The Organization of Language and the Brain

Language disorders after brain damage help in elucidating the neural basis of verbal behavior.

Norman Geschwind

Many problems relating to the functions of the nervous system can effectively be studied by investigation in animals, which permits controlled and repeatable experiments on large groups of subjects. When we come, however, to consider the relationship of the brain to language, we must recognize that our knowledge is based entirely on findings in man. Some authors would even argue that language is exclusively a human attribute, so that no experiments on animals could ever be relevant. Although I believe that forerunners of language do exist in lower forms (*1*), the direct contributions to this area of experimentation on the brains of animals still lie in the future.

Brain Lesions in Man

Information in this area has come from several sources. Cases of brain tumor are of limited value, since tumors distort the brain and produce effects at a distance. Cases of penetrating brain wounds (*2*) have been of considerable use but are not the best source of anatomical data, since postmortem information is usually lacking. Analysis of the sites at which the skull was penetrated is of use statistically, but, because of variations in the paths taken by missiles, cannot provide precise data concerning the location of lesions producing language disorders. Stimulation during surgery (*3*) has been another most important source of information but, because of limitation of time at operation and the accessibility of only certain structures, has not covered the full range of phenomena observed clinically.

The elegant studies of Milner and her co-workers on patients undergoing excision of cortical regions for epilepsy represent the largest corpus of truly experimental studies of the higher brain functions in man (*4*). They are limited, however, with respect to the range of phenomena observed. Furthermore, since most of these patients were undergoing removal of areas of brain which had been the site of epileptic discharges since childhood, there is reason to believe that the effects seen after surgery may not represent the full range of phenomena seen after damage to the adult brain. The Wada test (*5*), in which sodium amytal is injected into one carotid artery, has been a major source of knowledge concerning the lateralization of language functions in the brain.

Although important information has been obtained by the above methods, it is still true that the bulk of our knowledge concerning the relationship of the brain to language has been derived from the study of adults in whom delimited areas of brain have been damaged as the result of occlusion of blood vessels, who have been studied carefully over long periods, and whose brains have been subjected to careful postmortem examination. Although fully suitable cases of this type are not common, the experience of nearly 100 years of study has built up a large body of reliable knowledge.

Aphasic Disorders

The generic term *aphasia* is used to describe the disorders of language resulting from damage to the brain. Early in the history of the study of aphasia the distinction between language and speech was stressed. In disorders of speech the verbal output was impaired because of weakness or incoordination of the muscles of articulation. The criterion of a disorder of language was that the verbal output be *linguistically* incorrect. The muscles of articulation might be used normally in nonlinguistic activities. Similarly, in aphasic disorders of comprehension the patient might lose the ability to comprehend spoken or written language and yet show normal hearing or vision when tested nonverbally. Furthermore, these disorders could occur without impairment of other intellectual abilities. The aphasias were thus the first demonstrations of the fact that selective damage to the brain could affect one class of learned behavior while sparing other classes, and thus gave origin to the field of study of brain-behavior relationships. The discovery of these phenomena was one of the greatest achievements of the last half of the 19th century.

Some cases of aphasia had been described before the mid-1800's, but it was Paul Broca who in 1861 began the study of the relationship of aphasia to the brain, with two major contributions (*6*). He was the first to prove that aphasia was linked to specific lesions, and to show that these lesions were predominantly in the left half of the brain. The man who was, however, most responsible for initiating the modern study of this field was Carl Wernicke (Fig. 1), who in 1874, at the age of 26, published his classic work, *The Symptom Complex of Aphasia*, which carried the appropriate subtitle, "A Psychological Study on an Anatomical Basis" (*7*). Wernicke established clearly the fact that there were linguistic differences between the aphasias produced by damage in the left temporal lobe, in what is now called Wernicke's area, and those produced by lesions in the frontal lobe in Broca's area (Fig. 2) (*8*).

