DISCONNEXION SYNDROMES IN ANIMALS AND MAN[1]

BY

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PART II

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TABLE OF CONTENTS

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Introduction

The first part of this paper (Geschwind, 1965) defined disconnexion syndromes and discussed the anatomical bases of such disorders. Particular emphasis was placed on the pattern of cortico-cortical connexions in primates, and the disturbances resulting from lesions of these connexions. This was followed by a discussion of possible developments in the pattern of cortico-cortical connexions which had favoured the development of language. Some examples were given of syndromes of disconnexion in man. Part II will be devoted primarily to a discussion of some of the disturbances of the higher functions in man.

IV. The Agnosias

The agnosias have always represented an unusually difficult group of disturbances. Classically they were defined as disturbances of "recognition" without disturbances of elementary sensation. Some observers have simply denied the existence of disturbances of this type. There has been the attempt on one side to reduce them to perceptual defects of complex nature, such as continuously shifting thresholds. On the other hand there has been the tendency to deal with such disturbances in terms of rather complex psychological malfunctions such as loss of ability to perceive Gestalts. A more classical approach was that of Lissauer (1889) who divided agnosias into apperceptive and associative, or disorders of primary and secondary recognition. The theory that I will present here will propose that the agnosias are indeed associative disorders; what will be added to the classical notion will be an analysis of just what type of associative disorder is involved and an explanation of certain clinical features that have in the past acted as stumbling blocks to analysis of these disturbances. I have already discussed in detail the agnosias of animals. The human cases are also based on disconnexion but I hope to show that the mechanisms are different in important ways from those operative in animals.

Workers in this field have tended to use a certain set of criteria for calling a disturbance an agnosia. First it is necessary to show either (1) that elementary sensation is intact or (2) that if an impairment of elementary sensation is present it is not sufficiently severe to explain the disturbance of recognition. Assuming this to have been demonstrated, how has it been possible to demonstrate that the failure of recognition was not simply a failure of naming? There have been several criteria generally employed. I will list them first and discuss their usefulness afterwards:

(1) *The "agnosic" disturbance is a circumscribed one.*—There is a common view that aphasic misnaming cannot be confined to a single modality; even more strongly it is assumed that it cannot be confined to a class within a modality. From this point of view an aphasic misnames everything; by contrast there can be a visual agnosia or even an agnosia for colours alone.

(2) *"Agnosic" errors differ in character from "aphasic" errors.*—It is often assumed that aphasic errors are of one type, agnosic of another; the aphasic error is a "linguistic" one, the agnosic a "perceptual" one. According to this view, "chair" for "table" is an aphasic error but "a small package" for "a book" is an agnosic error; "something to eat with" for "spoon" is an aphasic error but "pencil" for "knife" is an agnosic error.

(3) *The "agnosic" cannot choose the correct response.*—The aphasic who has failed to name is usually described as being readily able to accept the correct name when it is offered. The "agnosic" by contrast is said to be incapable of selecting the correct name.

(4) *The "agnosic" describes his perceptual difficulty.*—The aphasic is usually pictured as saying, "I know what it is but I can't find the name." When asked about the appearances of objects he asserts that they appear normal to him. The "agnosic," by contrast, informs the examiner that things look different to him or that he has difficulty in seeing clearly. Alternatively he may offer dramatic descriptions of bizarre perceptions, thus apparently providing direct evidence that he is experiencing distortions of his perceptions.

(5) *The "agnosic" cannot show the use of the object.*—This criterion is actually the most complex. The aphasic is said to be able to show the use of the object. The "visual agnosic" cannot show the use of the object but can manipulate it correctly if it is actually placed into his hand. The "apraxic" according to this classical analysis cannot handle the object correctly even when it is placed in his hand.

As I hope to demonstrate, all of these criteria are actually inadequate for the separation of patients into two classes of distinct physiological disturbance. The fundamental difficulty has been in the acceptance of a special class of defects of "recognition," lying somewhere between defects of "perception" and of "naming." What indeed are the criteria for "recognition" and is it a single function? I believe in fact that there is no single faculty of "recognition" but that the term covers the totality of all the associations aroused by any object. Phrased in another way, we "manifest recognition" by responding appropriately; to the extent that any appropriate response occurs, we have shown "recognition." But this view abolishes the notion of a unitary step of "recognition"; instead, there are multiple parallel processes of appropriate response to a stimulus. To describe the behaviour correctly we must describe the pattern of loss and preservation of responses to each particular type of stimulus.

Let us make this more concrete by referring to a particular patient's behaviour. I will return for my example to the case of Geschwind and Kaplan (1962) to which I have several times made reference. Let us consider his difficulty in identifying objects placed in the left hand. He incorrectly named objects placed in the left hand. He could, however, draw the object

afterwards with his left hand or select it from a group tactilely or visually. It is obviously correct to describe this patient as showing correct identification by nonverbal means and incorrect identification verbally of objects held in the left hand. It is difficult to see how this could in any way be described as a "perceptual" disorder since perception must have been intact to insure correct selection from a group or the drawing of the object.

We have interpreted this disturbance as a result of disconnexion from the speech area. In addition the patient showed other disturbances of identification not due to disconnexion from the speech area. Thus, he could not select or draw with the right hand an object held with the left, concealed from vision, or select or draw with the left hand an object held with the right. But again "perceptual" disturbance in either hand is excluded by the patient's ability to select or draw with a given hand an object that had been held in the same hand. We can only regard the defect as associative—what was perceived by the right hemisphere could not be relayed to the left and vice versa.

Let us return for a moment to the failure to identify verbally with the left hand objects held in that hand which we have attributed to disconnexion from the speech area. It may be argued that we have evaded the fundamental issue, that although we have shown that the patient could not name the object placed in his left hand but yet could draw it or select it afterwards from a group, we have not really shown whether the patient had recognized the object. This question is based on two assumptions which I believe are incorrect. The first assumption is that "the patient" is an entity. But in someone whose two hemispheres operate as independently as this patient's the word loses its ordinary meaning. There is no answer to "Did the patient recognize?" There are, however, clear answers to "Under what conditions did the right or the left hemisphere recognize?"

The second assumption is that recognition is more than the sum of the individual acts of naming, drawing, handling, selecting, etc. If it is more than these, how would one test for this faculty and how would one show that this test established some superordinate function of recognition? I do not know of an answer to this question.

I have argued that the patient's failure to identify verbally objects held in the left hand must be regarded as a result of separation of the right somesthetic region from the speech area and can in no sense be regarded as a perceptual defect. In the light of this analysis, let us now consider the patient's responses from the point of view of the classical criteria listed above which were used to distinguish "agnosic" from "aphasic" disturbances. This analysis will demonstrate the inapplicability of these criteria.

(1) *Our patient's disturbance was highly circumscribed* to one modality and indeed one-half of the body. Classically such circumscription was often regarded as being incompatible with an aphasia. Yet as soon as one admits the substantial restriction of speech to one hemisphere, the

possibility of cutting off one-half of the brain from speech must be considered. There is no basis therefore for this classical distinction.

(2) *The errors made by our patient were not "errors of naming"* in the sense of the commonly used criteria. He could call a "screw-driver" a "rubber band," a "coin" a "cigarette lighter." These are not (without meaningless extension of the term) errors in sphere nor are they errors based on similarity of sound. They are, therefore, not "aphasic" errors according to the usual criterion. But it is easy to see that if a particular part of the brain is fully disconnected from the speech area, there is no reason that an erroneous name should be related in any way to the correct term. The more complete the disconnexion, the more random the errors must be. The less complete the disconnexion, i.e. if some information gets through to the speech area, the more the errors may be related to the correct response. It should be added that in fact the classically anomic patient often makes naming errors not related by sphere or sound.

(3) *Our patient showed inability to choose the correct verbal response when it was offered to him.*—When he misnamed an object we would offer him several choices. The patient would generally not select the correct answer from the group offered. If, however, the speech area is fully disconnected from the right hemisphere, there is no reason why the speech area should select the correct term. It is obvious that the ability to select the correct word depends on there being some connexion between the site of perception and the speech area. The ability to select the correct word from a group when the patient cannot find it spontaneously indicates a lesser degree of disconnexion. Phrased in another, diagrammatic way, one could conceive that when one offers a word to a patient he in some way compares the images or memories aroused by this word with the sensations he is receiving from the object. If there is no site for such comparison, the patient will not recognize the correct word when it is offered. I would like to point out that in fact the obvious nominal aphasic often fails to accept the correct word when it is offered. Furthermore, even when the patient insists that "I know what it is but I can't tell you the name" he may fail to accept the correct name from a group.

(4) *The patient gave descriptions of his "perceptions."*—Thus, when holding a half-dollar, the patient said that it was a cigarette lighter. When asked if it had corners he said, "Oh, yes, there's one, there's another." He would describe incorrectly the object held in his hand in most instances. The same objection applies here as to the previous categories. If the patient's speech area is disconnected from a site of primary perception why should his speech area be able to describe what is going on at the site of the primary perception? Clearly, it should not. The fact that "the patient" (i.e. the speech area) gives a description does not mean that we are getting an actual description of the perceptions going on in another disconnected part of the brain. We must again remember that we are dealing with more

than one "patient" here. The "patient" who speaks to you is not the "patient" who is perceiving—they are, in fact, separate.

(5) Our patient lacked only one of the classical criteria of "agnosia." According to these classical criteria, a patient with "tactile agnosia" should mishandle objects which he has not seen but should respond correctly to the sight of the object (i.e. his behaviour should be opposite to that of the visual agnosic). Our patient in fact handled objects perfectly correctly when blindfolded. It will return in a later part of my discussion of the agnosias to this question of the use of objects.

The Problem of Confabulatory Response

I have discussed in some detail a disturbance which might have been called "agnosic" and tried to show that this disturbance is much more meaningfully described as a naming defect resulting from disconnexion from the speech area. There are certain further implications of this interpretation which I would like to present now. One most important implication is that the "introspections" of the patient as to his disability may be of little or no use to the examiner. The patient cannot "introspect" about the activities of a piece of brain which has no connexion to the speech area. What he tells you is of little value in elucidating the mechanism and may indeed be actively misleading. Indeed, it becomes clear that many of the patient's responses can only be described as confabulatory, i.e. they are attempts to fill gaps in the information available to his speech area; phrased in more conventional terms they are attempts to explain what the patient cannot understand.[1]

These confabulatory responses have been a major source of confusion for many years, particularly in the assessment of difficulties of sensory identification. It is curious that sophistication concerning confabulatory behaviour has been much more widely developed in certain areas than others. When the patient with a severe recent memory defect asserts that the examiner spent the previous evening with him in a bar, we neither ask ourselves why the patient had hallucinations of seeing the examiner the night before nor why he is now having false memories. We see this instead as the verbal filling in of a gap. We discount the attribution by the same patient of his lack of knowledge of current events to the fact that he has never been interested in politics. Similarly, if an aphasic patient tells us that his trouble in speaking is the result of his ill-fitting dentures or a sore throat, we do not take the attitude that he is giving us any very useful insight into his illness but rather that he too is attempting to explain a gap in his performance. An even more extreme example is that of patients with denial of

[1] Dr Edwin Weinstein and his co-workers have been instrumental in stressing in recent years in a series of careful papers the importance of confabulatory responses. The stimulation of his work was a major factor in attracting my attention to this problem.

blindness who may be prepared to describe in detail many of the objects in the room; once we know that the patient is blind, we do not attempt to make use of his "introspections" about his visual perceptions. We do not take seriously this patient's protestations that he has failed in some task because the light is poor or he is not wearing his glasses. Furthermore, we do not assert as an alternative that the patient is really hallucinating.

The "higher" the function impaired, the more readily the patient may succeed in fooling others with confabulatory responses. I recall one patient with pure alexia without agraphia who asserted that his trouble in reading was due to his "blindness in his right eye." The resident staff correctly realized that he meant a right visual field defect when he spoke of a blind right eye. However, they incorrectly accepted the patient's attribution of his reading disturbance to this visual impairment. Only when the discrepancy between reading and object-naming was pointed out to them did they realize that they had too readily accepted the patient's "explanation" of his disability.

There are many occasions in which patients with aphasia, dementia, blindness, or recent memory defects produce confabulatory responses which are recognized. There are probably equally numerous situations in which such confabulatory responses are not recognized as such by the examiner. Most of the conditions called "agnosias" fall into this category. Much time has been spent in the vain attempt to analyse the supposed disorder of perception which has been regarded as underlying the patient's responses.

In summary, the theory here proposed is that most of the classical agnosias are highly isolated disturbances of naming which are the result of disconnexions from the speech area. The prominence of confabulatory responses may incorrectly lead the examiner to believe that he is dealing with a perceptual disorder.

Inability to Identify Colours

The patient of Geschwind and Fusillo (1964) (discussed in an earlier section of the paper) showed pure alexia without agraphia and a failure to identify colours. This disturbance could readily have been called an "agnosia for colours." Yet, analysis of his responses makes it clear that his difficulty could be correctly described as a disturbance in the naming of colours. By all nonverbal criteria he identified colours correctly—he matched and sorted correctly, did the Ishihara test perfectly and even properly matched colours to pictures of objects, yet he failed consistently in verbal naming. It is obviously appropriate to describe his disturbance as a colour-naming defect since by no criterion could he be shown to have a perceptual defect. His pattern of responses also illustrates the lack of usefulness of the classical criteria. Thus he exhibited a very circumscribed naming defect. Only once in many testing sessions did he misname an

object, yet his colour-naming remained consistently extremely poor. He generally failed to accept the correct name of the colour when it was offered. Indeed, when we told him, for example, that something was not grey but red, he said, "Well, it looks grey to me—maybe it's a reddish-grey." On another occasion he said, "You say it's white but it looks tan to me." Again the most parsimonious assumption is that he had a colour-naming defect with confabulatory response. Whenever we asked the patient to give a *verbal* account of the colour experiences of his right hemisphere, he produced a confabulatory response; if we tested the right hemisphere *nonverbally*, we got evidence of perfectly normal colour perception.

I would like to discuss the one instance of poor matching by this patient since the careful analysis of this failure was most illuminating. The patient was given a pile of colour chips which contained chips of two different shades of each of the primary colours. He was shown a chip of one of the two shades of red and told, "This is red. Now pick up all the red chips." He picked each chip up slowly and examined it. He finally separated out all of the chips of the selected colour as well as all the chips of one of the two shades of green. When asked what colour the chips were which he had selected he said, "These are red and these others (indicating the green ones) are a different shade of red." He was then able to separate these chips into two piles. When shown a chip of the *other* shade of green and asked which of the two piles it was most like, he unhesitatingly classified it with the pile of green chips.

There was clearly a sorting error here but full consideration of the patient's behaviour seems to exclude the possibility that we are dealing with a perceptual error. I believe the sequence of events to have been as follows. We had asked the patient to "pick up all the red chips," rather than to "pick up all the chips like this one." He had done the task slowly probably because he did it by picking up each chip and naming it. Having misnamed a chip of one of the shades of green as red, he had then selected the remaining chips of this shade. We may consider this as a sorting error resulting not from failure of discrimination or perception (which his subsequent behaviour in this task excluded, i.e. the rapid separation of the two colours) but as a "secondary sorting error" which resulted from doing the task verbally. Thus, even failures in sorting should be analysed carefully to determine whether they are secondary to misnaming or are truly failures of discrimination.[1]

Classical Visual Agnosia

There are many remarkable accounts of this disturbance in the literature (e.g. Lissauer, 1889; Lange, 1936; Brain, 1941; Macrae and Trolle, 1956;

[1] Pick (1931) pointed out that sorting errors may result from difficulties in verbal mediation in patients with colour-naming troubles.

