobe, especially area 37 of Brodmann. Hence we derive the terms *visual temporal object aphasia* or *tactile temporal object aphasia* and both are usually present in the same case. The same syndrome applied to colors need not be encumbered with the adjective visual since colors are recognized only

by vision. We therefore select the term color aphasia but as the lesion may be occipital or temporal, we have *temporal color aphasia* and *occipital color aphasia.* Loss of ability to identify fingers by name is exactly parallel and is *visual finger temporo-occipital aphasia.* In this defect we have two categories, *visual temporo-occipital finger aphasia* and *tactile parieto-occipital finger aphasia.* When other parts of the body are affected the part may be named accordingly.

Gustatory and olfactory aphasias are at present unknown.

Auditory aphasia consists in the loss of ability to associate the name with the sound of an object such as loss of ability to name a typewriter by hearing its sound. It practically always accompanies auditory verbal agnosia, only one case having been reported in which it occurred separately. *Auditory musical aphasia* if it ever occurs is extremely rare. In auditory aphasias the lesion is always in the temporal lobe.

Semantic aphasia is aphasia of significance. It does not come into play relative to objects because by the time one has recognized the object, and has named it, its significance is known. Thus if one recognizes the sound of a saw and is able to name the object the significance is complete. This is not true with reference to language symbols. One may see a certain word or phrase and be able to select from a given copy all similar words or phrases and still be totally unaware of their significance. Or one may hear and recognize a word (as shown by ability to repeat it) and still be totally unaware of its significance. Such defects are *semantic aphasias.* The visual semantic aphasias may be described as alexias but they are still aphasias.

We have *visual semantic external capsular aphasia* (loss of significance of written or printed matter through removal of the practic component), *visual semantic temporal aphasia* (loss of significance of written or printed matter due to a lesion between the angular gyrus and Wernick's area or in Wernicke's area itself), *visual semantic third frontal aphasia* (loss of significance of written or printed matter from destruction of the practic component), *auditory semantic temporal verbal aphasia* (loss of ability to determine significance of spoken language though able to recognize it as spoken language), and *auditory semantic temporal musical aphasia* (loss of ability to determine significance of the music though able to recognize it, type of auditory amusia).

There is also *formulation aphasia* or inability to formulate language (the old paraphasia or jargon aphasia).

The Apractic Aphasias

Inasmuch as the types of aphasia which have been called "motor" are all forms of apraxia and as apraxia is only one form of motor disturbance the term apractic aphasia is far more exact than "motor aphasia." It is necessary to retain the two designations "apractic" and "aphasia" because some apraxias do not concern language at all.

The motor patterns located in the convolution of Broca are engrams of the memory of how to make certain sounds with the organs of speech. Disease of these engrams causes motor pattern apraxia (the type which Liepmann called kinetic apraxia of the limb but which in

this instance does not affect a limb). As the impulses from these cortical engrams must reach the precentral gyrus in order to produce speech, it is feasible for a lesion between Broca's convolution and the precentral gyrus to render speech impossible. It is also possible for a lesion of the anterior portion of the insula to extend deeply enough (a few millimeters to a centimeter into its substance) to give the same syndrome.

Beside these mechanisms which can render speech impossible, there is the mechanism of preventing formulation of language. Lesions at the temporal isthmus, by destroying the cross-road of association fibers, can so interfere with language reminiscence that the patient has nothing to say, i.e. he has no speech ideation. For this reason when the general physical examination indicates a severe posterior temporal lesion and the patient is also speechless, it is not necessary to assume an apraxia of speech. The patient must have ability to formulate his language before he can send language-composition impulses to the motor area and the motor area must not be blamed for failure to dispatch impulses which do not reach it.

The apractic aphasias are, then, *frontal apractic aphasia* (the old Broca's aphasia), *insular apractic aphasia, frontal triangularis apractic amusia* (lesion of pars triangularis, which contains engrams for praxia of music), *second frontal apractic agraphia* (due to lesion of Exner's writing center at the second frontal convolution), and *third frontal apractic agraphia* (the agraphia accompanying apractic aphasia).

Apraxia

The physiologic basis of the apraxias has already been discussed. It has also been pointed out that the motor aphasias, some types of agraphia, some disturbances of conjugate lateral turning of the eyes and some cases of dysarthria and dysphagia, etc., are types of apraxia. There is no difference between apractic amusia and musical apraxia, or between apractic aphasia and aphasic apraxia; the selection of the term in a given case of that sort depends on one's viewpoint at the moment of discussing the subject. One point, however, must remain clear, namely that agnostic apraxia is an impossibility because apraxia based on agnosia is a form of agnosia. There cannot be a sensory apraxia of any sort. But we may have the reverse, apractic agnosia which is an agnosia resulting from destruction of a motor pattern (an apraxia) which prevents the sensory pattern from functioning. In such cases the anatomic term will clarify the idea in each case.

The apraxias will therefore embrace: *apraxia* (for any particular act or in general), *cortical motor pattern apraxia, ideokinetic callosal apraxia, ideokinetic parietal apraxia, ideational apraxia.*

Agraphia

A separate discussion of the terms in agraphia is necessary purely for coordination of the elements. This is because agraphia may be due to an irreminiscence, to ideokinetic apraxia, to cortical motor pattern apraxia, or to lesions of engrams which the individual in question has, by training, intimately associated with the function of writing. We have

irreminiscence angular agraphia, ideokinetic apractic parieto-occipital agraphia, ideokinetic callosal apractic agraphia, formulation agraphia, third frontal apractic agraphia, second frontal apractic agraphia, and *external capsular agraphia* (the fibers conveying impulses from the angular gyrus to the frontal lobe for writing coursing in the external capsule). It will be evident from this that the subject of agraphia is the ultimate in difficulty of cerebral localization on the basis of it alone. Isolated agraphia occasionally results from a lesion of the foot of the second frontal convolution and from a lesion of the gyri of Gratiolet (border between the angular gyrus and the occipital lobe). *Isolated agraphia,* however, is exceedingly rare.