Linguistic Changes in Aphasia

The aphasic of the Broca's type characteristically produces little speech, which is emitted slowly, with great effort, and with poor articulation. It is not, however, only at the phonemic level that the speech of these patients is

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abnormal, since the patient clearly fails to produce correct English sentences. Characteristically the small grammatical words and endings are omitted. This failure persists despite urging by the examiner, and even when the patient attempts to repeat the correct sentence as produced by the examiner. These patients may show a surprising capacity to find single words. Thus, asked about the weather, the patient might say, "Overcast." Urged to produce a sentence he may say, "Weather . . . overcast." These patients invariably show a comparable disorder in their written output, but they may comprehend spoken and written language normally. In striking contrast to these performances, the patient may retain his musical capacities. It is a common but most dramatic finding to observe a patient who produces single substantive words with great effort and poor articulation and yet sings a melody correctly and even elegantly. Because Broca's area lies so close to the motor cortex (Fig. 2), this latter region is often damaged simultaneously, so that these patients frequently suffer from paralysis of the right side of the body.

The Wernicke's aphasic contrasts sharply with the Broca's type. The patient usually has no paralysis of the opposite side, a fact which reflects the difference in the anatomical localization of his lesion. The speech output can be rapid and effortless, and in many cases the rate of production of words exceeds the normal. The output has the rhythm and melody of normal speech, but it is remarkably empty and conveys little or no information. The patient uses many filler words, and the speech is filled with circumlocutions. There may be many errors in word usage, which are called paraphasias. These may take the form of the well-articulated replacement of single sounds (so-called literal or phonemic paraphasias), such as "spoot" for "spoon," or the replacement of one word for another (verbal paraphasias), such as "fork" for "spoon." A typical production might be, "I was over in the other one, and then after they had been in the department, I was in this one." The grammatical skeleton appears to be preserved, but there is a remarkable lack of words with specific denotation.

The Wernicke's aphasic may, in writing, produce well-formed letters, but the output exhibits the same linguistic defects which are observed in the patient's speech. He shows a profound failure to understand both spoken and written language, although he suffers from no elementary impairment of hearing or sight.

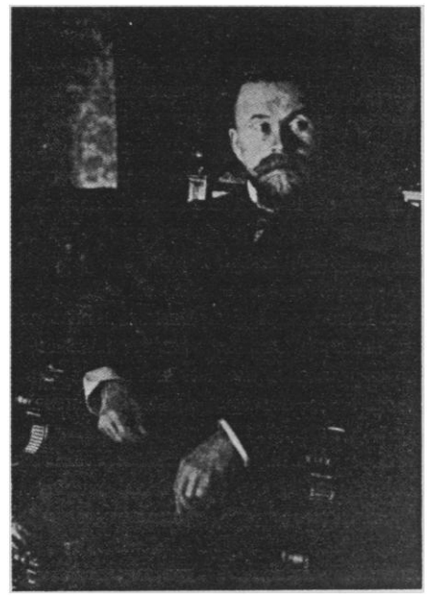

Fig. 1. Carl Wernicke (1848–1904), who, at the age of 26, published the monograph *Der aphasische Symptomencomplex*, which was to be the major influence on the anatomical study of aphasia in the period preceding World War I. During his tenure as professor at Breslau, his assistants and students included many of the later leaders of German neurology, such as Otfrid Foerster, Hugo Liepmann, Karl Bonhoeffer, and Kurt Goldstein.

The localization of these forms of aphasia has been confirmed repeatedly. It is important to stress this point, since there is a common misconception that the classical localizations were rejected because powerful arguments were raised against their validity. The two authors whose names are most frequently quoted as critics are Kurt Goldstein and Henry Head. As I have pointed out in greater detail elsewhere (*9*), Goldstein, who had been a student under Wernicke at the University of Breslau, despite the holistic views which he expressed in his philosophical discussions, actually explicitly stated his support of the classical localizations throughout his career. Head did indeed violently attack these views early in the first volume of his famous work on aphasia (*10*). His argument was, however, vitiated by the fact that, later in the same volume, the localizations which he himself supported turned out to be essentially identical to the ones he had previously dismissed as invalid.