Ettlinger and Wyke, 1961; Hécaen and Angelergues, 1963). In going through these accounts one is immediately struck by the frequent evidence of preservation of nonverbal visual identification while verbal identification is impaired. One striking feature of many of these cases is the fact that the patient's ordinary behaviour is in marked contrast to the supposed perceptual disturbance. Thus, one may read an account of a patient who cannot "identify" a glass of water and yet a few minutes later picks it up and drinks from it. Much stress has been laid on the fact that a patient may show normal behaviour in a "natural" situation but not in an "artificial" one. Once one abandons the notion of a unitary process of "recognition," it is easy to see that the patient may fail to "identify" an object, i.e. fail to give a verbal account of what is going on in his visual receptive regions and yet be able to respond nonverbally to a nonverbal stimulus. Most of the pathways involved in the nonverbal response may be quite different from those involved in naming. For a thirsty man to respond appropriately to the sight of a glass of water by grasping it and drinking from it is *anatomically* a different task from that of responding to a verbal request for identification. It is certainly reasonable that a naming defect should not produce gross functional impairment; by contrast it would be very difficult to understand how a "higher-order perceptual defect" could leave ordinary perceptual function unimpaired. A similar preservation of function appears in those cases who can draw the object which they cannot recognize (Lange, 1936) a behaviour obviously compatible with a naming difficulty.

The patient of Ettlinger and Wyke (1961) made many errors in visual naming (e.g. "a box" for "a glove," "for cooking" for "a spoon") and many fewer in tactile naming. This case can be used as further evidence against the idea that an aphasic naming disorder must cut across modalities. The data of these authors show that while "agnosic" errors (e.g. "box" for "glove") were more common visually than tactilely, the same was true for "aphasic" errors (e.g. "for cooking" for "a spoon"). A striking feature of this case was the matching of outline shapes without error even though the patient made nine errors in naming outline pictures of ten objects. Certainly a naming defect is the simplest explanation of this disorder. This case was similar in this respect to the earlier case of Brain (1941).

The remarkable case of Macrae and Trolle (1956) presents another example of one of these striking discrepancies. The authors point out that when the patient was pressed with regard to his method of finding his way to and from work, "he could not visualize the street plan or the route." Despite this he did in fact drive two miles a day to work without difficulty. I would suggest that "visualization" here means either developing or describing an internal picture in response to a verbal stimulus—but this will not occur if the visual system is separated from the speech area. Yet, the subject may respond nonverbally to the actual route.

Patients with classical visual agnosias have often been presumed to be

suffering from "perceptual" disorder on the basis of their "introspections" concerning their illness. The case of Lissauer (1889) illustrates how little such "introspections" are to be trusted. Thus, Lissauer comments that when the patient misidentified an object, he would, after learning its true nature, excuse himself by saying that "his eyes had deceived him at first." As Lissauer comments, this statement, coupled with the fact that some of the misidentifications were so curious, might have led the examiner to believe that the patient had been having sensory illusions of some type. But if the examiner then asked the patient whether the object, now that he knew what it was, looked any different from the way it had before, he received a vigorous denial. The patient in fact now asserted that the object had looked the same before but that "his poor memory was responsible for the fact that it had not occurred to him what the thing was called and what it was." Had Lissauer pushed his analysis somewhat further here he might well have been able to see that the "misidentification" was actually only misnaming. Lissauer himself comments elsewhere in the paper, "The patient succeeded . . . in drawing simple objects which he didn't recognize, a striking proof that he was well able to perceive their forms." Similarly, although the patient failed to name colours, he succeeded without any difficulty in matching tasks.

The Lesions of Classical Visual Agnosia

At first it would be tempting to ascribe classical visual agnosia simply to disconnexion of the visual region from the speech area. This interpretation, however, runs into obvious difficulties. Thus, we have already explained in an earlier section that pure alexia without agraphia (with its frequent accompaniment of colour-naming difficulty) is the result of disconnexion of the visual cortex from the speech area. In these cases there is usually destruction of the left calcarine cortex. In addition there is a lesion of the splenium of the corpus callosum which isolates the right visual region from the speech area. It was particularly noted that these patients did not show difficulties in object-naming and often showed retained ability to read numbers. I said in the earlier discussion that it seemed likely that the explanation for the preservation of object naming was that objects could arouse somesthetic and other associations more anteriorly in the right hemisphere; these would then traverse the callosum in the preserved portions anterior to the splenium and thus reach the speech area. For the development of a naming defect for objects, failure to arouse these tactile associations (a visual-tactile disconnexion) is a necessary addition to the disconnexion of visual regions from the speech area.

The lesions found in the majority of cases of visual agnosia support this notion. Most commonly observed are very large bilateral parieto-occipital lesions. There are probably no direct connexions between the visual and the somesthetic cortex so that visual-somesthetic connexions must be

mediated via the intervening association cortex. A large parieto-occipital lesion would effectively cut off such connexions. One can also see the reason for the rarity of classical visual "agnosia." If the lesion is too small, a large enough extent of association cortex is left to make the defect a minor one. If the lesions are too large, the patient may show gross aphasic impairment as well as marked disturbances of visual-motor connexions. In this case his impairment of speech function may make analysis exceedingly difficult. The cases with very extensive lesions might with some justification be regarded as manifesting disturbances of the general ability to manifest visual "recognition" since all outflow channels may be cut off except possibly some primitive and not very extensive pathways descending subcortically.

There is another but much smaller group of visual "agnosias" who have unilateral lesions. This group shows destruction of the left visual cortex. In addition there are always lesions of other structures. One group of these patients shows involvement of the splenium. Others have such extensive involvement of the white matter or of the association cortex in the left occipital and parietal lobes as to have destroyed either the fibres coming from the splenium or their terminations in the left hemisphere (Lange, 1936; Nielsen, 1937).[1] The beautifully described case of Lissauer (1889) turned out to have such a lesion (Hahn, cited by Meyer, 1895–1896) although he himself had expected bilateral lesions.

Why do these cases with unilateral lesions manifest more than pure alexia? It is difficult to be certain and I would hope that more careful study of cases in the future may provide the answer. One possibility is that in some people visual stimuli in the right hemisphere fail to arouse tactile associations or do so weakly. These patients should therefore develop a more extensive naming defect when the right visual regions are cut off from the speech area. Another possibility is that in these cases the lesion in the

[1] A brief note is appropriate here on Nielsen's interesting and important review. In his introduction he points out that many authors have stressed the bilaterality of lesions in most cases of this disorder. He then goes on in his study to confine himself only to the unilateral cases on the grounds that "if in any case a unilateral lesion produced visual agnosia, there can be no point in citing cases of bilateral lesion of the occipital lobes." This argument is of course based on the reasoning that a bilateral case must only be a unilateral case with an extra unnecessary lesion. The fact that probably the *majority* of cases of visual "agnosia" have had bilateral lesions militates against this thesis. Furthermore it should always be kept in mind that many different *combinations* of lesions may lead to the same effect when the physiological mechanisms are kept in mind. He also neglects the possibility of individual differences. The paper of Nielsen is therefore a contribution to the anatomy of the unilateral group of cases but not necessarily to that of the bilateral cases. Many of Nielsen's cases show the discrepancies between naming and other functions that I have cited above although his interpretation of much of the material is different from mine because he accepts the idea of defects of "recognition." He frequently uses as one of his criteria the failure of the patient to *behave correctly* in relation to the "unrecognized" stimuli; at the end of this section on the agnosias I will discuss this problem briefly.

splenium extends further forward than it does in the cases of pure alexia without agraphia. The lesion would thus cut off not only fibres from the visual association cortex but also fibres from the somesthetic association cortex. In the case of Trescher and Ford (1937) the surgeon is said to have divided the posterior half of the corpus callosum; it seems likely that the lesion must have involved more than the splenium. The description of the case would suggest that the patient in addition to being alexic in the left half-field, also could not name objects in that field. This case therefore provides suggestive evidence for the hypothesis that lesions extending somewhat anterior to the splenium may cut off impulses from the somesthetic association cortex and thus prevent visual stimuli from reaching the speech area via this detour.

The Handling of Objects

It was pointed out earlier that one of the classical criteria for a visual agnosia was failure of the patient to demonstrate the use of objects shown to him. It was pointed out that according to this scheme the "aphasic" could show the use of an object shown to him; the "visual agnosic" could not do this but could manipulate the object correctly if it was placed in his hand; the "apraxic" could not perform even with the object in his hand. I have reserved this criterion for special discussion since it appears at first glance to provide a very reasonable basis for separating disturbances of recognition.

This criterion, however, also fails as the mark of a separate category of "recognition" disturbances. In the first place, as I have already commented, some patients who are severely "agnosic" by the other classical criteria listed earlier can in fact respond correctly to the sight of objects. If one simply interpreted agnosias as deficits of "recognition" there should be no reason why correct demonstration of the use of a seen object should be preserved in the presence of a severe disturbance of this type. Furthermore, as I will show in the section on motor disturbances, there are severely apraxic patients who fail to respond to the sight of an object yet handle the actual object correctly when it is placed in the hand. By the classical criteria these patients should be called agnosic. Yet these same patients may be able to name the object and describe its use, behaviour certainly incompatible with a loss of "recognition."

As I will point out in the section on the apraxias, they are also the result of disconnexions. The lesions producing these disturbances separate sensory from motor regions. It is obvious that the kind of lesion which causes classical visual agnosia will frequently although not invariably result in the disconnexion of the visual regions not only from the speech area and from the somesthetic areas but also from the motor region.

This discussion illustrates again the principle that division of phenomena into aphasic, agnosic or apraxic often obscures underlying mechanisms. It

is far better in general to specify the classes of response which are lost or are preserved in response to each class of stimuli.

The Conditions for Confabulatory Response

I have advanced a theory of classical "agnosic" disturbances which views them as naming disturbances which are frequently overlaid with confabulatory responses. Why do these confabulatory responses occur? Weinstein, Cole, and Mitchell (1963) have stressed certain features of this type of response. Let me paraphrase some of their major conclusions: (1) Confabulation does not occur in the absence of a defect; it is, indeed, clearly an attempt to fill in a gap of performance of some type. (2) Confabulation is less marked in the presence of aphasia; this seems reasonable since it clearly is a response of the speech areas. (3) Confabulation is more likely in the presence of some over-all impairment of awareness, such as generalized dementia or clouding of awareness.[1] To these three conditions I would add a fourth (Geschwind, 1963*b*): Confabulation is much more likely in the presence of disease of association cortex or association fibres (either commisural or intrahemispheric) than it is in cases of damage of the primary sensory pathways up to and including the primary sensory cortex. We can combine this with the third condition above to make the following statement: The more demented or clouded the patient, the more likely he is to show confabulatory response in the presence of any defect of whatever origin; the less dementia and clouding are prominent, the more confabulation depends on lesions of association cortex or fibres; the two mechanisms interact frequently. These, of course, are not the only conditions; the influence of personality prior to illness may also be highly important.

Many examples come to mind to support this contention. My personal experience of several cases of denial of blindness illustrates this rule. The first case was that of a man suffering from a septicæmia, markedly febrile, drowsy and confused, who was blind as the result of infection of the anterior chambers of both eyes. Another was a man with a large subfrontal meningioma with advanced optic atrophy and a classical severe frontal syndrome with dementia. In these cases denial of blindness was related to marked general impairment of function of the nervous system. I have seen a confused patient who gave bizarre confabulatory responses when asked to name objects held in the hand. He could not, however, select the objects correctly from a group afterwards. This therefore must have represented the confabulatory response to an actual sensory loss. The same patient gave confabulatory responses to visual field testing when there was no stimulus in the field but gave correct answers to stimuli within the bounds of a normal visual field.

[1] Although I have leaned heavily on the work of Weinstein and his co-workers in these preceding statements, I would by no means wish to suggest that Dr. Weinstein would have used the phraseology I use here.

In contrast with these cases, patients with bilateral occipital infarcts (which probably usually involve much visual association cortex) are likely to manifest denial of blindness without marked dementia. Blindness from peripheral lesions is far less likely to produce such confabulatory denial in the absence of dementia or clouding. Confabulatory denial of a lost limb is uncommon; although the patient frequently has a phantom limb, he nearly always treats it as a phantom. But denial of hemiplegia is common and probably more common in the presence of accompanying lesions of association areas; even if it were insisted that all of these patients show some dementia or alteration of awareness, it is obvious that it is far less than the degree of dementia needed for the denial of a peripheral defect. I have, of course, already cited many examples of confabulatory response in the earlier sections of this part of the paper.

There is some suggestive but not definitive experimental evidence for this hypothesis. Warrington (1962) carried out experiments on visual completion in patients with hemianopias. She found that those patients who were unaware of a hemianopia were likely to "complete" forms across a field defect while those who were aware of their hemianopias did not. Thus, the first group would report a segment of a circle in the good field as "a circle" while the second group would report it as an incomplete circle. She found that this tendency to complete forms across field defects was strongly associated with the presence of a parietal lobe lesion, i.e. in our sense a lesion of association cortex. The anatomical and other data are however not fully convincing. Thus, of the 11 patients who showed completion, 6 had tumours and 3 of the remaining 5 showed mental deterioration. Thus, 9 of 11 had evidence either of widespread deterioration or of a lesion likely to cause more than focal disturbance. (It must be admitted, however, that the six tumours were all parietal in location.) In the group of 9 patients with little completion only 2 had tumours (neither of them parietal) and only one other case of this group was said to have shown deterioration. Thus, only 3 of 9 patients in this group had evidence of dementia or of a lesion with possible widespread effects. It is therefore possible to use Warrington's data in support of a different thesis from the one I have advanced—that patients with dementia or tumours are more likely to develop unawareness of a hemianopia and to complete forms across a field defect. Further studies of this type with greater description of the degree of dementia should help to resolve the issue in favour of one or the other interpretation.

Another paper by Kinsbourne and Warrington (1962) is also suggestive evidence for the thesis I have presented. They found patients with right hemisphere lesions who tended to misread the left halves but not the right halves of words ("novel" for "level," "cucumber" for "encumber"); 4 of these cases showed complete left hemianopias but 2 did not. The authors thought that all of these patients had right parietal disease. It would be

most interesting if it could be shown that this type of confabulatory reading error resulted from right parietal association area lesions, but this would require a control group of patients matched for dementia and with proven nonparietal right-sided lesions who would not show the defect.

Why should such confabulatory responses be less common in lesions of primary sensory cortex and more common in lesions of association cortex? I cannot answer with assurance, but I can propose a highly hypothetical mechanism which is at least suggestive of how such a situation could arise. Since the speech area has in the normal no direct contact with the visual cortex proper, destruction of the calcarine cortex leaves the speech area still innervated by visual association areas. The association areas in this case receive no stimulus from the visual cortex and send the "message" to the speech area that there is no visual "message," i.e. that all is black. This presupposes that the association areas never fail to send a "message" to the speech area and that they always send *positive* messages of light or darkness. The destruction of association cortex or fibres corresponds to a totally unphysiological state—one in which *no* message is received by the speech area. Since this is not interpreted (not being one of the normal states) the speech area begins to react to random messages received from subcortical pathways or indeed responds to its own spontaneous firing. It may respond to *incomplete* information coming over subcortical pathways which are inadequately extensive to carry all the information about the seen object; this may lead to errors which are less bizarre than those in which no information at all gets to the speech cortex. As I have stressed this is a highly speculative mechanism, but certainly not an impossible one.

The Problem of Right Parietal Dominance

It may be useful to speculate briefly on another problem of major interest in the current neurology of the higher functions, the question of the dominance of the right parietal lobe in spatial functions. This problem has been a perplexing one, perhaps because there is said to be a range of disturbances from right parietal lesions unlike those seen with left-sided lesions. On the one hand there is the fairly clear-cut syndrome of inattention to the left side of space, manifested by a tendency to bisect lines to the right, a tendency in copying to omit features in the left half of the picture, neglect of the left side of the body as shown by failure to dress it or even denial of its existence. The corresponding syndrome of neglect of the right half of space seems to be less common. At the other extreme one sees patients with right parietal lesions with the grossest impairments of function apparently not confined to the left half of space. These patients may show gross disturbances in drawing figures or copying involving much more than neglect of the left half of the object and difficulties in dressing not confined to the left half of the body. A common argument has been that since such gross impairments are more common with right than with left-sided lesions, the right parietal region is the dominant one for spatial

functions. However, there is a difficulty with this argument. In the first place the sheer loss of the left half of space in some right parietal lesions suggests that a dominant left hemisphere is being deprived of information from the right hemisphere. By contrast, if the right side were usually dominant then one should frequently see left parietal lesions with neglect of the right half of space; this picture is, in fact, less common than cases with neglect of the left half of space. Thus, in particular, drawings with the right side omitted are less common than those with the left half omitted.