Alexia

Alexia (a type of aphasia in the broad sense) must also be the subject of correlation because it may result from lesions in any one of a great variety of locations and may be agnostic or semantic and may result from loss of motor patterns which in a given case have been associated with the patient's system of reading. Alexia may affect letters, words, musical symbols, and numerals. We have *occipital agnostic verbal alexia, angular agnostic verbal alexia, angular agnostic musical alexia, angular agnostic literal alexia, parietal agnostic numeral alexia, temporal agnostic verbal alexia, temporal semantic alexia, agnostic external capsular alexia, semantic external capsular alexia, agnostic third frontal alexia, semantic third frontal alexia,* and *tactile parietal agnostic alexia.*

It will be clear from this presentation that one must analyze with great care the reason for alexia in a given case. The causative lesion may be located anywhere from the occipital to the frontal lobe and it may be due to loss of recognition or loss of knowledge of significance of the recognized written matter.

Acalculia

Acalculia, the loss of ability to calculate, is also aphasia since it is a loss of language function. It results from a lesion of the parieto-occipital area.

Amimia

Amimia has been used as a descriptive term to designate inability of a patient to mimic the movements and gestures of the examiner. Amimia depends on a loss of either comprehension of the act (based on agnosia or on a higher mental function akin to "simultanagnosia") or an apraxia. While the term amimia may be a useful shorthand term for such a loss, it in no way is conclusive of the site of the causative lesion. The reason for the amimia must be analyzed.

Amusia

Amusia has been extensively used to indicate loss of the musical functions. It may be used as a convenient designation but its causative elements should be appreciated. It is based on agnosia for the symbols, aphasia for their comprehension, or apraxia for the execution of vocal or instrumental music.

PROPOSED NOMENCLATURE FOR AGNOSIAS, APRAXIAS AND APHASIAS

Localization of Lesions
Definitions and Old Terminology

1. Acalculia.

   Loss of ability to calculate with or without agnosia or loss of revisualization of symbols.

   O.T. Acalculia.

   Focus. The lesion affects the posterior parietal or parieto-occipital region.

2. Agnosia, Auditory temporal (general).

   Loss of power to recognize sounds heard.

   O.T. Auditory or acoustic agnosia, psychic deafness, Wernicke's aphasia.

   Focus. The lesion affects the posterior portion of the superior temporal convolution.

3. Agnosia, auditory, temporal, musical.

   Loss of power to recognize music as music by sound alone.

   O.T. Musical agnosia, psychic amusia.

   Focus. The lesion affects the anterior extremity of the temporal lobe.

4. Agnosia, auditory, temporal, verbal.

   Loss of power to recognize words heard.

   O.T. Auditory or acoustic verbal agnosia, psychic deafness, Wernicke's aphasia, subcortical acoustic verbal agnosia.

   Focus. The lesion affects the posterior superior temporal convolution.

5. Agnosia, tactile, occipital.

   Loss of power to recognize objects by the sense of touch alone even with the use of both hands. The loss is based on loss of revisualization (loss of visual reminiscence) and because of this loss the patient cannot form a visual image of the object by touch alone. The condition occurs in those particularly visual-minded.

   O.T. Apperceptive blindness of the senile of Pick, ideational agnosia of Liepmann.

   Focus. The lesion affects area 19 of Brodmann in its convex portion.

6. Agnosia, tactile, parietal.

   Loss of power to recognize by touch alone objects which are recognized by the other senses, or in the other hand, as this agnosia is unilateral.

   O.T. Astereognosis, tactile agnosia.

   Focus. The lesion affects the parietal lobe and may be cortical or subcortical.

7. Agnosia, tactile, parieto-occipital.

   Definition is identical with that of occipital tactile agnosia (5).

   O.T. Apperceptive blindness of the senile of Pick, ideational agnosia of Liepmann.

   Focus. The lesion affects area 19 of Brodmann and the posterior portion of the parietal lobe.

8. Agnosia, tactile, parieto-occipital, finger.

   Loss of power to recognize one's fingers by the sense of touch alone.

   O.T. Finger agnosia.

   Focus. The lesion affects the convolutions of Gratiolet.

9. Agnosia, visual, angular, literal.

   Loss of power to recognize letters by vision alone, due to a lesion of the angular gyrus.

   O.T. Visual literal agnosia, literal alexia, part of Wernicke's aphasia.

   Focus. The lesion affects the angular gyrus.

10. Agnosia, visual, angular, verbal.

    Loss of power to recognize written or printed words by vision alone (embossed letters may still be read) due to a lesion of the angular gyrus.

    O.T. Visual verbal agnosia, alexia, part of Wernicke's aphasia.

    Focus. The lesion affects the angular gyrus.

11. Agnosia, visual, angular, musical.

    Loss of power to recognize musical notes by vision alone, due to a lesion of the angular gyrus.

    O.T. Visual agnosia for musical symbols, visual amusia.

    Focus. The lesion affects the angular gyrus.