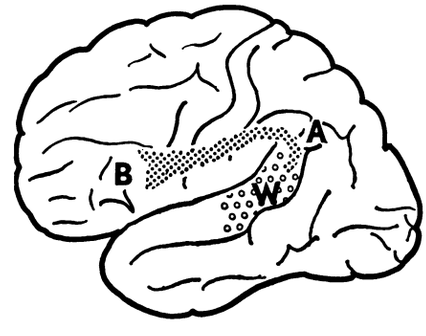

Fig. 2. Lateral surface of the left hemisphere of the human brain. *B*, Broca's area, which lies anterior to the lower end of the motor cortex; *W* (open circles), Wernicke's area; *A* (closed circles), arcuate fasciculus, which connects Wernicke's to Broca's area. (See text.)

Wernicke's Theory

Wernicke's contribution lay not only in establishing the syndrome patterns and their localizations but also in providing a theoretical analysis of the mechanisms of aphasia (Fig. 2). He pointed out that Broca's area was located just in front of the cortical region in which lay the motor representation for the face, tongue, lips, palate, and vocal cords—that is, the organs of speech. It seemed reasonable to assume that Broca's area contained the rules by which heard language could be coded into articulatory form. This formulation still appears reasonable. There is no need to assume that this coding need be a simple one. By contrast, Wernicke's area lies next to the cortical representation of hearing, and it was reasonable to assume that this area was somehow involved in the recognition of the patterns of spoken language. There is also no need to assume that this coding is a simple one.

Wernicke then added the natural assumption that these two areas must be connected. The general pattern was now clear. Destruction of Wernicke's area would lead to failure to comprehend spoken language. Wernicke pointed out that, for most people, written language was learned by reference to the spoken form and that therefore a lesion of this region would abolish comprehension of printed and written language. The act of speaking would consist in arousing in some way the auditory form of words, which would then be relayed forward to Broca's area to be transduced into the complex programming of the speech organs, and therefore, with damage to Wernicke's area, language output would also be disordered.

The model could readily be compli-

cated further. Wernicke himself and those who followed him filled in further details. The comprehension of written language would require connections from the visual to the speech regions, and destruction of these connections should be able to cause isolated difficulties in reading comprehension. Since the language abilities were localized in the left hemisphere, language performances by the right hemisphere would depend on information transmission over the corpus callosum.

Clearly the validation of a theory is not a function of its surface plausibility but is dependent on other factors. It is important to remember that Wernicke's theory has been the only one in the history of aphasia which could in a real sense be put to experimental test. It was possible, on the basis of the theory, to predict that certain lesions should produce syndromes not previously described. Furthermore, it was possible, on being confronted with previously undescribed syndromes, to predict the site of the anatomical lesion. The most dramatic examples of this appear in the writings of Hugo Liepmann (*11*) on the syndromes of the corpus callosum. On the basis of his clinical examination he predicted the presence of callosal lesions, which were later confirmed at postmortem examination.

Several remarkable disorders of language have been described which fit readily into the Wernicke theory. In pure word deafness, the patient, with intact hearing as measured by ordinary nonverbal tests, fails to comprehend spoken language although he has essentially normal ability to express himself verbally and in writing and to comprehend written language. In this syndrome the area of damage generally lies deep in the left temporal lobe, sparing Wernicke's area but destroying both the direct auditory pathway to the left hemisphere and the callosal connections from the opposite auditory region. Although elementary hearing is intact because the right auditory region is spared, there is no means for auditory stimulation to reach Wernicke's area, and therefore the patient does not understand spoken language, although his ability to express himself in spoken and written language and his comprehension of the written language are essentially intact (*12*).