I would like to offer a tentative resolution of this paradox. In the first place, let us consider the possibility that the main effect of a right posterior parietal lesion is to disconnect the right visual and somesthetic cortex from the dominant left hemisphere. (Whether the effects are due entirely to disconnexion from the speech area, I would hesitate to say.) The left side of the body and of space is then "lost." The patient will then respond in many instances by using the technique of confabulatory completion that I have discussed above and for which I have already cited experimental evidence.

The confabulatory response may perhaps be responsible for some of the more severe syndromes with right parietal lesions. If the confabulatory response is very bizarre, then the patient must make a major readjustment of all of space in order to deal with his "experiences." For example, an occasional patient will express the feeling that he has two sets of left arms, one which he sees and another which he feels. If he tries to order this bizarre sensation according to his previous experience he must grossly distort his entire body image. It will be no surprise if he now has trouble dressing even the right half of his body. Similarly, if in copying a picture he has a bizarre confabulatory structure in the left half of the field, he may find it necessary to readjust the right half—albeit unsuccessfully, in order to make his copy satisfactory. As I have already noted, this is a very tentative suggestion which probably needs recasting in a clearer form but I believe it may have the kernel of a new approach to the problems of right parietal syndromes.[1]

[1] It should be noted here that Denny-Brown (1962) also believes the left hemisphere to be generally dominant and rejects right parietal dominance but on quite a different basis from that presented here. Since the initial writing of this paper my attention has been called to the results of Kimura (cited by Shankweiler, 1964 and confirmed by him) who found that in normals melodies were better recognized when presented to the *left* ear (in contrast to the greater efficiency of the handling of digits by the *right* ear). These differences in melody recognition, although significant, were small. This suggests that although the right hemisphere may be more *efficient* in certain perceptual tasks, it is not *dominant* in the sense that the left hemisphere is dominant for speech. This view is further supported by Shankweiler's observations that even the presence of a right epileptogenic lesion shifts the balance in favor of the right ear in melody recognition. In any case even the confirmation of some greater efficiency of the right hemisphere in certain tasks would not invalidate the mechanisms I have suggested for some of the dramatic right-hemisphere syndromes.

Let us consider briefly the vexing problem of why a left parietal lesion less often produces neglect of half of space than does a right parietal lesion. In the first place the right hemisphere is probably always at a disadvantage in relation to the left since the pathway from any part of it to the speech area is probably at least one neuron longer than the pathway from the corresponding part of the left hemisphere (there must be at least one extra neuron to take the impulse across the corpus callosum). One extra neuron may be of little import when added to a short path but probably the extra degradation of the stimulus becomes more important the longer the route. Any "detour" around a lesion used by the right hemisphere always suffers this extra disadvantage and therefore makes it likely that the right hemisphere will be more completely disconnected. If in addition the right hemisphere responds less well than the left to stimuli (see the earlier discussion on dominance), it is subject to a further disadvantage. Thus disease may simply aggravate the normal disadvantage of the right hemisphere in being further away and responding less well to stimuli.

There is another mechanism which may contribute to the special disadvantage of the right hemisphere. The speech area and other dominant areas of the left hemisphere conceivably might receive *some* sensory information directly from the left thalamus in the situation in which the parietal association cortex was destroyed. This short cut is however not available on the right; the thalami have no significant commissural connexions (the massa intermedia is of minor significance and in any case is very frequently absent) and therefore impulses from the right thalamus must still reach the cortex of the right side before crossing to the other side.[1]

According to this view of right-sided association area lesions, they are more likely to lead to disability and to confabulatory response than those of the left side because the normally poorer linkage of the right side to the speech area, and possibly to other "dominant" areas of the left hemisphere, is further weakened by the lesions. This approach may contribute to further understanding of some other phenomena which occur more frequently in right hemisphere lesions. Thus, Hughlings Jackson (1880) thought that *déjà vu* was more likely to result from right than from left cerebral lesions. Bingley (1958) confirmed this result. Mullan and Penfield (1959) found that visual illusions and *déjà vu* phenomena arose predominantly from the hemisphere nondominant for speech. Cole and Zangwill (1963) also found a predominance of *déjà vu* from the minor hemisphere although the predominance was not quite as great as in Mullan and Penfield's series. Teuber, Battersby and Bender (1960) found seizures

[1] Weinstein, Cole and Mitchell (1963) argue that denial is less common in left-sided lesions because speech is less available; while this view, in my opinion, explains many of the phenomena, it does not deal fully with nonverbal aspects. To round out the theory it must be assumed, as I have already commented, that left dominance is for more than speech.

involving visual experiences in 15 patients in all; there was a definite preponderance of right hemisphere lesions. These authors cite the study of Hécaen and Badaraco who found that 14 of 16 cases with visual experiences had right hemisphere lesions.

I would suggest that perhaps this tendency of the minor hemisphere to give rise to such illusions is not the result of localization of emotion or memory in the minor hemisphere but another example of the increased tendency to confabulatory response in cases of lesions of the association cortex on the right side. The reasons for the right-sided predominance would be the same as those given above.

It is interesting to note what Hughlings Jackson (1880) said about this predominance of lesions in the right hemisphere in producing what he called intellectual auræ: "These 'dreamy states' . . . cannot be owing to an epileptic discharge. It would be a remarkably well-directed and distributed epileptic discharge which would give rise to the exceedingly compound mental state of being somewhere else . . . I submit that (such an elaborate physical state) is owing to but slightly raised activity of healthy nervous arrangements consequent on 'loss of control' . . . possibly of some in the *cerebral hemisphere opposite the one*,[1] which I believe to be nearly always the right, in which the discharge begins. . . ."

I would like to cite one further recent piece of work to illustrate that it may be possible to reinterpret certain studies which apparently support the idea of right hemisphere dominance for certain functions. I have chosen only one result in one of the many important papers principally by Milner and Kimura published from the psychological laboratory in Montreal. Kimura (1963) used a test in which a subject was shown a preliminary series of picture cards. After this he was presented with a test series of 140 cards; he was to reply, "Yes" if he thought he had seen the picture in the preliminary series, "No" if he thought that he had not. The total score was the number of correct recognitions minus the number of false positive responses. The group with right temporal lobe lesions scored significantly worse on this task than the group with left temporal lesions. Further analysis of these data, however, showed the interesting fact that the number of correct "yes" responses was almost the same for both groups but that nearly all the difference in scores was due to the large number of false "yes" responses by the right temporal group.

Could this not be interpreted as another example of confabulatory response resulting from a lesion of right hemisphere association cortex rather than as a special deficit of the right hemisphere in the retention of unfamiliar material? Kimura gives two reasons why the patients with right hemisphere lesions cannot be interpreted as having "a general tendency to make false recognitions." The first argument is that in an auditory memory task the right-temporal subjects showed no more tendency than

[1] The italics are mine (N. G.).

the left temporal to give false positives. But since I have already given several examples of confabulatory response confined to a *single* modality, this argument would not rule out that the major effect of the lesions was to produce confabulatory response only to unrecognized visual stimuli. The other argument is a somewhat stronger one. Kimura points out that in another task, the overlapping Nonsense Figures test (where immediately after being shown a nonsense figure tachistoscopically the subject is required to select it from a group of figures on a card) the right temporal group performed more poorly than the left, despite the lack of any penalty for false positives. Inspection of the data shows, however, that the significance level on this test for differences between the post-operative right and left temporal groups is between 0·01 and 0·02 while on the recurring figures test the significance level for the difference between the groups was less than ·001. Furthermore, the difference between the post-operative groups on the nonsense figures test was less than 2 items. By any standard this difference appears far less impressive than the tendency for the right temporal group to produce false positive responses on the recurring figures test. Even if one were to accept the result on the nonsense figures test it would argue for only a slight, if significant, superiority of the right as against the left hemisphere on this task; it would not support the idea that the right dominance for this activity was anywhere near as marked as left dominance for speech. In any case it is obvious that many further careful studies of the type carried on by the Montreal workers will be needed to settle this issue as to whether the right hemisphere manifests dominance in any sphere.

Visual Imagery

Related to the problem of the agnosias is the problem of loss of visual imagery. Wilbrand's (1887) monograph on psychic blindness actually concerned itself primarily with patients in whom visual imagery was lost. I have already commented briefly in discussing Macrae and Trolle's interesting case on the patient who could not "visualize" a route but could travel it correctly. The word "visualize" has two connotations. We may, as it were, see a picture in "our mind's eye," and can then say that we are visualizing. At a simpler behavioural level we may describe an absent scene. A classical notion was that visualization was an activity of the visual association areas. The evidence for this is suggestive but by no means complete. The peripherally blind can indeed describe absent scenes and say that they have internal visual imagery. Would a patient with a pure destruction of the visual cortex proper bilaterally still retain imagery? The answer to this question is not known.

Loss of the ability to describe an absent scene might reasonably result from a disconnexion of the visual regions from the speech area, as probably was the case in MacRae and Trolle's patient. That this failure to give a

verbal account need not be associated with a loss of visual memories is also illustrated by the same patient who could in fact correctly drive to work although he could not verbally describe the route. Was this patient capable of developing inner pictures? There would be no way of finding this out by interrogation since if the region in which such pictures were taking place were disconnected from the speech area, we would be unable to get a verbal account from the patient. The question is not a purely academic one since the eventual development of more advanced physiological techniques may settle some of these questions without recourse to a verbal account from the patient. This would be analogous to recent work on dreams for which objective criteria are now being developed.

The question of dreams and their locus is related to that of waking visual imagery. Are dreams functions of the visual cortex or of the visual association areas? These problems of imagery and dreams deserve further study using both standard clinical testing procedures as well as some of the new techniques employed so effectively in the study of dreaming.

V. The Mechanisms of the Apraxias

Disconnexion from the Speech Area

The term "apraxia" in its modern usage goes back to Liepmann's (1900, 1906) employment of the term in describing the classic case of the Regierungsrat. Despite the great complexity of the case, Liepmann devised such remarkably appropriate methods of testing and analysed his findings so adroitly that he was able to predict the patient's lesions. At the patient's death two years after the publication of the paper containing Liepmann's clinical description, the post-mortem confirmed his predictions; subsequent study of the material in whole-brain sections established in detail the correctness of his ideas. The case has often been criticized as having too many lesions to be useful. It is difficult to understand this point of view when it is appreciated that the prediction of the loci of all the lesions was made in advance despite the fact that no similar patient had previously been described. These papers therefore differ importantly from those in which the correlation of the clinical picture with the lesions is only made post mortem.

In a group of papers published in the first decade of this century, Liepmann established the clinical pictures of apraxic disturbances from lesions in the supramarginal gyrus region and from lesions of the corpus callosum. Since that time Liepmann's conclusions have been amply confirmed. Even such workers as von Monakow who purported to be highly critical of Liepmann actually confirmed his results. It is of interest that Kurt Goldstein (1908) was one of the first after Liepmann to publish a case of callosal disconnexion with motor disturbances.

Liepmann's analysis of the Regierungsrat was based on a disconnexion

approach. Unfortunately the very fact which makes Liepmann's accomplishment so remarkable, namely, that he was able clinically to separate out very precisely the patient's many disturbances into a small group of functional impairments, each correlated to a particular lesion, makes the case a very difficult one from the heuristic view. I will, therefore, use instead as a case illustrative of the mechanisms of apraxia the patient reported by Edith Kaplan and myself (Geschwind and Kaplan, 1962), the mechanism of whose disturbance is much simpler. The reader is again referred to the original paper for detailed discussion of the findings.

Our investigation of this case followed on Mrs. Kaplan's discovery that the patient could write correctly with the right hand (despite the presence of a severe grasp reflex and mild pyramidal disturbance in this hand) but not with the left hand which showed no elementary motor disturbance. In addition to this disturbance of writing with the left hand, we found that the patient consistently carried out verbal commands with the right hand but frequently failed to carry them out with the left hand. In classical terms the patient showed an "apraxia of the minor hand." By further testing we were able to go beyond this bare statement and found it possible to delineate the mechanisms of these disturbances.

The disturbance of writing with the left hand could be shown to be aphasic. Thus, when the patient wrote to dictation with the left hand he did sometimes produce an illegible scrawl. On many other attempts, however, he would produce perfectly written but incorrect words (e.g. "run" for "go," "yonti" for "yesterday"); furthermore he misspelled words when typing with his left index finger and could not correct the errors which he was, in fact, able to recognize. By contrast he could copy correctly with the left hand, but he could not with this hand turn print into script although he could copy both print and script as such. This series of studies excluded any elementary "motor" disturbance as being at the root of the left-sided agraphia.

He often failed, as we have noted, in carrying out verbal commands with the left hand. He could, however, copy the movement if it was made before him by the examiner. In addition, he would, given an object, handle it correctly with the left hand although he had failed to show how the object would be used in response to a verbal command alone. Thus, "Show me how to use a hammer" resulted in a failure of movement or an incorrect movement; he could, however, imitate the examiner's movements or could use the actual hammer correctly. With his right hand he showed none of these difficulties.

We interpreted these disturbances as the effects of disconnecting the right motor cortex from the speech area. The post-mortem showed, as I have already noted earlier, an extensive infarction of the corpus callosum which must be regarded as the cause of these symptoms. The aphasic writing with the left hand and the failure to carry out verbal commands

with the left hand could quite easily be explained as a result of disconnexion of the right motor cortex from the speech area. The ability to copy writing correctly but slavishly with the left hand; the ability to imitate seen movements with the left hand; the capacity to handle objects correctly with the left hand, all illustrate that the right hemisphere could perform correctly when no information was required from the opposite side. By contrast the inability to "copy" print into script is an expected one since such a transformation implies the participation of the speech areas. Certain movements in response to verbal command which involved *both* the right and left sides simultaneously were preserved as were facial movements to command. I will deal subsequently with the analysis of these two special classes of movements.

The case has several features worthy of comment. It illustrates that the designation "apraxic" is an inadequate one unless the stimulus conditions are specified. The left hand in this patient was apraxic to verbal command but not on imitation or object handling; the *right* hand failed to perform correctly when a response was demanded from this hand to somesthetic stimuli applied to the *left* hand. Rather than use the term "apraxia" it is therefore preferable to specify the stimulus-response combinations which fail.

It is not possible to assert that the failures of this patient resulted from a general conceptual disturbance or a failure of abstract attitude. The patient could not, indeed, pretend to perform an act with the left hand, but he could pretend with the right hand! It would seem highly unlikely that a disturbance of conceptualization or of the abstract attitude could be confined to one-half of the body. "Dementia" as an explanation runs up against the same difficulties. Hysteria or malingering cannot, of course, be seriously considered at all.

The case also points to the probable incorrectness of the classical distinction between aphasic and apraxic agraphia. Many writers have assumed that agraphia confined to the left hand must be "apraxic." According to this argument an apraxic agraphia is the result of failure to form the individual letters properly and should result in a pattern of meaningless loops and scrawls. Further support for the distinction between aphasic and apraxic agraphia came from the argument that aphasia could not be confined to one limb. Our case, however, showed both "apraxic" and "aphasic" types of writing defect in the left hand. In the early stages his writing was in fact usually but not invariably a totally illegible scrawl. But, particularly after some improvement had occurred, he produced perfectly written words which were incorrect, e.g. "yonti" for "yesterday"; his failures at typing were, of course, also misspellings. It would not be possible to term these other than aphasic errors. It seems unnecessary to assume the presence of two separate writing disturbances in the patient. Both types of error (i.e. incorrectly formed letters and aphasic words) can

easily be seen as resulting from a disconnexion between the speech area and the motor cortex.

I have also made no attempt to deal with this disability in terms of the classical grouping of apraxias into limb-kinetic, ideomotor, and ideational which Liepmann (1905*a*) advanced. It would lead us too far astray to discuss this classification in any detail in this paper. It is my opinion that this classification has not been useful and that it has in fact frequently proved very confusing. It is interesting that the classification plays almost no role in Liepmann's earlier classic discussions of the Regierungsrat and even in the later writings is clearly secondary to the intimate discussion of mechanisms. This formal listing of types of apraxia probably had in practice the unfortunate effect of overshadowing Liepmann's much more important detailed analyses of the mechanisms underlying disturbances in motor performances.