12. Agnosia, visual, frontal, literal.

    Loss of power to recognize letters by sight alone, due to a lesion of the frontal lobe. Through destruction of Broca's convolution or of the frontal writing center at the foot of the second frontal convolution the motor associations (which the patient has maintained since childhood and which to him are necessary for visual recognition) even visual recognition has become impossible. This is a rare occurrence.

    O.T. Visual literal agnosia, literal alexia, part of Wernicke's aphasia.

    Focus. The lesion affects the foot of the second or third frontal convolutions. The explanation is identical with not in the angular gyrus is determined by the presence in the case of apractic aphasia or other apraxia, especially cortical motor pattern apraxia.

13. Agnosia, visual, frontal, verbal.

Definition is identical with (12) above except that words alone and not letters are effected.

O.T. Visual verbal agnosia, element of Wernicke's aphasia, alexia.

Focus. The lesion affects the foot of the second or third frontal convolutions. The explanation is identical with that given in (12) above. The reason that recognition of letters is preserved is that they are stored as engrams of objects in area 18 of Brodmann in many instances.

14. Agnosia, visual, insular, literal.

Loss of power to recognize letters by sight alone due to a lesion of the insula or pathways immediately subcortical to it. By destruction of tracts, which in such a case are necessary for association of the convolution of Broca or of the foot of the second frontal convolution with the angular gyrus, the angular gyrus cannot function alone in recognition of letters. This is a rare occurrence and is based on retained childhood associations.

O.T. Visual literal agnosia, alexia, part of Wernicke's aphasia.

Focus. The lesion affects the insula and probably of necessity the external capsule through which the association fibers go.

15. Agnosia, visual, insular, verbal.

The definition is identical with that of (14) above except that the loss affects words and not letters.

O.T. Visual verbal agnosia, alexia, part of Wernicke's aphasia.

Focus. The explanation of the syndrome and its rarity are identical with those given under (14) above.

16. Agnosia, visual, occipital (general). This includes visual agnosia for animate and inanimate objects.

Loss of power to recognize objects or symbols by vision alone in spite of adequate visual perception (and with sufficient mental capacity in general) due to a lesion of the occipital lobe. The visual agnosias for symbols are called agnosias rather than aphasias because the term agnosia is more specific.

O.T. Visual agnosia (qualified), psychic blindness, mind-blindness.

Focus. The lesion affects area 18 of Brodmann in its convex portion.

17. Agnosia, visual, occipital, musical.

Loss of power to recognize musical symbols by vision alone due to a lesion of the occipital lobe. There is nearly always, if not always, an associated homonymous hemianopia.

O.T. Visual musical agnosia, subcortical musical alexia.

Focus. The focus is subcortical (and perhaps also cortical) in the occipital lobe. The agnosia is caused by the

fact that impulses from both areae striatae fail to reach the angular gyrus for recognition.

18. Agnosia, visual, occipital, literal.

The definition is identical with that given in (17) above except that letters instead of musical notes are affected.

O.T. Literal agnosia, subcortical literal agnosia, literal alexia.

Focus. The focus and explanation are identical with that given in (17) above.

19. Agnosia, visual, occipital, numeral.

Loss of power to recognize mathematical symbols due to a lesion of the occipital lobe. There is usually an associated homonymous hemianopia.

O.T. Visual numeral agnosia, numeral alexia, psychic blindness.

Focus. The focus is in the occiptal lobe so placed as to prevent impulses from reaching the borders of the interparietal sulcus from both areae striatae.

20. Agnosia, visual, occipital, verbal.

The definition is identical with that given under (17) above except that words instead of musical notes are affected.

O.T. Pure visual verbal agnosia, pure word blindness.

Focus. The lesion is in the occipital lobe, subcortical (and often cortical). It prevents recognition of words by interrupting impulses from both areae striatae to the angular gyrus. It does not cause agraphia because the angular gyrus itself is spared. That is the basis for the old terminology "pure."

21. Agnosia, visual, parietal, numeral.

Loss of power to recognize mathematical symbols due to a lesion of the parietal lobe, either at the borders of the interparietal sulcus or subcortical to that area.

O.T. Visual agnosia for mathematical figures, figure agnosia.

Focus. The lesion is cortical on the borders of the interparietal sulcus or subcortical to that area.

22. Agnosia, visual, parieto-occipital, finger.

Loss of power to recognize fingers by vision alone, those of the patient or those of the examiner or both.

O.T. Finger agnosia.

Focus. The lesion affects the convolutions of Gratiolet.

23. Agnosia, visual, subcortical, angular, musical.

Loss of power to recognize musical symbols due to a subcortical lesion of the angular gyrus. There is usually an associated homonymous hemianopia.

O.T. Musical agnosia, visual agnosia for musical notes.

Focus. The lesion is subcortical to the angular gyrus and prevents recognition by interrupting impulses from both

areae striatae to the angular gyrus. There is no agraphia for musical notes because the cortex of the angular gyrus is not affected.

24. Agnosia, visual, subcortical, angular, verbal.
The definition is identical with that of (23) above except that words instead of musical notes are affected.
O.T. Pure visual verbal agnosia, alexia, subcortical visual verbal agnosia.
Focus. The lesion affects the subcortical region of the angular gyrus. There is no agraphia because the cortex is spared.

25. Agnosia, visual, temporal, literal (and verbal).
Loss of power to recognize letters and words due to a lesion of Wernicke's area or the small space between Wernicke's area and the angular gyrus.
O.T. Visual literal (and verbal) agnosia, literal (and verbal) alexia, Wernicke's aphasia.
Focus. The lesion either separates Wernicke's area in the posterior superior temporal convolution from the angular gyrus or affects Wernicke's area itself. It causes visual agnosia because in the great majority of cases the angular gyrus cannot function for recognition of symbols without association with Wernicke's area. This is because visual engrams are laid down in close association with auditory ones.

