WERNICKE'S REGION—WHERE IS IT?

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In this subject, the first question both logically and chronologically was and is: Can a lesion (focal damage) of the cerebrum cause a loss of language without causing a loss of intelligence? That is the original question, still debated hotly by many people. Much of the heat is attributable to the way in which the question is phrased. Suppose we phrase it relatively, as follows: Can a lesion of the cerebrum produce a deficit in language that is *far in excess* of the concomitant deficit in intelligence? Asked in this way, almost everyone would answer yes. There are worthy persons who are still arguing that anyone who has a loss of language from a cerebral lesion must have some accompanying loss of intelligence. Similarly, there are equally worthy persons recurrently showing us that intelligence can be preserved in spite of severe aphasia. Both parties are undoubtedly correct. But the force of either argument is largely dissipated when the question is rephrased in the relative way. Of course, how much intelligence is lost (or retained) depends upon how one goes about measuring intelligence; but with almost any measures, except those strictly linguistic, the answer will be yes. Indeed, if the answer were *not* yes, there would not be such a thing as aphasia, since a "selective loss of language from a cerebral lesion" is what the word "aphasia" means in contemporary usage.

Once we all understand that there *is* such a thing as aphasia, we come to a second question. It is: Can one indicate those places in the cerebrum where an aphasiogenic lesion is likely to occur? Again the answer is yes—an aphasiogenic lesion occurs in right-handed people in the right hemisphere 1% of the time, perhaps 2% at most. If you have a person who is definitely right-handed, and he has a cerebral lesion that produces a loss of language far out of proportion to the loss of intelligence, the odds are about 50 to 1 that the lesion is in the left hemisphere. Indeed, one can localize better than that. It is rather unlikely that the lesion will be in the left occipital pole. It is even less likely that it will be in the left temporal pole and it is very unlikely, although not impossible, that it will be in the left frontal pole. So, in this negative way, we can narrow down to some extent where an aphasiogenic lesion will occur.

There is a third question, raised by Wernicke [1] about 1874: Is there more than one kind of aphasia? In other words, when a person suffers linguistic loss with relative preservation of general intelligence, can the linguistic loss be of more than one kind? When we look at the patients, it is obvious that they are different. The question is, should those differences be emphasized, and how? There are almost as many classifications of aphasia as there are aphasiologists. Rephrasing this third question does not seem to help with this argument, which has continued unabated for over a century. In the words of Lhermitte and Gautier: [2]

> For the present there is no satisfactory classification of aphasias. (page 97)

The characterization of aphasias is not a central concern here, but we would suggest that in order to deal with the question in a precise manner, it will be necessary to use the mathematics of multidimensional vector spaces; that is, we could characterize a patient's condition with a vector, each of whose coordinates represent

the degree of some specific deficit such as anomia, nonfluency, phonemic disintegration, syntactic confusion, and the like. Or we might incline to the system of Hécaen and Angelergues,[2a] who semiquantified the language deficit in their aphasic patients in terms of seven features: disturbance in articulation, fluency, verbal comprehension, naming, repetition, reading, and writing. Until there is a more widespread acceptance of this (vector) approach, it will be difficult to decide upon a system of independent dimensions. What we want eventually is a function describing the changes (including rotation as well as a decrease in vector norm) over time, as the patient recovers. This approach introduces a quantification in the place of arbitrary boundaries for categories of aphasia; and it can precisely represent the qualitative variability of aphasic syndromes with time.

There is another familiar question: If there *are* different kinds of aphasia, are the respective lesions in different places? It's at this point that such agreement as may exist among aphasiologists seems to vanish altogether. Of course, there could not be much agreement on *where* the loci would be for lesions that produce different kinds of aphasia if people cannot agree on what *kinds* of aphasia there are to begin with.†

But let us consider a particular, single dimension: verbal comprehension. It was emphasized by Wernicke, and is considered important by almost all authors. There are patients who have trouble comprehending spoken language. They vary as to whether they can read out loud, or whether they can repeat what they hear, or whether they can do this or that. But whatever else they can or cannot do, they cannot comprehend spoken language very well. Suppose we ask: Is there a region in the cerebrum where a lesion will produce a serious deficit in comprehension of language which is all out of proportion to the loss of nonlinguistic abilities? It was Wernicke who first raised this question, and he gave an answer. He gave, in fact, *several* answers, and we will refer to three of them.

Before we get to the whereabouts of Wernicke's region, we should take note of the fact that this question does not arise with respect to Broca's area. Broca's area is defined anatomically. It is the foot, that is, the posterior third of the inferior frontal gyrus (See FIGURE 1). The question about Broca's area is not "where is it?"—there is no question about *where* it is. The question about Broca's area is "what good is it?" At this time we consider only Wernicke's region, of which the question is *not* "what good is it?" because it's defined in terms of what it's good for—it's the area where a lesion will cause language comprehension deficit. The question with Wernicke's region is "where is it?"