The occurrence of such unilateral agraphias is also of considerable interest when the hand involved by the agraphia has been the one generally used by the patient for writing. Thus, Nielsen (1946) described the remarkable case of a man who had been taught to write with his right hand but used his left hand for all other skilled movements. He suffered a transient left hemiplegia; this was accompanied by loss of speech, strongly suggesting that the right hemisphere was dominant for language. Some months after recovery from this episode he developed a paralysis of the right leg and lost the ability to write with his right hand. He could, however, write with his left hand although he had not used the left hand previously for this activity. In interpreting this case one must assume that although the patient had always written with the right hand, the left hemisphere had, in fact, always been passively under the control of the speech area in the right hemisphere.

This case is also useful as a comment on an old idea, that cerebral speech dominance is much weaker in illiterates and that the act of writing, being unilaterally performed, influences the opposite hemisphere (*see* Critchley, 1962, for a brief review of the history of this idea). Cases like the one just cited, however, cast serious doubt on this possibility since it is clear that Nielsen's patient had not succeeded in establishing speech in the left hemisphere. The case also makes somewhat unlikely the old suggestion that training in ambidexterity makes a severe aphasia less probable. This, of course, is not in conflict with the assertion that those who are naturally ambidextrous have milder aphasias (Zangwill, 1960).

Extension of the Theory of the Apraxias

In the preceding section I have developed the theory of the simplest type of apraxia, that due to disconnexion of motor regions from the speech area and from other sensory inputs. The interpretation of the case of Geschwind and Kaplan is clear: callosal disconnexion in man prevents the right

hemisphere from carrying out language tasks and also from performing those tasks for which the stimulus comes from non-language sensory areas of the left hemisphere. This theory depends on the assumption that the right hemisphere will deal with non-language tasks correctly as long as appreciation of the stimulus and production of the response both depend on the right hemisphere itself. The patient of Geschwind and Kaplan exhibited independence of the right hemisphere in non-verbal tasks; this hemisphere functioned successfully in imitation of movements, object handling and slavish copying.

A deeper search into the literature and further clinical observations soon convinced me that the independence of the right hemisphere in non-language functions which was manifested by our patient is not universal and may indeed be the exception. It was the work of Liepmann which again contains the earliest and probably the best evidence for this view.

The first description of the clinical picture of extensive disconnexion of the corpus callosum is contained in the paper of Liepmann and Maas (1907). Their patient (Ochs) had a right hemiplegia which at post-mortem was found to be due to a lesion of the pyramidal tract in the left pons. The patient could carry out many actions very well with his left hand, e.g. buttoning his coat if the hand was placed on the first button, or carrying a glass of water (if it was placed in his left hand), to his lips. He failed to write with the left hand; in fact, he could not even copy. When given anagram letters he could not form his name with his left hand; instead he collected all the m's (yet when asked, he could correctly say the letters of his name). He made many errors in carrying out verbal commands with the left hand but selected the correct response from a series of movements made by the examiner. He did not, however, improve on imitation. This patient frequently mishandled objects placed in the left hand. At post-mortem there were found the already mentioned lesion in the pyramidal tract in the pons as well as an infarct of the corpus callosum which spared the splenium. Other patients with callosal lesions have shown a similar disturbance to that shown by Ochs. Not only can they not carry out verbal commands with the left hand, write with this hand, or form words with anagram letters (in which respects they resemble the case of Geschwind and Kaplan), they also show significant impairment in imitating seen movements with the left hand and difficulties in object handling with this hand. The case of Bonhoeffer (1914), for example, showed these deficits. Why do the cases fall into two such groups? It is interesting that Maas (1907) himself called attention to this problem. His explanation was that it depended on the intensity of the callosal lesion, but I do not believe that this is correct.

Because the first case of "motor" disturbances due to callosal disconnexion seen by Liepmann and Maas showed the more extensive syndrome of left-sided apraxia to verbal command, on imitation and in the handling

of objects, these authors regarded this clinical picture as the "normal" syndrome of callosal disconnexion. Liepmann explained the data by assuming that the left hemisphere is dominant not only for speech but also for movement. This dominance was assumed to manifest itself trivially in the usual greater dexterity of the right hand; a more important aspect of this dominance, however, was that the left hemisphere contained the "memories" of movements.

Evidence from other types of patients support Liepmann's contentions that the left hemisphere is dominant for more than speech. Thus, Liepmann (1905*b*) described the syndrome of apraxia of the left hand in patients with right hemiplegia and aphasia ("sympathetic" dyspraxia as it is sometimes called). I have reinvestigated this problem (Geschwind, 1963*a*) and been able to confirm Liepmann's observations in detail. This syndrome is of special interest both because of its great frequency (it is, in fact, much the most common cause of apraxic disturbances) and its theoretical interest. Liepmann found this syndrome in 14 of a group of 18 patients with right hemiplegia and severe aphasia. By contrast it occurred in only 6 out of 23 right hemiplegics without severe aphasia. It did not occur at all in the right hands of a group of left hemiplegics nor in a group of senile and demented patients. These controls effectively remove the possibility that the syndrome is the non-specific result of brain damage or dementia. They also show its close relationship to lesions producing aphasia. The fact that it did not appear in all the hemiplegic aphasics and that it appears occasionally without aphasia suggests that the relationship is one of anatomical propinquity of lesions; the apraxia frequently accompanies the aphasia but is independent of it.

The syndrome of sympathetic dyspraxia is essentially the same as that described by Liepmann as occurring in the left hand in callosal lesions. The patient carries out verbal commands either very poorly or not at all with the left arm. Demonstration by the examiner helps in some instances but in most cases little or not at all. Usually the actual handling of objects in the left hand is significantly better than movements to command or in imitation of the examiner, but major failures in the handling of objects do occur in a significant minority of these patients.

Several interpretations of this syndrome can be excluded. I have already noted the impossibility of attributing this picture nonspecifically to brain damage or dementia. One obvious interpretation is that since these patients are generally aphasic, their failures to respond to verbal command are the result of comprehension deficit. Liepmann showed in several ways that this interpretation is untenable. In my cases I have also been able to rule this out. Thus, one of my patients showed this syndrome in especially clear form, failing very badly on verbal commands and imitation and also doing poorly in object handling. He had a marked limitation of speech, being able to produce only single words and never producing a sentence. He

could, however, answer questions very well with single words. He failed to carry out very simple commands such as to point to the floor but correctly answered verbally much more complex questions as "What occupation were you engaged in before you became ill?" and "Can you name some tools used by carpenters?" Furthermore, the patient could answer specific questions about the task. When asked, "Do you know how to use a hammer?" he said, "Nails" but could not show how a hammer was used. He could, however, indicate when the examiner had made the correct movement although he could not produce it himself. In other cases a strong clue that comprehension is intact is given by the fact that the patient may respond with a movement that is a clear distortion of the correct response. For example, a patient exhibiting this syndrome when asked to salute may place his hand in the proper attitude but several inches in front of his forehead. These facts all contribute to rule out incomprehension as the cause of the failures. They also highlight a very important aspect of the examination of aphasia. Many textbooks suggest examining for comprehension by means of the ability to carry out verbal commands. It is obvious from the results I have cited that while the ability to carry out verbal commands indicates retained comprehension, its absence does not exclude the preservation of excellent comprehension.

The fact that my patient could give good single word verbal responses while failing to carry out verbal commands probably rules out another possible interpretation. Some would argue that aphasia is not a disturbance of speech but one of symbolization and that my patient was incapable of symbolic manifestations in any form. The patient would under this interpretation be regarded as having lost gesture as well as spoken language. However, aside from the difficulty of regarding some of the movements involved as symbolic, it would be necessary to assume in my patient that *non-verbal* symbolization was worse than *verbal* symbolization! Once one admits the separability of the disturbance in movement from the disturbance in language, the concept of a general asymbolia loses its value.

Liepmann felt that this group of patients consisted predominantly of cases with lesions near Broca's areas as evidenced by their aphasic symptomatology (paucity of speech with good comprehension). He thought that the lesions probably would be found in the white matter immediately beneath Broca's area but above the internal capsule. A lesion in this location would involve not only descending fibres but would also destroy fibres going into the corpus callosum and thus to the opposite side. In those brains which he had had the opportunity to observe at post-mortem he did find such subcortical lesions. He did not make clear exactly from what part of the cortex of the left frontal lobe the callosal fibres originated, a point of importance to which I will return. It is clear that he thought the lesion resulted in disconnexion of the right motor region from some portion of the left frontal region; the lesion was regarded by Liepmann as cutting

off the right hemisphere from the memories of movements stored on the left side.

The Apraxias of the Supramarginal Gyrus Region

Further details of the above mechanism can be still further elucidated by considering another group of "apraxic" patients, those with lesions of the left supramarginal gyrus region. Liepmann (1900, 1906) first described this syndrome in the famous case of the Regierungsrat. The general picture which emerges from the literature is a consistent one. Unilateral lesions penetrating deeply in this region may lead to an apraxia which is either equal bilaterally or worse in the right hand. Except for its bilaterality, it is similar in its pattern of disabilities to that usually seen in the two preceding conditions (apraxia from a callosal lesion and "sympathetic dyspraxia"). The patient performs badly to verbal command, imitation of the examiner is usually poor and object handling is involved, but less often. Liepmann stressed that the responsible lesion was one which cut off white matter connexions (lying deep to the supramarginal gyrus) between posterior parts of the left hemisphere and motor regions. The retained connexions on the right side would not have been able to substitute, in most cases, since there had been no learning on the right side. The lesion in the supramarginal gyrus would cut off connexions running from Wernicke's area to motor regions via the arcuate (superior longitudinal) fasciculus and also connexions from the visual region to the motor area.

There is a problem raised by this simple disconnexion hypothesis. This lesion deep to the supramarginal gyrus should disconnect Wernicke's area from motor regions and the visual region from motor regions; the failure of the patient to carry out movements to verbal command or on imitation is therefore understandable. Why should movements be impaired in the actual handling of objects? There are several possible explanations, but we cannot be sure which, or indeed, whether any of them is the correct one. Liepmann suggested the following mechanism. He asserted that even the manual handling of objects was frequently learned "visually." One can cite simple examples which are suggestive. Many acts, such as combing the hair, may be routinely carried out under visual control. When they are done without vision Liepmann's suggestion would be that they are done with "visual mediation." In other words cross-modal transfer would be involved in the learning of many tasks, and the performance under tactual control would depend on transfer from visual learning.

Liepmann brought forward as evidence for this view the pattern of *retained* responses to somesthetic stimulation. He pointed out both for the Regierungsrat and Ochs that while they handled most objects poorly, they performed well on somesthetic stimulation in those tasks which were highly overlearned and which were usually done independently of vision. Thus, the Regierungsrat and Ochs both failed in some very simple object

manipulations, yet were able to button their clothing blindfolded so long as the hand was first passively placed on the garment. Liepmann attributed this to the ability of the isolated sensory and motor cortex to do this highly overlearned task without the mediation of vision. Liepmann made use of the preservation of such movements as important evidence. It enabled him clinically to rule out any sort of elementary motor or sensory disturbance since these would be incompatible with the deft buttoning of a coat while blindfolded. This fact emphasizes again the point that "apraxia" is not a unitary disturbance since under appropriate conditions these patients could carry out complex motor tasks.

There is another possible explanation for the difficulty in object handling. Liepmann had felt it necessary to account for the fact that the patients could not carry out all tasks of object handling correctly even though the primary sensory and motor cortex retained their direct connexions. We know that there are connexions via U-fibres between the postcentral and precentral gyri. However, are these connexions used for the learning of complex tasks under somesthetic control? It is not at all unreasonable to assume that for the learning of such complex tasks the pathway from primary sensory to primary motor cortex may be via association cortex, as in the case of connexions of other modalities to the motor system. The pathway from sensory to motor cortex would thus run from sensory cortex via somesthetic association areas and then would travel forward to the motor region. If this were the path then a lower parietal lesion might well involve part of this somesthetic-motor pathway. Sparing of this pathway in its entirety might leave object handling totally unaffected. If this explanation were correct then one would have to assume that the preservation of some movements depended on their having been overlearned, i.e. on their being so redundantly represented in the somesthetic association cortex that partial lesions left them unimpaired. This problem of the factors affecting movements made under somesthetic control is one which obviously deserves further study.

Liepmann repeatedly insisted that the critical lesion in the region of the supramarginal gyrus involved not the cortex but the white matter running beneath the gyrus. An alternative explanation would be that the "memories for movements" were in the supramarginal gyrus region, i.e. that the cortex was involved. Against this and in favour of the notion of white matter disconnexion is the fact that lesions *anterior* to the left Rolandic fissure produce apraxia on the left side of the body despite an intact left supramarginal gyrus. One would expect that, if the *cortex* of the left supramarginal gyrus were the important structure and were not involved by the lesion, the impulse could travel across the callosum to the right supramarginal gyrus and thence forward to the right motor region. In this instance no apraxia should be seen. The other possible pathway for control of the left side of the body involves a path running beneath the left

supramarginal gyrus to the left frontal lobe and from here to the right hemisphere. In this case apraxia of the left side could occur with a lesion anterior to the left supramarginal gyrus.

The sinistral unilaterality of the pathway in most people for the carrying out of acts by the motor cortex under sensory stimulation is emphasized by the rarity of well-attested cases of left-sided apraxia occurring as the result of an isolated right parietal lesion. Foix (1916) emphasized right parietal lesions as a cause of left-sided apraxia, but his evidence is not convincing. He even said that "Liepmann's principal case" had a right parietal lesion, but since this statement applies neither to the brain of the Regierungsrat nor that of Ochs, we may dismiss this assertion as simply being incorrect. Hécaen and Gimeno Alava (1960) have discussed this problem. They found 16 cases, 3 of their own, the remainder from the literature, in which patients with apraxia of the left side had clinical evidence of a right hemispheric lesion. However of these, 11 almost certainly also had lesions of the left hemisphere. Among the remaining 5, 2 were known left-handers. Of the remaining 3, 1 came to post-mortem and showed no involvement of the left hemisphere. If one can accept these findings it would appear that a right parietal lesion causes apraxia of the left hand only exceptionally unless there is an associated left hemispheric lesion. This conclusion is further borne out by the study of Ajuriaguerra, Hécaen, and Angelergues (1960). If we consider only those cases in their series which are relevant to our discussion, we find that they had 58 cases of apraxia (divided by them into 11 cases of ideational apraxia and 47 cases of ideomotor apraxia), of which 48 occurred in their 206 cases of left-sided post-Rolandic lesions, and 10 occurred in their 55 cases of bilateral lesions. There was not a single case of apraxia in their 151 cases of right-sided post-Rolandic lesions.[1] I would in fact expect that there exist occasional cases in which a right supramarginal gyrus lesion might lead to an apraxia of the left arm on imitation and object handling (but possibly not in response to verbal command). This unusual situation would exist only in those patients in whom the right hemisphere was relatively independent.

I have already noted that deep to the supramarginal gyrus are fibres from the visual association areas running into the frontal lobe and fibres from the speech area (which is, of course, auditory association cortex) also coursing to the frontal lobe. These are probably the pathways by which motor responses are carried out in response to complex visual or auditory stimulation. Where in the left frontal lobe do they terminate and by what precise pathway does stimulation get across to the opposite motor region?

One's first inclination would be to assume that these pathways terminated in area 4, the primary motor cortex. But again, the rule of Flechsig

[1] Let me stress that I am confining the term apraxia to the sense in which Liepmann used it. I am not including the cases separately tabulated by Ajuriaguerra *et al.* as "constructional apraxia" and "dressing apraxia."

(1901) comes into play. The motor cortex, at least in the primate, receives no direct fibres from the visual association cortex. Chusid, Sugar and French (1948) produced a most instructive study of the frontal connexions of the visual association cortex in the macaque which brought out clearly the fact that many of these important connexions arise from the tissue buried in the depths of the lunate sulcus of the occipital lobe. They found with strychnine neuronography that the anterior bank of the lunate sulcus strongly fired the cortex immediately anterior to itself. The only distant region where they found strong firing was the cortex of both banks of the arcuate sulcus of the frontal lobe and the cortex immediately anterior and posterior to this sulcus. There was no firing in the motor cortex proper. The posterior bank of the arcuate sulcus in turn fired strongly into areas lying immediately posterior to itself including the *homolateral* area 4 and also strongly fired the *contralateral* arcuate sulcus region. Bonin and Bailey (1947) report that FA (area 4) received fibres from FB (lying above the superior half of the arcuate sulcus, often called area 6) and from several areas in the parietal lobe but not from any other part of the frontal lobe. Furthermore, FA had no callosal connexions in the primate except for some restricted parts of the trunk and lower face regions. Their results and those of Chusid, Sugar, and French thus are complementary. Similarly, Bailey, Bonin, and McCulloch (1950) state that in the chimpanzee the most numerous afferents to FA come from FB and PC (the postcentral gyrus). The pattern of callosal projection is the same as in the macaque.