26. Agraphia.
A descriptive term when not qualified, meaning loss of the power to write.
O.T. Agraphia.
Focus. On the basis of agraphia alone it is usually impossible to make a focal diagnosis. See agraphia, isolated.

27. Agraphia, apractic, second frontal.
Loss of power to write due to a lesion of the writing center at the foot of the second frontal convolution. The patient usually is unable to write by any method, even by building words with blocks or by writing on a typewriter.
O.T. Motor agraphia, isolated agraphia.
Focus. The lesion is in the cortex of the foot of the second frontal convolution.

28. Agraphia, apractic, third frontal.
Loss of the power to write due to a lesion of Broca's convolution. There is an associated apractic aphasia.
O.T. Broca's aphasia, motor agraphia.
Focus. Broca's convolution is affected either cortically or subcortically. The agraphia results secondarily because most patients are unable to write unless they have the motor patterns for speech associated with their writing mechanism.

29. Agraphia, external capsular.
    Loss of the capacity to write due to a lesion of the external capsule.
    O.T. Motor agraphia.
    Focus. The lesion affects the external capsule and causes agraphia because the impulses from the angular gyrus (where reminiscence of words occurs) fail to reach the writing center at the foot of the second frontal convolution.
30. Agraphia, formulation, temporal.
    Loss of ability to write because of inability to formulate language, all due to a lesion of the temporal lobe.
    O.T. Amnesic agraphia, paragraphia.
    Focus. The lesion affects the posterior part of the temporal lobe. It may be small or large. It interferes with the fuction of the language formulation area.
31. Agraphia, ideokinetic apractic, callosal.
    Loss of ability to write because of a lesion of the corpus callosum which causes ideokinetic apraxia.
    O.T. Apractic agraphia.
    Focus. The lesion is in the corpus callosum, usually in the anterior portion. Unless the lesion is a slowly progressive one the patient usually recovers ability to write.
32. Agraphia, ideokinetic apractic, parieto-occipital.
    O.T. Apractic agraphia.
    Focus. The lesion affects most often the subcortical structures of the general region of the supramarginal gyrus.
33. Agraphia, irreminiscence literal (or verbal) angular.
    Loss of ability to write due to loss of ability to recall the appearance of letters or words, and caused by a lesion of the angular gyrus.
    O.T. Agraphia.
    Focus. The lesion affects the cortex of the angular gyrus.
34. Agraphia, isolated, parieto-occipital.
    Loss of ability to write due to loss of knowledge of visual or visual memory guidance of hand movements and due to a lesion of the convolutions of Gratiolet.
    O.T. Isolated agraphia (it being the only aphasic element in the case) as part of Gerstmann's syndrome.
    Focus. The lesion affects the convolutions of Gratiolet at the border between the angular gyrus and the occipital convolutions.
35. Agraphia, isolated, second frontal, apractic.
    Loss of ability to write due to cortical motor apraxia for writing only and due to a lesion at the foot of the second frontal convolution.
    O.T. Motor agraphia, isolated agraphia.
    Focus. The lesion affects the cortex at the foot of the second frontal convolution.

36. Agraphia, temporal.

Loss of ability to write due to crippling of the angular gyrus through destruction of its associative connection with Wernicke's area and caused by a lesion of the superior temporal convolution between the angular gyrus and Wernicke's area.

O.T. Agraphia accompanying Wernicke's aphasia.

Focus. The lesion affects the small area of cortex between the area of Wernicke and the angular gyrus (part of area 22 of Brodmann).

37. Alexia.

Descriptive term signifying loss of ability to read without reference to the physiologic cause except that it is due to a focal cerebral lesion. Sufficient visual perception and mental capacity are presupposed.

O.T. Alexia.

38. Alexia, agnostic, external capsular, literal and verbal.

Loss of ability to recognize words and letters due to a lesion of the external capsule and based physiologically on dissociation of the angular gyrus from Broca's convolution. This is a rare occurrence and is found only in persons who have retained childhood associations between vocal reading and visual reading.

O.T. Alexia, visual, verbal or literal alexia.

Focus. The lesion affects the external capsule.

39. Alexia, agnostic, literal and verbal angular.

Loss of ability to recognize letters and words due to a lesion of the angular gyrus.

O.T. Agnostic alexia, alexia, visual literal and verbal agnosia.

Focus. The lesion affects the cortex of the angular gyrus.

40. Alexia, agnostic, musical, angular.

Definition and physiologic-anatomic basis is identical with (39) above except that musical symbols are affected instead of letters and words.

O.T. Musical alexia, visual musical agnosia.

Focus. The lesion affects the cortex of the angular gyrus.

41. Alexia, agnostic, numeral, parietal.

Loss of ability to recognize mathematical figures due to a lesion of the parietal lobe (borders of the interparietal sulcus).

O.T. Visual agnosia for mathematical figures, mathematical alexia.

Focus. The lesion affects the cortex of the parietal lobe at the borders of the interparietal sulcus.

42. Alexia, agnostic, third frontal, literal and verbal.

Loss of ability to recognize letters and words due to a lesion of Broca's convolution. This is a rare occurrence and is found only in those who have retained childhood associations between the angular gyrus and Broca's convolution as a necessity for recognition.

O.T. Visual verbal agnosia, alexia.

Focus. The lesion affects the cortex of Broca's convolution.