In conduction aphasia, there is fluent paraphasic speech, and writing, while comprehension of spoken and written language remains intact. Despite the good comprehension of spoken language there is a gross defect in repetition. The lesion for this disorder typically lies in the lower parietal lobe (Fig. 2), and is so placed as to disconnect Wernicke's area from Broca's area. Because Broca's area is preserved, speech is fluent, but abnormal. The preservation of Wernicke's area insures normal comprehension, but the gross defect in repetition is the result of disruption of the connection between this region and Broca's area. The disorder in repetition exhibits some remarkable linguistic features which are not yet explained. The disorder is greatest for the small grammatical words such as *the*, *if*, and *is*; thus, a patient who may successfully repeat "big dog" or even "presidential succession" may fail totally on "He is here." The most difficult phrase for these patients to repeat is "No ifs, ands, or buts." In many of these patients the ability to repeat numbers may be preserved best of all, so that, given a phrase such as "seventy-five percent," the patient may repeat the "seventy-five" rapidly and effortlessly but may fail on "percent" (*12*).

Pure Alexia without Agraphia

Many examples of pure alexia without agraphia were described in the 1880's, but the first postmortem study of this syndrome was described in 1892, by Dejerine (*13*). His patient suddenly developed a right visual field defect and lost the ability to read. He could, however, copy the words that he could not understand. He was able, moreover, to write spontaneously, although he could not read later the sentences he had written. All other aspects of his use and comprehension of language were normal. At postmortem Dejerine found that the left visual cortex had been destroyed. In addition, the posterior portion of the corpus callosum was destroyed, the part of this structure which connects the visual regions of the two hemispheres (Fig. 3). Dejerine advanced a simple explanation. Because of the destruction of the left visual cortex, written language could reach only the right hemisphere. In order to be dealt with as language it had to be transmitted to the speech regions in the left hemisphere, but the portion of the corpus callosum necessary for this was destroyed. Thus, written language, although seen clearly, was without meaning. This was the first demonstration of the effects of a lesion of the corpus callosum in preventing transfer of information between the hemispheres.

Dejerine's thesis has received striking confirmation. In 1925 Foix and Hillemand (*14*) showed that destruction of the left visual cortex in the absence of a callosal lesion does not produce this syndrome. In 1937 Trescher and Ford (*15*) described the first case in which a surgical lesion of the corpus callosum was shown to have a definite effect. Their patient had sustained section of the posterior end of the corpus callosum for removal of a tumor from the third ventricle. The patient could not read in the left visual field, but could read normally on the right side. This result is implied by the Dejerine theory and was confirmed by Maspes in 1948 (*16*) and more recently by Gazzaniga, Bogen, and Sperry (*17*). Many authors have confirmed Dejerine's anatomical findings. Michael Fusillo and I studied a patient with alexia without agraphia who demonstrated another intriguing disorder (*18*, *19*). For approximately 3 months after his stroke he suffered from a disorder of verbal memory, which then cleared, leaving him with the reading difficulty, which remained unchanged until his death several months later. At postmortem, in addition to the anatomical findings of destruction of the left visual cortex and of the posterior end of the corpus callosum, the brain showed destruction of the left hippocampal region. It is now generally accepted that bilateral destruction of the hippocampal region leads to a permanent memory disorder. The transient memory disorder in our patient appeared to be the result of the destruction of the left hippocampal region—that is, the one located in the same hemisphere as the speech areas. Presumably it is the left hippocampal region which is necessary for the memory functions of speech cortex. After a period, the brain manages to compensate, presumably by making use of the opposite hippocampal region. Since publication of our paper (*18*), I have seen several other cases of this syndrome in which memory disorder was present at the onset. It is well known that the posterior cerebral artery supplies not only the visual cortex and the posterior end of the corpus callosum but also the hippocampal region. In a certain number of cases of occlusion of the left posterior cerebral artery, all of these structures are damaged. In other cases, however, the hippocampal region is

spared. Meyer and Yates (*20*) and Milner (*4*) have demonstrated that, after removal of the left anterior temporal region for epilepsy, a verbal memory disorder is observed, which is, however, generally much milder than that found in the case Fusillo and I reported, and which is not present after right anterior temporal ablation. The mildness of the disorder after left temporal ablation is probably the result of the fact that these patients had suffered from left temporal epilepsy for years and had therefore already begun to use the right hippocampal region to a considerable degree.