Krieg (1954) studied the efferent connexions of the frontal lobe in monkeys by the Marchi method. He found that each area 6 has strong callosal connexions to the opposite area 6. In addition each portion of area 6 sends numerous fibres to the part of area 4 directly behind it. Krieg's findings are therefore in keeping with the physiological findings of the workers cited above. Krieg did find callosal fibres from area 4. He comments, however, that the number and calibre of these fibres is very small and his illustrations of the actual lesions and the consequent degeneration support this assertion. Krieg notes that his anatomical findings are in keeping with the poor callosal responses obtained from area 4 by Bailey, Bonin and McCulloch (1950).

We might guess from these data that connexions between visual and motor cortex run as follows: from the visual cortex proper to the visual association cortex to area FB (roughly area 6) and thence to the motor cortex. Area FB thus becomes the association cortex of the motor system. Similarly, the callosal connexions of the motor system would have to be via area FB.

The view that the premotor cortex acts as the association area of the motor cortex receives support from the connexions to the motor region of the auditory system. Thus, Sugar, French, and Chusid's (1948) study showed that the important frontal projections from the supratemporal

plane are to the cortex lying within the depths of the arcuate sulcus and to the cortex immediately posterior to this sulcus, in particular to the regions behind the lower limb of the sulcus. In other terms the projections from the supratemporal region are to areas 8, 6b (lower half of area 6), and 44; there are no direct connexions from the supratemporal plane to area 4. The earlier study of Ward, Peden, and Sugar (1946) which was the first to discover afferent connexions to area 6 had also revealed the connexions from the supratemporal plane to this part of the frontal lobe.

Thus, it would appear that the auditory system like the visual system projects to this "motor association cortex" lying anterior to area 4, and that this region in turn projects to area 4. It seems reasonable that this is the pathway by which motor tasks are carried out to auditory stimulation.

These studies all support the theory that connexions from primary sensory areas to the motor cortex involve a multisynaptic pathway which travels by way of the association areas adjacent to the sensory areas in question and the "motor association areas" lying anterior to the motor cortex proper. The startling experiments of Welch and Stuteville (1958) are readily explained by this theory and also constitute further evidence for it. These authors placed small lesions in the depths of the posterior part of the superior limb of the arcuate sulcus in monkeys. Confirming earlier results by Kennard and her co-workers (summarized in Kennard, 1939) they found that these animals disregarded visual stimuli on the side opposite the lesion, and indeed even collided with objects on that side. The animals did not respond to auditory stimuli coming from the side contralateral to the lesion by turning towards the stimulus (as a normal monkey would); instead they turned towards the side opposite the stimulus. Finally, these animals exhibited little response to tactile or painful stimuli on the side opposite the lesion. These animals showed a marked poverty of movement on the involved side. Tactile and visual placing reactions were lost on that side. Recovery from this dramatic syndrome began in five to seven days and was complete in two weeks.

Welch and Stuteville state, "How vision, hearing, tactile sensibility, movement and placing reactions are so seriously impaired by a lesion of the cortex which does not directly interfere with any of the several receiving areas or the motor area of the cortex is difficult to understand." The results, however, become easy to understand when one considers that the area involved by the lesion is in fact part of the pathway from all sensory modalities in one hemisphere to the motor cortex of that hemisphere. The lesion therefore effectively disconnects the motor cortex of that side from all sensory stimuli. Why does the animal not respond with the hand opposite the *normal* hemisphere to stimulation on the side opposite the damaged hemisphere? I would suspect that it is probably because the lesion has also cut off the origin of callosal fibres between the two areas 6. This explanation of the Welch and Stuteville data may well apply to other

studies in which lateral frontal lesions have been said to lead to difficulties in sensory discrimination (e.g. Weiskrantz and Mishkin, 1958)—the real deficit may be in motor response.

> Some aspects of the Welch and Stuteville experiments deserve brief comment. Ettlinger and Kalsbeck (1962) found a loss of tactile placing responses on the side contralateral to either a lesion of the primary somesthetic cortex or of the parietal association areas. Welch and Stuteville found that these responses disappeared in their experiments. The normal pathway for a tactile placing reaction would appear to travel not via the U-fibres connecting the primary somesthetic and primary motor cortex but rather by way of the association areas. A striking finding of the Welch and Stuteville experiments was the transience of this dramatic disturbance. This is another example of the tendency for small association area lesions to be compensated. Was the compensation by way of normal cortex adjacent to the damaged region, or were the animals using totally new pathways? This remains to be studied. There was an even more startling result in the Welch and Stuteville study. An animal showing this syndrome behaves as if he is blind in the field opposite the lesion. Following a removal of the occipital lobe on the side opposite the lesion there is a return of responsiveness to stimuli in the remaining half of the visual field although the animal continues to neglect tactile and auditory stimuli on that side. It would appear that some alternative pathways for motor response to visual stimulation of the hemisphere on the side of the frontal lesion are kept inhibited as long as the opposite visual region is operative. It would be most interesting to trace the anatomical substrate of this inhibition. If it is mediated via the corpus callosum it would appear likely that callosal section might cause the entire syndrome to vanish!
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> The first case of Hartmann (1907) might appear to be a human example of this syndrome of marked inattention to all modalities of stimulation coming from one side as the result of a frontal lesion. There are, however, certain difficulties in the clinical picture and pathology which prevent the full use of this case. Hartmann, however, must be credited with considerable prescience for he argues that since the right arm of his patient showed no movements in response to sensory stimuli, the frontal lobe (i.e. the region anterior to the motor cortex on the left side) was necessary for the conduction of sensory stimuli to the central gyri. This would agree of course with the thesis presented here that the premotor region is a way-station on the route from sensory cortex to primary motor cortex.

I have postulated a mechanism for the response of the motor to cortex sensory stimulation. If this thesis is correct we must assume that the apraxia of the left limbs in the patient with a right hemiplegia is not the result of the part of the lesion causing the hemiplegia. Rather it is the result of coincidental damage either to area 6 or to the callosal fibres to which it gives rise. Alternatively it is the result of damage to the afferents passing to area 6 from the visual, auditory, and somesthetic regions and travelling perforce beneath the motor cortex.

There are certain implications of this argument which are worth considering for the moment in more detail. The argument implies that a lesion in man of the regions anterior to the motor cortex on the left, but sparing area 4, should produce bilateral apraxia in our sense, i.e. failure to carry out movements to command and on imitation of the examiner but

with variable effects on actual object handling. As many readers will know it has in fact been asserted for a long time that lesions anterior to the motor cortex lead to apraxic disturbances (*see*, for example, Nielsen, 1946; Aring, 1944) and it has often been argued that these are "limb-kinetic" apraxias. Perhaps the best known of these disturbances from lesions anterior to the motor cortex are the facial apraxias (whose discussion I leave to the next section) and the pure agraphias which are said to arise from the posterior end of the second frontal convolution. I will not discuss the problem of "frontal apraxia" in detail since my own acquaintance with this large literature has not been intensive. My feeling has been that "limb-kinetic" apraxia has not been defined clearly enough to separate it from mild pyramidal disturbance. Liepmann (1905*a*) himself must have been somewhat unsure of limb-kinetic apraxia since he used as an example of this type of patient no case of his own but rather one published many years earlier by Westphal. The apraxias of the left side accompanying Broca's aphasias, which I have already mentioned, are due either to lesions of area 6 itself or of the callosal fibres arising from it. There is no clear-cut evidence to show that a lesion of the left-sided area 6 itself would produce a bilateral apraxia in virtue of its cutting off connexions both to the homolateral area 4 and to the contralateral motor region. The reason that such cases are lacking is probably the result of the fact that lesions of the left area 6 are very likely to encroach on the left area 4 and therefore to produce so much weakness in the right hand as to make assessment of apraxia difficult.

I believe, however, that this is an area where animal experimentation may be of greater use than it has been. Earlier experiments on ablation of area 6 have generally stressed "motor" functions (*see*, for example, the discussions in Bucy, 1944), rather than learning activities. The work of such investigators as Kennard (1939) Welch and Stuteville (1958), Weiskrantz and Mishkin (1958) should be extended. Of parallel interest would be further knowledge of way stations to the hippocampal region from area 6.

Facial Apraxia

This term is used as a shorthand for "apraxia of the cranial musculature," i.e. inability to carry out movements of this musculature either to command or on visual or tactile stimulation. It is the first form of apraxia to have been recognized (although not under that name) by Hughlings Jackson (1878) who commented on the inability of certain aphasics to protrude the tongue. It has continued to arouse interest sporadically. Many authors would, in fact, like to include Broca's aphasia as a *part* of such an apraxia.

Certainly this disturbance is the most common apraxic disturbance in aphasics. It has probably been recognized much more often than the more

extensive apraxia of the left side which I have mentioned earlier. I suspect that one reason for this is that when the patient fails to carry out facial commands but does perform limb commands, it is clear that comprehension is intact. When all commands on the intact left side are not performed, it is all too easy to attribute the failure to incomprehension.

The clinical picture is essentially similar to that of other apraxic disturbances but has some distinctive and intriguing characteristics. The patient usually does most poorly in carrying out facial movements to verbal command. He may simply fail to perform at all or may make an incorrect movement, e.g. he may open the mouth when asked to protrude the tongue, or blow instead of suck. He may make movements with one of his limbs to carry out the demanded task; thus, he may pretend to stub a match in an ash-tray or stamp on it with his feet when asked how he would blow out a match. He may remove imaginary crumbs from his lips with his fingers and not with his tongue even when asked repeatedly to use his tongue; he may even insist that he has always performed this action with his hand. Most interesting of all, the patient may echo the command or produce onomatopoietic responses. Thus, when asked to cough, he may say, "Cough"; when asked to blow out a match he may say, "Blow" or even, "Blow out a match," or "Puff." This type of response is all the more striking in that these verbalizations or vocalizations do not appear when the patient carries out the command correctly; similar disturbances are much rarer when patients fail to carry out *limb* movements to command.

The special peculiarity of facial movements is, of course, that they are generally bilateral. We must therefore ask how this bilaterality is achieved. A common view is that facial movements are integrated via the corpus callosum. This seems somewhat reasonable in view of the assertion that by strychnine neuronography (McCulloch and Garol, 1941) there can be demonstrated callosal connexions only between very limited parts of area 4, comprising the trunk and neck divisions and part of the face division. There is some reason, however, to question whether in man even these callosal connexions are of importance. Thus, the patient of Geschwind and Kaplan (1962) who performed poorly in carrying out commands with the left hand or left leg moved his face *bilaterally* in carrying out facial commands. This suggests that the pathway by which facial commands are carried out descends unilaterally to some brain-stem level where bilateral facial movement is integrated. It may be argued that a unilateral lesion of the face area of the right cortex produces weakness of the left lower face. This weakness, however, may be the effect of the withdrawal of tonic influence resulting from destruction of the right cortical face area and is probably not good evidence that movement of the left face to verbal command is mediated via impulses passing over the callosum. The right cortical face area would thus be regarded as providing a constant tonic influence but as having a phasic effect only under special conditions.

Alternatively one might speculate that normally these bilateral facial movements are integrated via the callosum but that when the callosum is gone there is ready replacement by means of directly descending pathways providing bilateral innervation. I think that this explanation is less likely than my earlier one, i.e. that each cortical face area separately is normally capable of causing bilateral activity at a brain-stem level.

We can now consider the pathway for facial movements to command or on visual stimulation. The pathway for facial movements to verbal command probably goes from the posterior speech area via the arcuate (superior longitudinal) fasciculus to the association cortex lying anterior to the face area. Similarly, the pathway to the face area from the visual region probably also passes in the inferior parietal region to some area anterior to the Rolandic face area.

The first implication of this is that lesions of supramarginal gyrus region should lead to facial apraxia. It has been our experience that they commonly do so. In particular facial apraxia to command and visual stimulation is likely, in our experience, to accompany the clinical syndrome of conduction aphasia, whose importance and whose anatomical basis have been emphasized lately by Konorski, Kozniewska and Stepien (1961) and to which I will return in a later section. These patients show no hemiplegia in the great majority of cases. Their speech may be fluent with many phonemic paraphasias. Even when speech is limited there are runs of fluency. While mild dysarthria is occasionally seen, the great effort and marked dysarthria of the Broca's aphasic are absent. Despite good to perfect comprehension there is marked difficulty in repetition. Their lesions probably lie low in the arcuate fasciculus (Konorski *et al.*, 1961). It is my opinion that the accompanying facial apraxia results from the same lesion. The fact that limb apraxia may be absent suggests that the fibres intended for facial "association cortex" run lower down than those going to the motor association cortex for the limbs.

The common occurrence of facial apraxia with lesions near Broca's area is readily understood. The lesion may destroy the association cortex anterior to the face area. Facial movements cannot be carried out to command or on visual stimulation because the lesion has cut off connexions to the left face area and cut off the origin of callosal fibres to the right face area. Alternatively a lesion in this neighbourhood may destroy left cortical face area or its projection fibres. In this case facial movements to command or visual stimulation must be carried out via the right face area receiving stimulation over the corpus callosum. If the lesion extends deep into the white matter it can involve the callosal fibres and facial apraxia will result. When a tactile stimulus, e.g. a drinking straw in the mouth, is used, the act, if overlearned, is often carried out correctly via the right sensory and motor cortex. In brief, we are regarding the facial apraxia of the patient with a Broca's aphasia as part of the left-sided apraxia of these

patients. Facial apraxia is more common than apraxia of the left limbs in this group of aphasics because a lesion producing aphasia will usually destroy the association areas and callosal fibres involved in face movements but will often spare those associative connexions lying more superiorly which are involved in limb movements.

Is the association cortex involved in facial movements to command or visual stimulation the same as Broca's area? I am not certain but some evidence suggests that they are different. For one thing, although some authors have chosen to regard Broca's aphasia as an apraxia of the speech organs, it has been clear in my experience that a very severe Broca's aphasia may be accompanied by little or no apraxia of cranial musculature; thus, some patients carry out movements with the cranial musculature to command and may imitate well despite marked restriction of speech. This would suggest some difference in the anatomical arrangements for speech and other cranial movements. Furthermore, I have already noted that patients with conduction aphasia commonly show facial apraxia and yet may show an aphasia which is quite different in character from a Broca's aphasia; in particular, dysarthria may be mild or absent. This too suggests that Broca's aphasia and facial apraxia may vary independently even though facial apraxia is most often seen with Broca's aphasia. Whether these differences are related to differences in the association cortex involved remains an open question. In any case, it is probably unjustifiable to call Broca's aphasia an apraxia if by this is suggested that it comprises part of a picture in which facial apraxia is necessarily present. The frequent tendency of the patient with facial apraxia to *repeat* the command which he fails to carry out also suggests a difference in anatomical arrangements for facial movements and speech. It cannot be ruled out that these differences are the result only of different degrees of overlearning of speech and non-speech movements.

Whole Body Movements

I will not discuss here the vexing question of what has sometimes been called "frontal apraxia" of gait and its possible anatomical substrate. I have not seen a clinical picture to which I would with assurance apply the term "apraxia of gait" although I have seen many examples of frontal gait disturbance. For a gait difficulty to qualify as an "apraxia of gait," it would have to meet the following criteria: the patient should perform whole body movements poorly to command or on imitation; the errors should not merely be failures to perform or clumsiness but should also include substitution of other well-performed movements for the desired ones. By contrast, under certain conditions, e.g. appropriate somesthetic stimulation, the patient should perform whole body movements perfectly well. This syndrome has not been seen to my knowledge—or at least not recognized. The patients generally described as manifesting frontal gait

disturbances show a fixed disturbance in performing integrated whole body movements, and I would accept Denny-Brown's (1958) view which regards this disturbance as involving impairment of more elementary motor mechanisms of some type. Similarly, I have not seen apraxic disturbances in the limbs in my cases of frontal gait disorder, i.e. my patients have made leg movements correctly to command and on imitation; I can conceive, of course, that in some instances apraxias of the legs might accompany a frontal gait disturbance but it is certainly not an obligatory association.