43. Alexia, agnostic, verbal, occipital.

Loss of ability to recognize words due to a lesion of the occipital lobe so placed that impulses from both areae striatae fail to reach the angular gyrus.

O.T. Pure visual verbal agnosia.

Focus. The lesion is in the occipital lobe and if unilateral affects also the splenium of the corpus callosum. There is nearly always an associated homonymous hemianopia.

44. Alexia, agnostic, verbal, temporal.

Loss of ability to recognize written or printed words due to a lesion of the temporal lobe. While the angular gyrus functions in recognition, in most cases it cannot function without association with Wernicke's area.

O.T. Visual verbal agnosia, verbal alexia. Part of Wernicke's aphasia.

Focus. The lesion affects the cortex of Wernicke's area itself or separates Wernicke's area from the angular gyrus.

45. Alexia, semantic, external capsular.

Loss of ability to determine significance of words, which are recognized, due to a lesion of the external capsule. In rare cases a lesion in this location even prevents recognition but usually interferes, if at all, with reading, only in determination of significance.

O.T. Semantic alexia, transcortical sensory aphasia.

Focus. The lesion affects the external capsule, usually also the insula.

46. Alexia, semantic, temporal.

Loss of ability to determine significance of words, which are recognized, due to a lesion of the temporal lobe. The physiologic basis is the requirement of the angular gyrus, in most cases, to have association with Wernicke's area in order to determine significance of what is read.

O.T. Semantic alexia, transcortical sensory aphasia.

Focus. The lesion affects the cortex of Wernicke's area or separates Wernicke's area from the angular gyrus.

47. Alexia, semantic, third frontal.

Loss of ability to determine the significance of words, which are recognized, due to a lesion of Broca's convolution. This form of alexia in a minor degree is common.

O.T. Semantic alexia, transcortical sensory aphasia.

Focus. The lesion affects Broca's convolution either cortically or subcortically.

48. Alexia, tactile, agnostic, parietal.

Loss of ability to read letters or words by touch, sufficient tactile perception presupposed, due to a lesion of the parietal lobes, cortical or subcortical.

O.T. Tactile aphasia, tactile agnosia for letters, astereognosis.

Focus. The lesion affects the parietal lobe opposite to the affected hand. It may be cortical or subcortical.

49. Amimia.

A purely descriptive term used to designate loss of ability to mimic gestures. As amimia may be based on agnosia or on apraxia, the cause must be analyzed in each instance.

O.T. Amimia.

50. Amusia.

A purely descriptive term used to designate a disturbance of the musical sense, auditory, visual, or motor. Its subdivisions are classified as agnosias, apraxias, and aphasias.

O.T. Amusia.

51. Aphasia.

A term restricted to mean "loss of language association with." It includes agnosias and apraxias which are concerned with language. Thus "finger aphasia" means loss of the ability to name or comprehend the names of the fingers. Visual object aphasia means loss of ability to name objects seen, etc. Aphasias based on apraxia are apractic aphasias. The term is therefore narrower than the old expansive definition.

52. Aphasia, apractic, insular.

Loss of ability to speak based on apraxia and due to a lesion of the insula (probably chiefly the subcortical structures, the external capsule in particular).

O.T. Subcortical or association motor aphasia, anarthria of Pierre Marie.

Focus. The lesion affects the insula probably always subcortically.

53. Aphasia, apractic, frontal.

Loss of ability to speak due to a lesion of Broca's convolution cortical or subcortical.

O.T. Broca's aphasia, subcortical motor aphasia, transcortical motor aphasia.

Focus. The lesion affects Broca's convolution, cortically or subcortically.

54. Aphasia, apractic, musical, triangularis.

Loss of ablity to sing words due to a lesion of the pars triangularis of the third frontal convolution.

O.T. Avocalia, motor amusia.

Focus. The lesion affects the pars triangularis of the third frontal convolution.

55. Aphasia, auditory, musical, semantic, temporal.

Loss of ability to comprehend played or sung music, which is recognized, due to a lesion of the temporal lobe. This is rare because of the facility with which the minor side assumes the function.

O.T. Auditory amusia.

Focus. The lesion affects the anterior portion of the temporal lobe.

56. Aphasia, auditory, object, temporal.

Loss of ability to associate the name of an audible object with the sound of the object, e.g. the patient cannot say "bell" when he hears the ring or "watch" when he hears the tick.

O.T. Amnesia aphasia.

Focus. The lesion affects the posterior part of the temporal lobe.

57. Aphasia, auditory, semantic, temporal.

Loss of ability to comprehend names of sounds in general in spite of recognition of the sounds, the disability being due to a temporal lesion.

O.T. Amnesic aphasia.

Focus. The lesion affects the temporal lobe.

58. Aphasia, color, occipital.

Loss of ability to name colors which are seen and recognized, due to an occipital lesion. The lesion breaks the association between the occipital area for recognition and the language formulation area.

O.T. Amnesic color blindness, optic aphasia for colors, visual aphasia for colors.

Focus. The lesion affects the occipital lobe close to the temporal lobe.

59. Aphasia, color, temporal.

This is identical with (58) above except that the lesion is in the temporal lobe close to the occipital.

60. Aphasia, formulation, temporal.

Loss of ability to formulate words and phrases into language.

O.T. Amnesic aphasia, paraphasia, agrammatism.

Focus. The lesion affects the posterior portion of the temporal lobe, chiefly area 37 and the posterior extremity of area 22 of Brodmann.

61. Aphasia, visual, semantic, external capsular.

Loss of comprehension of significance of written or printed words due to a lesion of the external capsule. This form of aphasia is based on the necessity of having association between the angular gyrus and Broca's convolution for full comprehension of written or printed words.