Isolation of the Speech Area

Another syndrome, called "isolation of the speech area," is explained readily by the Wernicke theory. This syndrome was described first by Kurt Goldstein (*21*) and has been described more recently by Geschwind, Quadfasel, and Segarra (*22*). We studied our patient for nearly 9 years after an episode of carbon monoxide poisoning. During this period she showed no evidence of language comprehension in the ordinary sense, and never uttered a sentence of propositional speech. She was totally helpless and required complete nursing care. In striking contrast to this state were her language performances in certain special areas. She would repeat perfectly, with normal articulation, sentences said to her by the examiner. She would, however, go beyond mere repetition, since she would complete phrases spoken by the examiner. For example, if he said, "Roses are red," she would say, "violets are blue, sugar is sweet, and so are you." Even more surprising, it was found that she was still capable of verbal learning. Songs which did not exist before her illness were played to her several times. Eventually, when the record player was started she would begin to sing. If the record player was then turned off she would continue singing the words and music correctly to the end, despite the lack of a model. Postmortem examination by Segarra showed a remarkable lesion, which was essentially symmetrical. The classical speech area, including Wernicke's area, Broca's area, and the connections between them, was intact, as were the auditory inflow pathways and the motor outflow pathways for the speech organs. In the regions surrounding the speech area either the cortex or the underlying white matter was destroyed. The speech area was indeed isolated. The patient's failure to comprehend presumably resulted from the fact that the language inputs could arouse no associations elsewhere in the brain, and since information from other portions of the brain could not reach the speech areas, there was no propositional speech. On the other hand the intactness of the speech region and its internal connections insured correct repetition. The preservation of verbal learning is particularly interesting. In addition to the speech area, the hippocampal region, which is involved in learning, was also preserved, and this probably accounts for her remarkable ability to carry on the memorizing of verbal material.

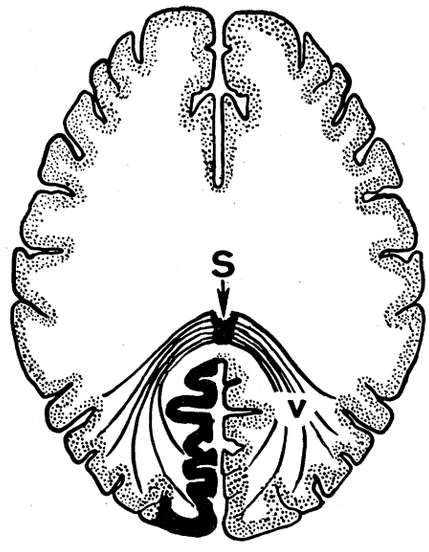

Fig. 3. Horizontal section of the human brain, illustrating the mechanism of pure alexia without agraphia; *V*, visual region. The visual cortex on the left is destroyed (heavy black line). As a result, the patient can perceive written material only in the intact right visual region. For this material to be appreciated as language it must be relayed to the speech areas on the left side through the splenium, which is the posterior portion of the corpus callosum. As a result of damage to the splenium (*S*), this transfer cannot take place, and therefore the patient cannot comprehend the written words whose form he perceives clearly.