What I will stress in this section is rather the apparently anomalous *preservation* of whole body movements in patients with otherwise widespread apraxic disturbances and will suggest a mechanism that may underlie this finding. I have now seen several such patients. The only paper in which to my knowledge such sparing is noted is Liepmann's (1900). The patient had failed to carry out limb movements to command and was thought initially to be suffering from a profound comprehension difficulty. However, Liepmann wrote, "Against the view that comprehension of speech was totally lost was the circumstance that the patient promptly carried out tasks which he could execute with the whole body, such as standing up, walking to the window or walking to the door." This brief and incisive observation was thus the first clue that the patient was not so severely demented as he had been regarded for some time. Liepmann did not, however, return to the discussion of this point and did not attempt to analyse the mechanism of the preservation of whole body movements.

It seems clear that the callosum is not necessary for the integration of this type of bodily movement. The patient of Geschwind and Kaplan (1962) who often failed to carry out movements to command with the individual limbs of the left side still walked normally to command. It seems likely that the act of walking is controlled as an integrated act at the level of the brain-stem and that a command may descend unilaterally to this integrating system and set it going.

In another patient whom I have investigated, there was a left parietal glioblastoma. The patient showed no significant motor findings but manifested a marked bilateral apraxia of face and limbs. He performed very poorly to command, showed little or no improvement in attempting to copy movements made by the examiner and even handled objects very poorly; e.g. he could not with either hand hold a hammer properly to extract a nail although this particular manœuvre was demonstrated to him several times. In striking contrast was his preserved ability to carry out whole body movements. He could carry out a command such as, "Stand up, turn around twice and then sit down again," although he had been unable to perform such simple commands as, "Make a fist." Even when he failed to execute a movement, e.g. a bow, correctly to command, he immediately carried it out after the examiner had demonstrated the movement. There was no clumsiness at all and indeed it was generally accepted that

the patient's bow was considerably more graceful than that of the examiner.

One of the most dramatic manifestations of this discrepancy was seen when the patient was asked to assume the position of a boxer. He immediately assumed the boxing stance, leading correctly with the left fist. When asked to punch he looked perplexedly at his fist. Several different terms were then used—"punch," "jab," "uppercut," but none of these succeeded in eliciting a response. This situation set sharply in relief his ability to perform whole body movements in the face of marked difficulty with movements of individual limbs. I have seen another patient who showed a marked bilateral apraxia, worse on the left side, which involved the face and individual limbs. This case also demonstrated a striking relative preservation of whole body movements.

These cases have several points of interest. The grace and elegance of some of the whole body movements in such patients rules out that their failures in individual limb movements are the result of some general clumsiness or inco-ordination. The preservation of whole body movements cannot be interpreted as resulting from the greater simplicity of the movements. It is difficult to see how, "Stand up, turn around twice and then sit down again" is simpler than "Make a fist" but our second patient could do the former and not the latter. Nor is it obvious that "Show me the position of a boxer" is simpler than "Show me how a soldier salutes" (all of these patients were ex-Servicemen). Finally these observations also rule out the notion that apraxia of individual limbs need lead to inco-ordination of whole body movements.

It seems to me that such a marked difference in performance of whole body movements to command must depend on the utilization of different anatomic arrangements than those involved in movements of individual limbs. The bundle of Türck may well represent the efferent pathway for whole body commands. In the macaque this bundle arises mostly from the posterior part of the superior temporal gyrus (Brodal, in Jansen and Brodal, 1954), the region corresponding roughly to Wernicke's area in man. According to Brodal this tract is present in man and is extensive. All authors who have described it feel that it arises from the posterior temporal region although the posterior parts of all three temporal gyri have been implicated by different observers.[1]

[1] Some authors have, however, denied the existence of this tract. Thus, Whitlock and Nauta (1956) mention that after placing lesions in the temporal lobe of monkeys, they found no degeneration running to the pons. These authors themselves admit, however, that their material did not include lesions in the more caudal temporal regions as is apparent from inspection of their diagrams. This explanation may apply to other instances of failure to demonstrate a temporo-pontine tract. Bucy and Klüver (1955) also strongly deny the existence of this tract but also had no case of a posterior temporal lesion. By contrast, in support of Brodal's view, Krieg (1963) points out that in the macaque the only downward connexion of area 22 goes to the lateral cells of the pons. It is clear that this problem deserves careful reinvestigation.

In the macaque, according to Brodal, Türck's bundle descends via the retro-lenticular portion of the internal capsule and the lateral part of the cerebral peduncle to the dorsolateral pontine nuclei. These nuclei project in turn bilaterally to the vermis of the cerebellum. The *fronto*-pontine projections descend to the medial pontine nuclei which also project bilaterally to the cerebellar vermis. Türck's bundle would thus appear to project to a system which on good grounds is regarded as being involved in the motor control of gait and whole body movements. It is of interest that the pontine nuclei receiving afferents from the parietal and occipital lobes do not project to the vermis; their projections go only to the cerebellar hemisphere of the opposite side, in contrast to the bilateral projections of the nuclei receiving connexions from the frontal and temporal lobes. Flechsig (1901) surmised, and I would suspect correctly, that the bundle of Türck subserved "motions of the body and head in consequence of auditory impressions." Further evidence for Flechsig's supposition is Foerster's finding (cited by Crosby *et al.*, 1962) of adversive movements on stimulation of area 22 in man and the occurrence of adversive seizures with lesions in this location. It appears likely that these effects are the result of excitation of connexions from this region travelling by Türck's bundle. I would argue that it is highly likely that the carrying out of whole body postures to command depends on this bundle. The projection from Wernicke's area via this structure could still be intact even when the connexions from Wernicke's area to the motor association cortex were cut off. I would suspect that an appropriate lesion of Türck's bundle might lead to that condition of "apraxia of gait" in the sense in which I used the term at the start of this section. In the monkey bilateral lesions of this bundle might lead to failure to perform learned whole body movements to auditory stimuli.

Other Bilateral Movements

We have so far considered two types of bilateral movements, facial movements and whole body movements, and analysed the special circumstances involved in either their special impairment or their preservation in the face of widespread impairment of individual limb movements. We now move to another type of bilateral movement, the type in which individual limbs are involved on both sides. We distinguish two varieties of this type of movement, the bilaterally symmetrical movement (e.g. making circles in the air with both hands), and the asymmetrical learned bilateral movement, e.g. the movements of tying shoelaces.

It is often thought that callosal integration is necessary for the successful performance of such movements. Most of our evidence so far in fact suggests that this is not necessarily the case. Thus, the patient of Geschwind and Kaplan (1962) who often failed to perform actions with the left limbs to command did tie his shoelaces to command using *both* hands correctly.

We were perplexed by this at first but then realized that as long as each hemisphere had learned its task such bimanual movements could be carried out. In our case a command to tie the shoelaces would thus have been conveyed to the left hemisphere; the right hand would then move to begin the task. But the visual regions of the *right hemisphere* could then observe the right hand perform; the right hemisphere would thus receive visual stimulation and proceed to do its part of the task. Presumably a more careful analysis of the latencies with which each hand began to do its task would have helped prove this mechanism. The fact that our patient could respond to visual stimulation correctly with either hemisphere supports this interpretation. Our patient carried out other bimanual tasks equally well.

Ettlinger and Morton (1963) have shown preservation of a bimanual task in a monkey after callosal section, and I presume that a similar mechanism is involved.

If this mechanism seems a bit unlikely, it should be recalled that intensely complex co-ordinations of movements can be carried out by totally separate individuals. The members of a corps de ballet succeed in carrying on such well-integrated movements on the basis of visual stimulation and the use of well-learned sequences. Presumably if two separate individuals can carry out such actions, then the two halves of a cerebral hemisphere separated from each other should be able to do so also, as they apparently did in our patient. Dr. Ira Sherwin (personal communication) has suggested to me that in animals a similar mechanism may be operative, i.e. one hemisphere may learn what has previously been learned by the other, not via the callosum or any other direct nervous connexion but rather by visual observation of the behaviour of the other hemisphere. This notion deserves further experimental investigation.

Presumably such a mechanism for the carrying out of complex bimanual movements in the presence of callosal disconnexion can come into play only when each hemisphere has separately learned its part of the task; therefore, this type of movement should be best preserved in those individuals in whom the right hemisphere has some significant degree of independence in the learning of motor acts, as was the case for the patient of Geschwind and Kaplan. In the older writings, such as those of Liepmann, poor performance of bimanual acts is mentioned as a consequence of disconnecting lesions. My guess would be that in many patients bimanual acts would be poorly performed since the right hemisphere does not in general learn motor patterns as well as the left, as I have noted in my earlier discussion. There will, however, be cases like that of Geschwind and Kaplan where the right hemisphere has learned its task independently.

An alternative possibility might present itself—that bimanual tasks are learned via pathways descending from only one hemisphere and are therefore likely to be preserved, just as whole body movements are. One patient

we have observed presents evidence against this supposition. He showed bilateral apraxia, worse in the left hand, to verbal command, on visual stimulation and on object handling. When asked to perform a bimanual act, the right hand could eventually be got with difficulty to perform its role but the left hand could not. In short each hand was as apraxic as it had been in unimanual tasks. Yet, this patient showed a striking preservation of whole body movements. Clearly, the mechanisms of whole body movements and bimanual movements are different.

I would suspect that a similar mechanism explains the preservation of symmetrical but not highly overlearned bimanual movements in patients with callosal disconnexion. Thus when the patient is given the command to make circles in the air, the right hand can begin and the left can then join in. The patient of Geschwind and Kaplan showed this phenomenon. When his eyes were closed, however, the performance appeared to change somewhat. He then tended to move his arms at different rates, a performance unusual in a normal and then only attained by deliberate effort. We are not certain that we can attribute this lack of synchrony to the absence of callosal connexions since the mild pyramidal signs in the right arm might have affected its functioning in this task. Further observations will be needed in future cases to confirm this result.

The Problem of "Motor" Versus "Cognitive" Learning

I would like briefly to refer here to a problem that I have avoided in the above discussion, i.e. the classical argument in learning theory as to whether motor learning is the formation of "stimulus-response" connexions or whether it is the learning of a "cognitive map." Thus, has the animal who has learned a maze acquired a "picture" of it or has he learned a sequence of motor responses to a sequence of stimuli? The distinction is potentially an important one for the investigation of disconnexion phenomena. We might consider a hypothetical illustration. Suppose an animal was taught to respond by pressing a lever when a particular visual stimulus appeared and always used, by preference, his right forepaw in making this response during the period of training. If the visual cortex to motor cortex connexions were cut would the performance be lost? If the animal had acquired a cognitive "map" then the disconnexion between visual and motor association cortex might be unimportant. He might then be able to use pathways descending to the brain-stem and perform the task with a whole body movement, e.g. by sitting on the lever or by making a crude unilateral movement which was subserved by pathways descending directly to the brain-stem. Suppose by contrast the animal is taught to make a series of distinct complex motor movements in response to the visual stimulus and in fact is taught that these and only these will be rewarded. It is much more likely that this learning will depend on the motor cortex and that a lesion causing a disconnexion of the cortical visual and motor

areas will permanently impair the performance of this type of task. Another example might be the contrast between learning the pattern of a walking maze and learning to type. The pattern of a maze once learned, a human could get through it readily, walking, crawling, or driving. But knowing the pattern of a typewriter keyboard alone does not solve the problem of typing. Similarly, it will take great effort for an untrained person to play the piano even when he has learned the simple pattern of the arrangement of the notes.

It might appear at first that my analysis here has been exclusively a stimulus-response analysis and hence antagonistic to the idea of cognitive "maps." I have actually stressed the simpler stimulus-response situations and have not considered such maps. There is no reason, however, for assuming that these two types of "motor" learning are mutually exclusive. It would be of great interest to study the pattern of lesions in the nervous system of animals which would abolish or prevent the formation of such apparently purely cognitive learning.

Similarly, I have not discussed the problems implicit in the carrying out of such complex commands as, "Draw a star in the pink square." This request is quite different from such commands as, "Show me how you would use a hammer." In the first command we have three separate elements, the auditory command, and the motor response which, however, must be carried out under the control of vision, thus bringing a third system into play. The anatomical pathways for such commands which involve more than two elements represent an intriguing problem for further study.

VI. Other Aphasic Disturbances

I have so far not discussed that condition whose name in classical neurology specifically reflected the theoretical assumption that it was the result of disconnexion, i.e. conduction aphasia or Leitungsaphasie. The term, of course, meant aphasia due to failure of conduction, i.e. aphasia due to disconnexion. In this section there will be presented a discussion of this aphasic disturbance as well as of certain other syndromes which are probably the result of disconnexions.

Conduction Aphasia

Interest in this condition has been revived by the recent work of Konorski and his co-workers (1961). Konorski's presentation at the International Neurological Congress in Rome in 1961 alerted my colleagues and myself to this condition and we have now seen several very characteristic examples. In our experience the clinical picture is the following. The patient usually shows little or no hemiplegia. His spontaneous speech is often, but not always, copious. Dysarthria tends to be absent or mild, and whatever phrases are produced tend to be fluent.

While articulation may be normal, the speech is obviously and often severely aphasic, usually highly circumlocutory and often grossly paraphasic with a tendency particularly to literal paraphasias. There is marked difficulty in naming. Writing suffers along with spontaneous speech. As we have already noted earlier, facial apraxia to command is often marked and may also be present on imitation.

The notable feature of these cases is the marked discrepancy between comprehension and repetition. In the most striking of these cases comprehension is excellent as manifested by the ability to pick out correct and incorrect sentences, and also in many cases by the capacity to carry out verbal commands with the limbs. In striking contrast is the difficulty in repetition. In some instances even the simplest words fail to be repeated. The patient often says, "Say it again" which may give the impression of his not having heard or comprehended; yet even when there is positive evidence of comprehension, repetition does not improve. Thus, a patient may, on being given the word "president," say, "I know who that is—Kennedy," but still fail to repeat. The failures may be manifested by total inability to repeat, paraphasic repetitions or in many instances the production of an association to the correct word.

A remarkable feature of many of these cases is the frequently preserved ability of the patient to repeat polysyllabic numbers, e.g. "seventy-eight," while he fails to repeat even shorter words or repeats them paraphasically. The contrast is brought out sharply by such phrases as, "fifty-five per cent" where our patient said, "fifty-five progum" and "eleven plus eight" where the patient would say, "eleven, eight . . . nineteen" but failed to produce the word "plus" on repeated trials. "Three-quarters" was repeated as "three-four" for several weeks. Although the patient could say the series, "Penny, nickel, dime, quarter," he could not use the word quarter in repeating "three-quarters." Another patient said "fifty" for "one-half" and "seventy-five" for "three-quarters." Even when these patients fail to repeat numbers correctly, their errors are different from those with words. Thus, the patient tends to substitute other numbers but rarely to produce grossly paraphasic responses and even more rarely not to repeat at all. Thus, the patient may say "six, eight" for "sixty-eight" or "nineteen seven" for "ninety-seven." This advantage of numbers is not confined to the spoken modality since our first patient read printed words paraphasically but read numbers aloud correctly whether printed as numerals or as words. Thus, he read "train" for "travel," but correctly read "twenty-eight" just as easily as "28." It was very striking in this patient that whenever he was presented with numbers, he manifested an immediate and obvious relaxation of effort.

The mechanism of conduction aphasia was discussed by Wernicke (1874). He assumed a lesion in the connexions between what later came to be called Wernicke's area in the first temporal gyrus and Broca's area in

the third frontal gyrus. He assumed that these connexions ran in the insula and called this "aphasia of the insular region." Some thirty years later Wernicke (1908) discussed this problem again. He stated here that the autopsy findings had not confirmed his view that conduction aphasia was the result of lesions of the insula. He points out later on in this paper von Monakow's emphasis on the importance of the fasciculus arcuatus. This tract runs from the posterior superior temporal region, arches around the posterior end of the Sylvian fissure and then runs forward in the lower parietal lobe, eventually to reach the frontal lobe, and in particular Broca's area.