O.T. Semantic aphasia, semantic alexia.

Focus. The lesion affects the external capsule.

62. Aphasia, visual, semantic, temporal.

Loss of comprehension of significance of written or printed words due to a lesion of the temporal lobe. This is based on the dependence of the angular gyrus in association with Wernicke's area for comprehension of written language.

O.T. Transcortical sensory aphasia, semantic alexia, part of Wernicke's aphasia, Wernicke's aphasia.

Focus. The lesion affects Wernicke's area or the short portion of cortex between it and the angular gyrus (posterior extremity of area 22 of Brodmann).

63. Aphasia, visual, semantic, third frontal.

Loss of ability to comprehend significance of written or printed matter due to a lesion of Broca's convolution. This is based on the physiologic necessity for association of the angular gyrus with Broca's convolution for full comprehension of written language.

O.T. Semantic aphasia, semantic alexia.

Focus. The lesion affects Broca's convolution.

64. Aphasia, tactile, finger, parietal.

Loss of ability to name or comprehend names of fingers held by the examiner due to a lesion of the parietal lobe.

O.T. Amnesic finger aphasia, finger aphasia.

Focus. The lesion affects the parietal lobe usually in the region of the supramarginal gyrus. It interrupts the association between the parietal lobe and Wernicke's area.

65. Aphasia, visual, finger, temporo-occipital.

Loss of ability to name or comprehend the names of fingers, seen by the patient, due to a lesion of the temporo-occipital area.

O.T. Finger aphasia, visual finger aphasia, visual agnostic element of finger agnosia, amnesic aphasia for fingers.

Focus. The lesion affects the temporo-occipital area and interrupts association between the occipital area for recognition (area 18) and Wernicke's area.

66. Aphasia, visual, temporal, object.

Loss of ability to name objects seen, though they are recognized, due to a lesion of the temporal lobe.

O.T. Amnesic aphasia, anomia.

Focus. The lesion affects area 37 of Brodmann or the posterior extremity of area 22, in general the posterior portion of the temporal lobe. It interrupts communication between area 18 and Wernicke's area for which reason the auditory memories of the names cannot be associated with the memory of the object seen.

67. Apraxia (for any particular movement, act, idea, or apraxia in general).

The term is only descriptive until qualified. Apraxia is loss of ability to perform as desired or as requested, through loss of

memory of how to perform. This loss of memory in turn is due to destruction of cerebral engrams. Ataxia, paralysis, and dementia must always be excluded as causes.

68. Apraxia, cortical motor pattern.

Loss of ability to execute a *movement* due to lesion of the cortical motor pattern. This type of apraxia is illustrated by Broca's aphasia, by agraphia as a result of a lesion in the foot of the second frontal convolution, by aphasia from a lesion of the precentral gyrus at the frontal operculum, or by apraxia of singing from a lesion of the pars triangularis of the third frontal convolution. The result is the same whether the cortical motor pattern itself is destroyed or whether its connections with the Betz cells, through which the projection produce the movements, are broken.

O.T. Kinetic apraxia of the limb, apraxia of lateral gaze, Broca's aphasia, subcortical motor aphasia, transcortical motor aphasia, apractic amusia, apractic agraphia, apractic aphasia, avocalia.

Focus. The lesion for each type is cortical if it destroys the motor pattern or subcortical if it prevents the organized plan of the movement from reaching the Betz cells where the projection fibers begin for execution of the movement.

69. Apraxia, ideational.

Loss of ability to carry out an idea due to a diffuse lesion of the brain affecting the ideational plan of action. The individual acts constituting the ideational plan can be executed without trouble but the entire ideational plan cannot be retained in consciousness long enough to be executed.

O.T. Ideational apraxia.

Focus. There is no single focus. The "lesion" is either a toxemia or is diffusely placed such as in multiple vascular softening, or multiple tumors.

70. Apraxia, ideokinetic, callosal.

Loss of ability to execute the plan of an act, the plan and the mechanism of execution both being undisturbed but the connection between them being broken, by a lesion of the corpus callosum. The loss may affect the fingers of the patient only.

O.T. Ideokinetic apraxia. Apraxia of finger demonstration, finger agnosia.

Focus. The lesion is in the corpus callosum, anterior for the face and upper limbs, posterior for the lower limbs, intermediate for the trunk. The apraxia does not remain unless the lesion is progressive.

71. Apraxia, ideokinetic, parietal.

This condition is identical with (70) above except that the lesion affects the subcortical structures of the parietal lobe, most potently at the supramarginal gyrus. When it affects the minor limb it is the old sympathetic apraxia.

72. Autotopagnosia, visual, occipital.

Loss of ability to recognize parts of the body. It is essentially visual agnosia for the body and hence is part of general visual agnosia.

73. Irreminiscence.

A term coined to designate a disturbance of ability to recall, i.e. a disturbance of power of reminiscence. It is only descriptive unless qualified.

O.T. Revisualization, re-auditorization, loss of power to recall, amnesia of certain types.

74. Irrimeniscence, auditory, musical, temporal.

Loss of power to recall into auditory memory musical compositions formerly known, due to a lesion of the temporal lobe.

O.T. Loss of musical recall.

Focus. The lesion affects the anterior end of the temporal lobe. (Irritation of the same area causes auditory musical hallucinations).

75. Irreminiscence, auditory (general) temporal.

Loss of power to recall auditory memories in general due to a lesion of the temporal lobe.

O.T. Loss of reauditorization, loss of recall of auditory memories.