Callosal Syndromes

Although pure alexia without agraphia (*13*) was the first syndrome in which damage to the corpus callosum was shown to play a role by interrupting transfer of information between the hemispheres, it was a group of Wernicke's students, including Hugo Liepmann, Kurt Goldstein, and Karl Bonhoeffer, who elucidated the full syndrome of callosal disconnection in cases in which eventually there was careful postmortem confirmation of the predicted sites of the lesions (*11, 12*). While the callosal syndromes continued to be recognized by German authors (*23*), their existence was either forgotten or indeed totally denied in the English-language literature. In November 1961, Edith Kaplan and I presented a patient to the Boston Society of Psychiatry and Neurology who was, we believed, suffering from a callosal disconnection syndrome—a diagnosis which was later confirmed at postmortem examination by Segarra. [Since that time several cases of confirmed callosal disconnection have been described (*24*).] I will mention here briefly only a few of the aspects of our patient's condition which fit into the Wernicke theory. When writing with the right hand the patient produced linguistically correct words and sentences and carried out calculations correctly. When writing with the left hand he produced incorrect words (for example, "run" for "go") and performed calculations incorrectly. The theory outlined above implies that, for writing to be carried out correctly with the left hand, the information must be transmitted from the speech areas across the corpus callosum, whose interruption in our patient explained his failures. Similarly, the patient could correctly name objects (concealed from vision) which he palpated with the right hand. On the other hand he would misname objects palpated with the left hand, although it could be shown by nonverbal means that his right hemisphere recognized the object. Thus, if a pencil was placed in his left hand the patient could draw the object previously held in that hand. Again, the Wernicke theory implies that, for an individual to correctly name an object held in the left hand, the information must be transmitted from the sensory regions in the right hemisphere to the speech regions via the corpus callosum, which had been destroyed in this patient. On the other hand, the patient could read in the left as well as the right visual field. This led us to conclude that the destruction of the corpus callosum had spared the posterior end, a prediction also confirmed at postmortem.

Cerebral Dominance

Let me turn to another bit of knowledge which fits very well into the scheme presented above. One of the most remarkable features of man is cerebral dominance—that is, the fact that in the adult the capacities for speech are overwhelmingly controlled by the left hemisphere. Out of 100 adult aphasics, at least 96 percent have damage to the left side of the brain (*25*). We do not know of any example in any other mammal of a class of learning which is predominantly controlled by one half of the brain (*26*). What underlies human speech dominance? It is widely stated in the literature that the human brain is symmetrical, and this had led either to the assumption that speech dominance must reflect some subtle physiological difference between the hemispheres, or indeed even to the assumption that speech dominance is somehow acquired as the result of postnatal experience. My colleague Walter Levitsky and I (*27*) decided to reinvestigate this problem, particularly since we found that some earlier authors had claimed that there were in fact anatomical differences between the hemispheres. We demonstrated that such differences exist and are indeed readily visible to the naked eye. The area that lies behind the primary auditory cortex in the upper surface of the temporal lobe is larger on the left side in 65 percent of brains, and larger on the right in only 11 percent. This region on the left side is, on the average, nearly a centimeter longer than its fellow on the opposite side—that is, larger by one-third than the corresponding area on the right. More recently Wada (*28*) has confirmed our results. He has, in addition, studied this region in the brains of infants and has found that these differences are present at birth. This region which is larger in the left hemisphere is, in fact, a portion of Wernicke's area, whose major importance for speech was first shown nearly 100 years ago. It is reasonable to assume that there are other anatomical asymmetries in the hemispheres of the human brain, reflecting other aspects of dominance.

The study of the organization of the brain for language has been based of necessity on investigations in man. The bulk of our information in this area has come from careful studies of patients suffering from isolated damage as a result of vascular disease, whose brains have, after death, been subjected to careful anatomical examination. Disorders of language resulting from brain damage, almost always on the left side, are called aphasias. Carl Wernicke, nearly 100 years ago, described the linguistic differences between aphasias resulting from damage in different anatomical locations and outlined a theory of the organization of language in the brain. Not only have Wernicke's localizations stood up under repeated examination but his theory has been the only one which has permitted the prediction of new phenomena, or has been able to account for new observations. Several remarkable disorders, such as isolated disturbances of reading and the symptomatology of the corpus callosum, are examples of the explanatory power of this theory.

The phenomenon of cerebral dominance—that is, the predominant importance of one side of the brain for a class of learned behavior—occurs, as far as we know, in no mammal other than man. The dominance of the left side of the brain for speech is the most striking example of this phenomenon. Contrary to generally accepted views, there is a striking anatomical asymmetry between the temporal speech region on the left side and the corresponding region of the right hemisphere.

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