Kurt Goldstein (1927, 1948) discussed this condition in great detail. He called it not "conduction aphasia" but "central aphasia" and defined it as "a speech disturbance, which, in the presence of relatively intact comprehension of speech, is characterized by a disturbance in repetition, paraphasic manifestations in spontaneous speech, reading and writing, disturbance in spelling. . . ." He goes on to say that it appears with lesions between the sensory and motor speech zones.[1]

Goldstein mentions several theories of this condition. Liepmann and Pappenheim (cited by Goldstein, 1927) thought that this syndrome was only a milder variant of ordinary sensory aphasia. Goldstein points out, however, that most of Wernicke's area was intact in their case. Goldstein himself supported Wernicke's original view that it was a lesion of the insula which caused this syndrome and that it was actually the insular cortex and short association fibres which were involved. He did not, however, stress the disconnexion between Wernicke's and Broca's areas that Wernicke had postulated.

Pötzl and Stengel (1936) pointed out in a highly detailed study the interesting combination of conduction aphasia and pain asymboly (I have discussed the latter condition earlier in this paper). They point out the sparing of the insula in several cases of this condition such as the classic case of Liepmann and Pappenheim. In addition, in their own case the lesion did not directly destroy the arcuate fasciculus. They point out that the lesion common to their case and other earlier cases was the involvement of the left Heschl's gyrus and the planum temporale. Their own case had in addition a lesion of posterior insula and supramarginal gyrus; these as well as the other lesions were purely cortical. They theorized that their

[1] It should be noted that Goldstein (1927) explicitly equates his central aphasia to conduction aphasia. Thus he writes, "The symptom-picture which is usually designated as conduction aphasia. . . . *I called it central aphasia.* . . ." The descriptions by Brain (1961) and Russell and Espir (1961) appear to me not to make this distinction and to intermix elements of other aphasias. Neither of these authors stresses the component of disturbed repetition with relatively preserved comprehension which is the essence of this disorder. In his later writings Goldstein (1948) continues to use this earlier definition; he writes, "Understanding is usually preserved best, repetition and spontaneous speech are always severely damaged. . . ."

lesions probably destroyed the cells of origin of the pathway connecting the sensory and motor speech regions which they assumed to run both via the arcuate fasciculus and the insula. In order to test this theory a more detailed knowledge of the precise cells of origin of the connexions from upper temporal lobe to Broca's area and other opercular regions is needed. One interesting feature of this case was the cortical destruction of the supramarginal gyrus. Since there is considerable evidence that the arcuate fasciculus consists in great part of short fibres, it is not at all unlikely that this lesion might in effect destroy part of the pathway. They attributed, in keeping with the discussion presented earlier, the pain asymboly to the supramarginal gyrus lesion.

Konorski *et al.* (1961) have, as I have noted, revived interest in the problem of conduction aphasia and championed the idea that conduction aphasia results from a lesion of the arcuate fasciculus. I would think that in the light of the preceding discussion their view is probably correct.

It has been suggested that the development of language in man depends on his possession of an arcuate fasciculus, while a monkey does not have this pathway. This seems at first a reasonable conclusion from the findings of Bailey, Bonin, Garol and McCulloch (1943*a*) who show no arcuate fasciculus in their diagram of the long association fibres in the chimpanzee. There are, however, several reasons for rejecting this conclusion: (1) They were studying only *long* association fibres. Since it is possible, as we have noted earlier that much of the arcuate fasciculus consists of short fibres (Crosby *et al.*, 1962), these would not have been demonstrated in the study of Bailey *et al.* (2) There are, however, also long arcuate fibres revealed in later studies by some of Bailey's pupils (Sugar, French and Chusid, 1948) which demonstrated unquestioned evidence of fibres from the temporal operculum to the parietal and frontal opercula. (Their diagram does not show these fibres arching around the back end of the Sylvian fissure. Since they were using strychnine neuronography, there would have been no way for them to know the course of the fibres involved but only the origins and terminations.) These had been missed in the earlier study because Bailey *et al.* had not explored the depths of the sulci. The study of Sugar *et al.* certainly proves the origin of such fibres in the supratemporal plane. Whether these definitely arise from primary auditory cortex or from association cortex is not completely clear because of the great crowding of these structures in the small primate brain. (3) Bailey and Bonin (1951) themselves state, "Physiological neuronography presents evidence for both uncinate and arcuate bundles. . . . Whether the firing of the inferior frontal gyrus and the parietal operculum by strychninization of the first temporal convolution, that of the inferior frontal gyrus from the second and of the middle frontal gyrus from the inferior temporal gyrus (Petr, Holden, and Jirout, 1949) is due to conductions in the arcuate or uncinate fascicle cannot be decided at present; that the last, if not the latter two, observa-

tions should be ascribed to the arcuate fascicle seems more plausible. The firing of the middle temporal gyrus from the inferior parietal lobule as well as the firings of the inferior parietal lobule from the inferior frontal gyrus can be taken as further evidence for 'arcuate' fibres."

There is one further theory of the mechanism of conduction aphasia, one first mentioned by Liepmann and Pappenheim but defended most recently by Kleist (1962). This author argued that conduction aphasia occurs in patients in whom there has been actual destruction of the left temporal speech area itself. This lesion would in most instances lead to profound comprehension defect but does not do so in certain people because their right temporal region can take over this function. He assumes that these patients, however, still must rely on the left Broca's area for speech. The pathway from the right temporal speech area to the left Broca's area is, however, interrupted in these patients by the destruction of the left temporal lobe and therefore repetition is poor. Kleist presents 4 cases in support of this thesis. The first case, Spratt, is particularly striking since the destruction of the left superior temporal region was indeed so extensive as to be expected to give rise to profound incomprehension. This case would appear to be strong evidence for Kleist's contention. It is further of interest that this patient was ambidextrous, using his left hand for many complex functions. By contrast the lesion of his fourth case, Treusch, involved the left temporal speech area very little and appeared to involve deep white matter, particularly in the inferior parietal lobule. This case would appear to be much better evidence for the theory of involvement of arcuate fasciculus than for Kleist's own theory. In fact, Kleist's own diagram implies that even if the mechanism he suggests is correct in some cases, involvement of arcuate fasciculus should still produce the same syndrome. In summary, it appears that Kleist has good evidence for his mechanism in at least some cases of conduction aphasia. I would think that if there are two forms of the disorder, they might well be distinguishable clinically. We are attempting in our own cases of conduction aphasia to ascertain whether the right hemisphere really plays a role in any of them, as asserted by Kleist.

A brief note is appropriate on the already mentioned observation that number repetition is better than word repetition in many of these cases. It is conceivable that this is the result of sparing of a different anatomical pathway for numbers. It would seem unlikely, however, that numbers are normally spoken over a different pathway from words. I believe that a more likely explanation comes from a consideration of how any repetition is carried out in these patients. Thus, although the patient fails to repeat the correct word he is likely to give an association of this word. In fact, in some cases when the correct word itself is repeated, it is likely that this has been accomplished only by way of associations. One patient on being given his own last name first replied with the given names of his brothers

and himself and then finally gave the family name. Had the intervening names not been spoken aloud the fact that the eventual correct repetition was by way of associations and not "direct" could easily have been overlooked. This type of associative repetition probably does depend on finding a "detour" around the damaged normal direct pathway from Wernicke's to Broca's areas. It is natural that this should lead to many errors in repetition of *words* which in general arouse a large reservoir of associations. The associations to numbers are likely to be more limited, and in fact are not at all unlikely to be confined to the number itself. The errors in number repetition support this theory; thus, "six, eight" for "sixty-eight" and even more dramatically "three-four" for "three-quarters," "fifty" for "one-half" and "seventy-five" for "three-quarters." The marked difficulty in nonsense syllable repetition stressed by Konorski would result from the paucity of associations to such material. In brief, I think that these patients probably do not repeat either numbers or words via the normally used pathways but that in response to a heard number or word, the patient gives an associative response; the associative response is more likely to resemble the original if this is a number but not a word. One might say that there is "pseudo-repetition" of numbers. It is interesting that, as pointed out by Dr. Susan Ervin (personal communication), "parrot-like repetition" (i.e. precise repetition of what has been said) is a late development in children acquiring language. Contrary to ordinary views such precise repetition may well be an advanced activity dependent on specialized use of the pathways between the posterior and anterior speech regions.

The Case of Bonhoeffer

Bonhoeffer (1914) described a remarkable case which, although it presents certain difficulties in interpretation, I believe is worth presenting here. Although it is apparently unique, I suspect that other such cases have been overlooked by other observers.[1] Bonhoeffer's patient sustained a transient right hemiplegia, the arm subsequently improving faster than the leg. There was, however, permanent reduction of speech to one or two words; verbal comprehension was much better preserved but reading and writing were both very poor. The patient showed difficulties in carrying out verbal commands, in imitation and in object handling, all these disturbances being much more marked on the left than on the right. At post-mortem there was an infarct involving the posterior parts of the left superior and middle frontal gyri and the anterior four-fifths of the left side of the corpus callosum. There was an infarct of the anterior limb of the

[1] In fact, after having read Bonhoeffer's observation, I have come to suspect that a case I saw several years ago probably was an example of the same disturbance but that I completely missed its significance at the time. Chance favours, as is well known, the prepared mind; Bonhoeffer's was obviously very well prepared for this patient.

left internal capsule; finally a small lesion was present under the left parieto-occipital region. The cause of death was a fresh hæmorrhage in the lenticular nucleus on the right but the right hemisphere was otherwise intact. For obvious reasons Bonhoeffer attributed the severe apraxia of the left arm to the callosal lesion. To explain the aphasic disturbance of verbal expression, he pointed out that the infarct of the anterior part of the internal capsule had cut the descending pathways from the speech area. The transient hemiplegia was, of course, probably due to transitory neighbourhood effects of this lesion. This capsular lesion alone would, of course, produce no aphasia; nor would the frontal lesion which clearly spared Broca's area. Bonhoeffer pointed out that normally no aphasia is seen with a capsular lesion. In the presence of such a lesion an outflow path still exists from Broca's area running across the callosum to the corresponding cortical area on the right, and from there eventually reaching the right internal capsule. The callosal infarct had, however, cut off this alternative pathway and Broca's area was isolated.

My own inclination would be to agree on the whole with this interpretation of Bonhoeffer's. There remain to be explained the mild right-sided apraxia as well as the alexia and agraphia. Bonhoeffer himself was uncertain as to their cause, but I would regard them as having been caused by the lesion of left motor association cortex as well as by the lesion under the left angular gyrus. It is important to note that the latter was a small lesion which Bonhoeffer felt would not be very significant clinically. Bonhoeffer, however, then goes on to express his agreement with von Monakow's view that combinations of lesions may produce clinical pictures that none of the lesions could cause in isolation. (I have, of course, given several examples of such combinations.) The left angular gyrus lesion may have been much more disabling in this patient because of the co-existing extensive callosal lesion, which must have cut off most of the callosal connexions of the parietal association areas.

Echolalia

Kurt Goldstein (1917, 1948) has shown great interest in this symptom; Stengel (1947) has written an excellent paper on the functional aspects of echolalia. A characteristic clinical picture is that of *marked* preservation of repetition, indeed automatic repetition, in the face of gross difficulty in comprehension. I would stress that what is being spoken of here is a retention of repetition which is in sharp contrast to the disturbance in comprehension; other explanations probably are forthcoming for the common situations in which repetition is only moderately better than comprehension. To explain the sparing of repetition in such cases on the basis that it is "simpler" runs counter to the fact that repetition can be especially impaired, as in conduction aphasia.

Goldstein stressed that for this syndrome to be present it was necessary that Wernicke's area, Broca's area and the connexions between them be

intact. This intactness of the speech area and of its afferent auditory connexions guarantees repetition. Coupled with this intact speech area there must be a lesion which isolates the speech area from much of the rest of the cerebral hemispheres, i.e. a large parietal lesion. It is the *isolation* of the speech area which tends to produce this picture. There is no comprehension because language arouses no associations; there is gross disturbance of spontaneous speech since the speech area receives no information from elsewhere in the brain.

Segarra and Quadfasel (1961) have recorded the post-mortem findings of a most interesting patient who had been studied extensively by Quadfasel. This patient, a woman of 32 years of age, suffered severe illuminating gas poisoning, following which she survived for almost ten years. During this period she never showed any evidence of comprehension of language, nor ever spoke spontaneously a propositional phrase, but uttered only expletives. She could do nothing for herself. By contrast, she echoed what was spoken to her and even more strikingly she echoed songs. In fact, she was *even able to learn new songs not in existence before the onset of her illness*. Following her death her brain was cut in whole-brain sections and stained for cells and myelin. Preliminary survey of these sections has shown intactness of the speech area (Wernicke's and Broca's areas) and of the arcuate fasciculus and insula. In addition the hippocampal region is intact as well as connexions to it within the temporal lobe. The corresponding structures on the right side are symmetrically preserved. By contrast there is extensive infarction extending in mantle form around these intact regions. This patient had essentially an intact speech area which was able to carry on repetition. It would even manifest verbal learning because of the preservation of the connexions to the hippocampal region. Comprehension and propositional speech were, however, lacking for the reasons which I have already presented.

Cases of isolation of the speech area such as these are most valuable in elucidating the functions of this region. It is most remarkable that this patient could learn verbal material. This task is intensely difficult for patients with partial lesions *within* the speech area who may show syndromes far less devastating than that revealed by this patient. It also suggests that language is not "comprehended" by Wernicke's area but rather that this region serves to arouse associations elsewhere probably by way of the inferior parietal region. It illustrates also that speech is not created in Wernicke's area; rather it serves to transform what has come from the remainder of the brain into language.

VII. Possible Objections and Pitfalls

The Results of Akelaitis and His Co-workers

I would like to deal briefly here with the objections that might be raised against this theory of the importance of lesions of association cortex and

association fibres in producing disturbances of the higher functions of the nervous system. These objections arise chiefly from the results of the generally negative investigations made by Akelaitis and his co-workers (Akelaitis, 1941*a*, 1941*b*, 1941*c*, 1942*a*, 1942*b*, 1943, 1944, 1945; Akelaitis *et al.*, 1942) in cases with surgical section of the corpus callosum for the treatment of epilepsy. I have discussed this problem briefly elsewhere (Geschwind, 1962). I believe that the patients were well examined; inadequate examination is therefore probably not the explanation for the discrepancies between Akelaitis's results and many of those which I have cited.

The majority of Akelaitis's patients had had cerebral lesions dating from early childhood. Such early lesions might well explain the absence of cerebral dominance for language. Thus, many of the functions which are normally unilateral could probably be carried on by both hemispheres. A similar explanation probably applies to the lack of such syndromes in cases of agenesis of the corpus callosum. The early lesion is more likely to lead to functional re-organization than lesions in adult life (as examples of this one may cite the work of Kennard, 1942 and of Scharlock, Tucker, and Strominger, 1963), i.e. it is more likely to lead to use of other pathways.

A second factor is the presence of epilepsy in nearly every case in Akelaitis's series. It is certainly highly possible that seizures may lead to the "learning" of new pathways (I have commented earlier in the paper on Morrell's work on the parallels between epilepsy and other learning). Similarly, Penfield and Boldrey (1939) wrote, "An habitual seizure, by virtue of its frequent repetition, may eventually establish a complicated neurone pattern . . . (The spread of a seizure) is not diffuse but . . . along a definite neurone system which may be preformed or acquired. By 'preformed' is meant a system of neuronal connexions ordinarily recognized as physiologically functional in normal brains. By 'acquired' is meant a pattern of neuronal connexions established by the conditioning influence of previous experience. . . . In complicated seizures the advance of discharge along this neurone pattern may be so slow and episodic as to suggest that isolated ganglionic collections are fired in an advancing series. . . ." The general failure of callosal section as a therapeutic agent in Akelaitis's cases is compatible with the seizures having long since "learned" some complex or unusual pathways or having "learned" such new pathways after the transection. Erickson (1940) pointed out that section of the corpus callosum in monkeys prevented electrographic spread of the seizure to the opposite hemisphere and altered the pattern of the seizure. Whether electrographic spread to the opposite hemisphere might have continued to take place despite transection of the corpus callosum if the experiments had been done on chronically epileptic monkeys rather than in acute experiments is not known. Such an experiment would,

however, be of great interest in determining whether seizures "open" less commonly used pathways to the other hemisphere. The second part of Erickson's experiment seems to confirm strongly that with a lesion confined within the bounds of a hemisphere seizures rapidly "find" new paths to circumvent the obstruction. Thus, in one monkey a seizure initiated in the right cortical arm area spread in the intact brain to the right leg area, left leg area, and left arm area. After a wide section at right angles to the Rolandic cortex superior to the right arm area, the seizure travelled from the right arm area to the left arm area then to the left leg area and finally to the right leg area. Section of the callosum abolished this new mode of spread.[1]

As an example of the joint effects of seizures and early lesions, we may compare the cases reported by Akelaitis in whom there had been section of the splenium, and those reported by other authors. There were 6 such cases in Akelaitis's series, all of them severely epileptic, 5 with lesions from early childhood. None developed alexia in the left visual field following section of the splenium. By contrast the case of Trescher and Ford (1937) and the 2 cases of Maspes (1948) all had colloid cysts of the third ventricle and none had had seizures. All three developed alexia in the left visual field as a consequence of having had the splenium sectioned.