Focus. The lesion is a large one in the temporal lobe.

76. Irreminiscence, auditory, verbal, temporal.

Loss of power to recall the auditory memory of words due to a lesion of the temporal lobe. (Irritation of this area causes hallucinations of words.)

O.T. Amnesia aphasia, loss of recall of auditory memory of words.

Focus. The lesion affects the temporal lobe, chiefly area 37 of Brodmann. It is the underlying factor in some cases of disturbance of language formulation.

77. Irreminiscence, color, occipital.

Loss of ability to recall visual images of colors. In some instances the patients recall all colors as being black. (Irritation of this area causes hallucinations of colors.)

O.T. Loss of revisualization of colors.

Focus. The lesion affects the occipital lobe, area 19 of Brodmann, especially the lateral convex surface, cortical or subcortical.

78. Irreminiscence, direction, occipital.

Loss of ability to recall directions, either the cardinal ones or as related to the body of the individual, due to a lesion of the occipital lobe.

O.T. Geometric-optic agnosia, disorientation in space.

Focus. The lesion affects the occipital lobe, chiefly area 19 of Brodmann on the inferior surface.

79. Irreminiscence, distance, occipital.
    This condition is identical with (78) above except that distance instead of direction, is affected.
80. Irreminiscence, visual, body, parieto-occipital.
    Loss of ability to revisualize the body of the patient or of another, due to a lesion of the parieto-occipital lobe. (Irritation of this area causes visual hallucinations of living things.)
        O.T. Disturbance of the body scheme, autotopagnosia.
            Focus. The lesion affects the parieto-occipital region, either cortically or subcortically, usually within a centimeter of the mesial surface (area 19 of Brodmann).
81. Irreminiscence, visual, finger, parieto-occipital.
    Loss of ability to recall visually one's own fingers or those of another due to a lesion of the parieto-occipital area.
        O.T. Finger agnosia, visual agnostic element of finger agnosia, disturbance of the body scheme, part of the Gerstmann syndrome.
            Focus. The lesion affects the border between the angular gyrus and the occipital lobe, the convolutions of Gratiolet.
82. Irreminiscence, visual, object, occipital.
    Loss of power to recall visual images of objects due to an occipital lesion (irritation of this area causes visual hallucinations of objects).
        O.T. Loss of revisualization of objects, disorientation in space, acoustic-optic agnosia.
            Focus. The lesion affects area 19 of Brodmann, chiefly the inferior convex portion, cortically or subcortically.
83. Irreminiscence, visual, literal, angular.
    Loss of ability to recall visual images of letters due to a lesion of the angular gyrus (cortex). This loss causes agraphia.
        O.T. Loss of revisualization of letters.
            Focus. The lesion affects the cortex of the angular gyrus.
84. Irreminiscence, visual, musical, angular.
    Loss of power to recall appearance of musical notes or musical composition due to a lesion of the angular gyrus.
        O.T. Loss of revisualization of musical symbols.
            Focus. The lesion affects the angular gyrus.
85. Irreminiscence, visual, numeral, parietal.
    Loss of ability to recall visual images of mathematical figures due to a lesion of the borders of the interparietal sulcus.
        O.T. Loss of revisualization of mathematical figures.
            Focus. The lesion affects the borders of the interparietal sulcus.
86. Irreminiscence, visual, verbal, angular.
    Loss of power to revisualize written or printed words, due to a lesion of the angular gyrus. This causes agraphia.
        O.T. Loss of revisualization of written or printed words, sensory agraphia.
            Focus. The lesion affects the cortex of the angular gyrus.

87. Irreminiscence, visual, verbal, temporal.

Loss of ability to revisualize words, due to a lesion of the temporal lobe. This causes agraphia.

O.T. Loss of revisualization of written or printed words, sensory agraphia.

Focus. The lesion affects the portion of cortex between Wernicke's area and the angular gyrus (posterior extremity of area 22 of Brodmann). The syndrome is based physiologically on the dependence of the angular gyrus on the area of Wernicke in the function of revisualization of words.

Classic Nomenclature with Proposed Nomenclature Which is Equivalent, is Embraced By, or Embraces the Classic

(O.T., Old Terminology. P.N., Proposed Nomenclature)

O.T. Acalculia.
P.N. Acalculia.

O.T. Agnosia. The term according to some authors included loss of recognition through several sense organs simultaneously.
P.N. Agnosia. The term is now limited to loss of recognition through one sense organ only.

O.T. Agnosia, acoustic or auditory.
P.N. Agnosia, auditory, temporal, (general).

O.T. Agnosia, acoustic or auditory, musical.
P.N. Agnosia, auditory, temporal, musical.

O.T. Agnosia, acoustic or auditory, verbal.
P.N. Agnosia, auditory, temporal, verbal.

O.T. Agnosia, disjunctive (agnosia, ideational).
P.N. In the new light this is interpreted as a combination of visual and tactile agnosia in various combinations.

O.T. Agnosia, dissolutive (agnosia, ideational).
P.N. This is somewhat similar to disjunctive agnosia and is interpreted as a combination of agnosias.

O.T. Agnosia, finger, of Gerstmann.
P.N. This is subdivided into visual agnosia for fingers, tactile agnosia for fingers, and ideokinetic apraxia of fingers.

O.T. Agnosia, geometric-optic.
P.N. This is a part of visual agnosia for objects, geometrical relations being part of objects small or large. That which formerly was considered geometric-optic agnosia is also in part visual object irreminiscence.

O.T. Agnosia, gustatory.
P.N. Gustatory agnosia whenever this may become established as a recognizable fact.