The notion that repeated seizures favour the opening of less used pathways leads to several interesting implications. One should be able in experimental animals to overcome the effects of disconnecting lesions by repeated seizures. Thus, an animal with the callosum sectioned who has had repeated seizures for a long period before callosal section might show interhemispheric transfer after surgery while a control without seizures might not. It goes without saying that the long-term effects of section of the callosum should be less disturbing if carried out in infant animals than in adults.

It is remotely possible that the effects of repeated seizures in favouring the opening of new pathways, if verified, might be useful therapeutically. Repeated induced seizures might favour recovery from disturbances following lesions of association cortex or fibres. Obviously, such a

[1] The term "arm area" does not necessarily mean area 4; the electrical disturbance was probably, in my opinion, going via area 6. This discussion is not meant to suggest that seizures spread only between cortical regions. Certainly some of Erickson's observations in the above paper confirmed that bilateral involvement of the body can occur with the callosum sectioned—but this is different in type from the kind of bilateral spread seen when the callosum is present. Obviously bilateral spread with the callosum sectioned depends on involvement of subcortical structures. Gastaut and Fischer-Williams (1959) have discussed such subcortical spread extensively in their review. One of the major reasons for the failure of surgery in Akelaitis's patients may have been such spread to subcortical motor systems rather than spread to the opposite hemisphere via "new" subcortical pathways. We do not have enough information to decide which is the explanation.

therapeutic investigation could be justified only after experimentation had confirmed the effectiveness of such procedures in animals.[1]

There are two other but less likely explanations for Akelaitis's results. One is that alternative pathways to the opposite hemisphere via subcortical routes are readily available in most people and that these syndromes therefore appear only exceptionally. The other is that the syndromes had cleared by the time the patients were tested. Although the patient of Geschwind and Kaplan (1962) did show definite improvement in his callosal disconnexion symptomatology over several months, it was still evident after this period. Furthermore, acquired lesions of many other association pathways may show no improvement with time. Restitution after white matter lesions might conceivably be less damaging permanently than lesions of the corresponding cells of origin; as long as the cells are intact, there is a possibility that collateral pathways might be brought into play. We know, however, almost nothing of the pathways of secondary importance.

This leads us to the greatest advantage but at the same time the greatest danger of thinking in terms of disconnexions. Theories of this type are rich in readily testable theoretical implications. They can readily degenerate into naïvely anatomical systems. This is, however, not inherent in the structure of this approach and there is no conflict between this kind of reasoning and sophisticated psychological analysis. In fact, this approach frequently demands a reinvestigation of standard psychological categories and opens new ways of looking at psychological phenomena. Perhaps the greatest danger is that of "working backwards" and of inventing pathways to correspond to every difference in behaviour. I have tried to show here that while some differences in behaviour are probably based on anatomically different pathways (e.g. the discussion on isolated limb movements versus whole body movements) others are dependent on non-anatomical factors (e.g. the discussion on the relative preservation of number repetition in conduction aphasia). I can only agree with the hope expressed by Adolf Meyer (1905) for "convincing observations of patients . . . with such anatomical examination as will put an end to the regrettable tendency of so many clinicians to consider the white matter of the hemispheres the cornucopia of all the desirable conduction paths."

VIII. Philosophical Implications

It is not my intention to embark on an extensive philosophical discussion in this section. It seems reasonable, however, to sketch rapidly the implications of many of the results discussed here for the philosophical foundations of study of the higher functions of the nervous system.

[1] To forestall any questions in advance I am in no way suggesting that the effects of electric shock therapy are related to such a mechanism!

The Whole Man

For the past forty years there have been schools of thought which have stressed the importance of thinking of the patient as a whole, of seeing his responses as those of an integrated unitary structure, even in the face of damage. The ramifications of this thinking in neurology, psychiatry, psychology and other fields must be well known to most readers. It should be clear from much of our discussion that this principle, while it may be useful in some cases as a stimulus, may be actively misleading when it is regarded as a philosophical law. When Edith Kaplan and I were studying our patient, we constantly found that many confusions about the patient in our own minds as well as those of others resulted from failure to do the exact opposite of what the rule to look at the patient as a whole demanded, i.e. from our failure to regard the patient as made of connected parts rather than as an indissoluble whole. We were constantly dealing with questions such as "If he can speak normally and he knows what he's holding in his left hand why can't he tell you?" We had to point out that we couldn't say that "the patient knew what was in his left hand" and that "the patient could speak normally," since that part of the patient which could speak normally was not the same part of the patient which "knew" (non-verbally) what was in the left hand. This is at first blush an odd way to speak —it is hard not to say "the patient" and yet it is clear that this terminology is misleading.

We have little difficulty with the concept of disconnexion at lower levels. If the spinal cord is transected we are usually capable of treating it as separate from the higher centres. We may say of such a person, "The patient urinated" since we know that in this case this means "The patient urinated involuntarily." Although "the patient urinated" is at first glance ambiguous, in practice we understand and do not find it necessary to use clumsy locutions such as "The patient's spinal cord urinated." We get into difficulties, however, with disconnexions at higher levels since we do not expect highly organized activities dependent on learning to be carried on in disconnected parts of the brain. We must become accustomed to thinking in this way in order to understand some of the more complex disturbances consequent on lesions of the brain. I am not advancing "the atomistic approach" as a basic philosophical postulate to replace "the holistic approach," but am rather suggesting that failure to consider the applicability of *either* type of analysis will in one situation or another lead to errors. It should be pointed out that the usefulness of sometimes considering animals or humans not as a unit but as a union of loosely joined wholes need not apply only to disease states although probably it will find its greatest use in that situation. Probably even in the normal person parts of the brain are so weakly connected as to make their interaction difficult. I have suggested that some connexions are normally present in adult man which are absent or of less extent in lower animals; these connexions may

take years to develop in some children. Perhaps an adult man is more unitary than a chimpanzee, but perhaps total unity is never obtainable because of the necessary separation of some structures.

The Unity of Consciousness

A corollary to the above discussion is that it forces us to be somewhat more precise than we have been about "the unity of consciousness." It would no doubt be startling to suggest that the patient of Geschwind and Kaplan had separate consciousness in each hemisphere; it would on the other hand be a little difficult to understand just what would be meant by saying that his consciousness was unitary. If the ability to give a verbal account is a prerequisite of consciousness then only the left hemisphere was conscious; if the ability to respond in a highly organized manner and to use the results of past experience constitutes consciousness then he had multiple consciousness. Perhaps there are better criteria of consciousness than these; this case and similar ones only re-emphasize the necessity of re-evaluating the idea of the unity of consciousness if it is to be at all useful.

I believe that Kurt Goldstein was perhaps the first to stress the non-unity of the personality in patients with callosal section and its possible psychiatric effects. Thus he wrote (Goldstein, 1927), "The separation of so large a part of the brain and the resulting impossibility of evaluating stimuli perceived with the right hemisphere . . . surely cannot be without effect on the total personality. . . . I have pointed out the presence in my patient of a feeling of strangeness in relation to movements of the left hand, which she described with such curious expressions (she would say that someone was moving her hand and that she wasn't doing it herself) that she was regarded at first as a paranoiac. It appears to me not to be excluded that on this basis and under certain conditions there may develop paranoid states, perhaps also the experience of doubled personality and above all the experience of being influenced from without. . . ."

The Value of Introspection

Still another corollary to the observation that parts of the brain may be disconnected is the conclusion that introspection may be an extremely ineffective way of obtaining information about many of the patient's experiences. I have already made this point in the presentation of some of the syndromes of disconnexion, e.g. in respect of the colour-naming disturbance of pure alexia without agraphia. If a part of the brain is fully disconnected from the speech area it will not be possible for the speech area to give an account of what goes on in that part of the brain. The patient with a colour-naming disturbance can give only a poor account of his colour-experience or indeed none at all since his speech area has little or no access to information about the colour-experiences of the visual cortex. Other examples were cited illustrating the same principle. I have also

discussed in some detail the tendency for the patient to show confabulatory response to demands for introspective observations. It is certainly well known that even in normals introspection may be misleading or incorrect.

Let me point out carefully that what is presented here is not the gross behaviouristic assertion that introspective information is in all situations uesless nor that one should never listen to what the patient is saying. The conclusion being presented is the less extensive one that there are certain situations, particularly in the presence of lesions of the brain, in which the patient for simple anatomical reasons is incapable of recounting verbally the experiences of parts of his nervous system which are functioning at a complex discriminative level.

Language and Thought

An old problem is the one of the extent to which language controls one's "perception" of the world. Whorf (1956) suggested an extreme form of the view that language influences what is perceived. The view at the opposite extreme is the naïve "natural" one that language is an infinitely flexible tool for describing without prejudice the impact of the world on one's nervous system. The behaviour of our patient with colour-naming defect is of interest here. Despite his inability to name colours correctly, he had no difficulty in sorting, and did so by colour. This, of course, does not necessarily reflect the behaviour of a "naïve" nervous system since the visual region may have been trained by earlier verbal experience. It is clear at least that words need not be available for the sorting process to take place.[1] On the other hand, as I noted in my discussion of secondary sorting errors, the use of the incorrect word may lead to errors in sorting under the conditions of certain types of instruction. This type of "secondary" error might be more marked in tests where the subject is required to remember colours after an interval since it seems reasonable that such memory is frequently mediated verbally (Brown and Lenneberg, 1954). The study of patients with such specialized disturbances may aid in further exploring the relationship of language to cognition.

SUMMARY

A complete summary of all the material presented would be much too extensive and, indeed, much too repetitious. I will therefore try to outline here the major points presented in Part I (Geschwind, 1965) as well as Part II. I have attempted to show that many disturbances of the higher functions of the nervous system, such as the aphasias, apraxias, and

[1] Some informal experiments on children suggest that perceptual differentiations precede naming. My 4-year-old son misnamed colours but could sort correctly and could trace out correct numbers on the Ishihara test; although I could name the colours of dots, I did much poorly than he did on these tests. His difficulty was one of colour-naming, mine was partial colour-blindness.

agnosias may be most fruitfully studied as disturbances produced by anatomical disconnexion of primary receptive and motor areas from one another. For a detailed discussion the reader is referred to the appropriate sections of the paper.

In the lower mammals connexions between regions of the cortex may arise directly from the primary receptive or motor areas. As one moves up the phylogenetic scale, these connexions come to be made between newly developed regions of cortex interspersed between the older zones. These regions are called "association cortex." As Flechsig pointed out for the human brain, all intercortical long connexions (whether in or between hemispheres) are made by way of these association areas and not between the primary motor or receptive areas. It follows from this that lesions of association cortex, if extensive enough, act to disconnect primary receptive or motor areas from other regions of the cortex in the same or in the opposite hemisphere.

The connexions of the visual association regions were discussed in some detail, and it was pointed out that the major outflow of these regions is to the lateral and basal neocortex of the temporal lobe which in turn connects to limbic structures. Lesions of the lateral and basal temporal lobe therefore tend to disconnect the visual region from the limbic system. This leads to a failure of visual stimulation to activate limbic responses, such as fight, flight, and sexual approach. It also leads to difficulties in visual learning. These can be thought of as resulting from the failure of the animal to form visual-limbic associations (such as learning that a visual stimulus equals the food reward given for correct choice) because of the lack of appropriate connexions. They can also be regarded as disturbances in visual recent memory resulting from a disconnexion between the visual region and the hippocampal region. The discussion was then applied to the tactile and auditory systems. Learning difficulties in primates involving these systems also were thought to result from disconnexions from the limbic system. "Agnosia" in the sense of failure to respond to stimuli within a single modality appropriately in the face of intact perception in that modality is regarded as being a part of the syndrome of disconnexion of primary sensory modalities from the limbic system. Since callosal fibres arise from association cortex, failures of interhemispheric transfer may result from lesions of association cortex. The problem of whether disconnexions of single modalities from the limbic system in man occur was briefly discussed.

While connexions between primary receptive regions and limbic structures are powerful in subhuman forms, intermodal connexions between vision, audition, and somesthesis are probably weak in these animals, a view for which evidence is available both on the basis of experimental behavioural investigations (e.g. studies on intermodal transfer of learning and on higher-order conditioning) and on the basis of anatomical evidence.

In man the situation changes with the development of the association areas of the human inferior parietal lobule, situated at the junction of the older association areas attached to the visual, somesthetic, and auditory regions. It is speculated that this new "association area of association areas" now frees man from the dominant pattern of sensory-limbic associations and permits cross-modal associations involving non-limbic modalities. It is particularly the visual-auditory and tactile-auditory associations which constitute the basis of the development of speech in most humans. In man the speech area (which constitutes the auditory association cortex, particularly that part of it on the convexity of the temporal lobe, also Broca's area and the connexions between these regions) becomes a structure of major importance in the analysis of all the higher functions.

Pure word-blindness without agraphia was then discussed as an excellent, classical example of disconnexion from the speech area; this syndrome results from a combination of lesions, the usual one being destruction of the left visual cortex and of the splenium of the corpus callosum. The association with this syndrome of colour-naming difficulties and inability to read music is noted, along with the relatively strong preservation of the reading of numbers and the naming of objects. Reasons are advanced for these discrepancies. The problem of childhood dyslexia and its associated disturbances and its possible relation to the acquired dyslexia of adults was briefly presented.

Other disorders with similar pathogenesis (isolation of a particular sensory modality from the speech area), i.e. pure word-deafness and tactile aphasia were then briefly discussed.

The problem of the "agnosias" was then presented. Evidence was presented against the idea that there exist disturbances of "recognition" regarded as a unitary faculty. It was argued that most of the "agnosias" are in fact modality-specific naming defects resulting from isolation of the primary sensory cortex from the speech area and associated with marked confabulatory response. A critique was presented of the classical "aphasic-agnosic" distinction. There was presented some further discussion on the determinants of confabulatory response. The problem of right parietal syndromes was presented in the light of the preceding discussion of the "agnosias."

"Apraxic" disturbances were analysed in detail and were regarded as resulting from disconnexions of the posterior speech area from association areas which lie anterior to the primary motor cortex, and from disconnexions of visual association areas from these "motor association" areas. The problem of left-sided predominance was discussed. In particular apraxic disturbances resulting from callosal lesions, from lesions of "motor association" cortex and from damage deep to the supramarginal gyrus were discussed. The apraxia of the left side ("sympathetic dyspraxia") of

aphasic right hemiplegics was discussed as well as facial apraxia. The sparing of certain types of movement in the apraxias was discussed, particularly whole body movements and was related to the probable preservation of Türck's bundle (whose connexions were presented in some detail) running from the posterior temporal region to the pontine nuclei and then via synapses to the cerebellar vermis.

Finally syndromes resulting from disconnexions within the speech area (conduction aphasia) and the pattern resulting from the isolation of the speech area were presented.

Some classical objections to the disconnexion approach were presented, in particular the results of Akelaitis and reasons for his negative results were discussed. This section closed by pointing out that this type of theory suggests many experiments and anatomical investigations. The dangers of *ad hoc* postulation of connexions were mentioned.

In a short section attention was called to some philosophical implications of these findings, particularly for the notions of "regarding the patient as a whole man," the unity of consciousness, the uses of introspection and the relations between language and one's view of the world.

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