O.T. Agnosia, ideational.
P.N. Various combinations of visual plus tactile agnosia.

O.T. Agnosia, olfactory.
P.N. Olfactory agnosia whenever this may become established as a recognizable fact.

O.T. Agnosia, subcortical acoustic or auditory verbal.
P.N. Auditory verbal agnosia in which there happens to be good function of the minor cerebral hemisphere for formulation of language and for production of speech.

O.T. Agnosia, subcortical, visual, verbal.
P.N. Agnosia, visual, subcortical, angular, verbal.

O.T. Agnosia, tactile (astereognosis).
P.N. Agnosia, tactile, parietal.

O.T. Agnosia, visual (or optic).
P.N. Agnosia, visual, occipital (general).

O.T. Agnosia, visual for letters.
P.N. Agnosia, visual, occipital literal; or agnosia, visual subcortical or cortical angular literal; agnosia visual temporal literal, etc.

O.T. Agnosia, visual for mathematical figures.
P.N. Agnosia visual parietal, or parieto-occipital numeral.

O.T. Agnosia, visual, for musical notes.
P.N. Agnosia, visual occipital (or angular, or subcortical angular) musical.

O.T. Agnosia, visual, for objects or pictures.
P.N. Agnosia, visual occipital (general).

O.T. Agnosia, visual, verbal.
P.N. Agnosia, visual, angular, verbal.

O.T. Agrammatism.
P.N. Aphasia, formulation, temporal.

O.T. Agraphia.
P.N. Agraphia analyzed as to cause and site of lesion. See each heading beginning with "Agraphia."

O.T. Alexia.
P.N. Alexia analyzed as to cause and site. See each heading beginning with "Alexia."

O.T. Amimia.
P.N. Amimia as a descriptive term, but it has little significance until analyzed into terms of agnosia and apraxia.

O.T. Amusia.
P.N. Amusia. The term is to be analyzed in each case as to agnostic, apractic, or aphasic basis. Otherwise, it is purely descriptive.

O.T. Anomia.
P.N. Aphasia, visual, temporal, object.

O.T. Aphasia.
P.N. Aphasia. However, the term is restricted and redefined to mean "the language component of."

O.T. Aphasia, acoustic-optic.
P.N. Irreminiscence, visual, object, occipital.

O.T. Aphasia, amnesic.
P.N. Aphasia, visual, temporal, object; or Aphasia, auditory, temporal, object; or Aphasia, formulation.

O.T. Aphasia, Broca's.
P.N. Aphasia, apractic, third frontal.

O.T. Aphasia, jargon.
P.N. Aphasia, formulation.

O.T. Aphasia, motor.
P.N. Aphasia, apractic (with various anatomical locations of the causative lesions).

O.T. Aphasia, optic.
P.N. Aphasia, visual, temporal, object.

O.T. Aphasia, optic for colors.
P.N. Aphasia, color (temporal or occipital).

O.T. Aphasia, semantic.
P.N. Aphasia, semantic (qualified as to type and anatomic site of causative lesion).

O.T. Aphasia, sensory.
P.N. The term is too loose to have much significance and should be analyzed into its specific components.

O.T. Aphasia, subcortical motor.
P.N. Aphasia, apractic (qualified as to anatomical site of causative lesion).

O.T. Aphasia, transcortical motor.
P.N. Aphasia, apractic (qualified as to site of anatomical site of causative lesion). Its occurrence is based on the appearance of cases in which the minor hemisphere has excellent repetitive capacity.

O.T. Aphasia, Wernicke's.
P.N. As this is a complex syndrome dependent on agnosia, irreminiscence and aphasia the elements should be analyzed in each case.

O.T. Apraxia.
P.N. Apraxia. The defect should be analyzed in each case as to movement, act, and idea.

O.T. Apraxia, constructive (of Kleist).
P.N. This is based on visual irreminiscence of finger movements.

O.T. Apraxia, ideational.
P.N. Ideational apraxia.

O.T. Apraxia, ideokinetic.
P.N. Ideokinetic apraxia.

O.T. Apraxia, kinetic of limb.
P.N. Cortical motor pattern apraxia.

O.T. Apraxia, sympathetic.
P.N. Ideokinetic apraxia affecting the minor limb.

O.T. Astereognosis.
P.N. Tactile agnosia.

O.T. Autotopagnosia.
P.N. Visual autotopagnosia.

O.T. Avocalia.
P.N. Aphasia, apractic musical, triangularis.

O.T. Blindness, amnesic color of Wilbrand.
P.N. Aphasia, visual, temporal, object.

O.T. Blindness, apperceptive of the senile.
P.N. Simultaneous visual and tactile agnosia.

O.T. Blindness, cerebral, color, acquired.
P.N. Agnosia, visual color, occipital.

O.T. Blindness, psychic.
P.N. Agnosia, visual, occipital (general).

O.T. Charcot-Wilbrand syndrome.
P.N. Agnosia and irreminiscence visual, occipital.

O.T. Deafness, psychic.
P.N. Auditory agnosia and auditory semantic aphasia.

O.T. Deafness, verbal.
P.N. Auditory verbal agnosia.

O.T. Disorientation, optical, in space.
P.N. Irreminiscence, object, occipital.

O.T. Imperception (Hughlings Jackson).
P.N. Agnosia, visual, object, occipital.

O.T. Paraphasia.
P.N. Aphasia, formulation, temporal.

O.T. Simultanagnosia (Wolpert).
P.N. A complex disturbance of synthesis of visual impressions.

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