There is, first of all, Figure 3 in Wernicke's classic monograph of 1874.[1] What does one see in this picture? For one thing, he shows the right hemisphere, rather than the left; but this is more in the nature of a printer's error than anything else, and we all now understand that we are talking about the left hemisphere. The feature that requires discussion is his little circle labeled "a_1," in the middle of the superior temporal gyrus (also called "the first temporal gyrus" or "T_1"). He obviously doesn't mean that the brain substance covered only by that little circle does all the comprehension. Rather, the little circle represents the "center," somewhat in the sense of a center of gravity. He does not delimit the entire region, but only localizes the center of it. One naturally asks, is the little circle right in the middle of the entire region or closer to one end? After all, the center of gravity of a thing does not have to be right in the geographic middle; it might be at one end. Wernicke gave us some idea of what he thought. He wrote in 1874 (on page 45):[1]

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†There is also the fact that people's brains differ. (See the article by Whitaker and Selnes in this volume.)

We will direct our attention to that gyrus of the cerebral convexity which circles around the Sylvian fissure in an arc directed superiorly and posteriorly. Anterior to the central fissure it runs longitudinally (as the first frontal gyrus, in Leuret's nomenclature), while its posterior limb is in the longitudinally coursing first temporal gyrus. That this entire structure is to be regarded as a single gyrus is evident from comparison with the brains of animals, e.g., those of dogs. Comparative anatomy has shown it to be a general law for the formation of the convolutions of the brain that they describe an arc around the fossa Sylvii, the peak of which is directed toward the occipital pole and both limbs of which run more or less parallel to the fossa Sylvii in the frontal and temporal portions of the brain. This law holds for humans as well."

Then on page 47:

The entire region of the first convolution, which circles around the fossa Sylvii serves in conjunction with the insular cortex as a speech center.
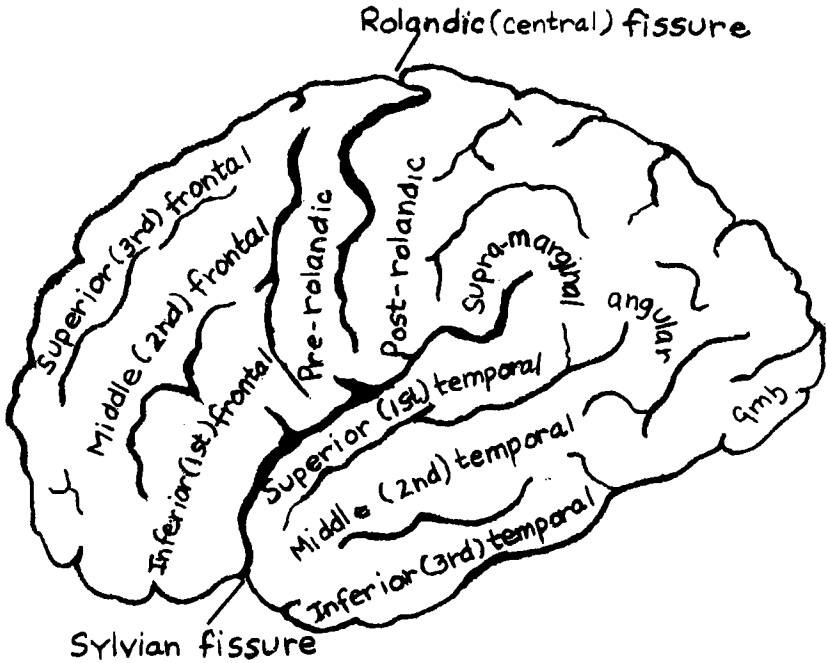

FIGURE 1. Lateral surface (convexity) of the left cerebral hemisphere, showing the more prominent fissures (sulci) and convolutions (gyri). The frontal gyri are numbered from below upward to conform with the concept of a "primeval first gyrus." But the overwhelming majority of neurologists number these gyri in reverse direction, so that the inferior frontal gyrus is called "the third frontal gyrus" or "F_3".

We all know that the word "center" is troublesome, and it is just as easy to talk about the speech "region." Also, Wernicke used the word "speech" instead of "language"; but he meant "language" the way we mean it now. So what Wernicke is telling us is that the primeval first convolution, together with the insula, acts as a language region or language zone. (Following the advice of E. A. Weinstein, we

generally avoid the two-dimensional word "area," since it incorrectly implies that the depth of the lesion is irrelevant).

As you may know, Wernicke did not believe that the language zone was all the same; some of it was more important for comprehension and some of it for expression and what not. So, where did he think was the region for comprehension? There is another picture, in his big text on neurology of 1881,[3] seven years later. This picture (Figure XX on page 205) shows the area for language comprehension to be almost synonymous with the first temporal gyrus. It seems to lap over a little bit, though, just barely into the second gyrus. Why does it lap over just a little bit? Probably, because he wasn't sure; he was rather fudging a little bit over the sulcus, so he couldn't be accused of using the sulcus as the boundary. That raises the question: does Wernicke's region (the region within which a lesion disturbs comprehension of language) include part of the second temporal gyrus?

One answer is in Charcot's opinion of 1888, as reproduced in an article by Pierre Marie,[4] his devoted student. Charcot definitely included the second temporal gyrus. Furthermore, Charcot included the angular gyrus. By this time Dejerine had pointed out that language comprehension deficits can occur from lesions in the angular gyrus. Just because Wernicke did not know that a lesion back there would cause a comprehension deficit does not mean that the angular gyrus is not part of Wernicke's region.

But then an instructive thing happened. Pierre Marie began to see patients for himself. And in the course of twenty years he came to realize that Charcot was not entirely correct. In 1906, Pierre Marie[5] said,

> The only cerebral territory whose lesion produces aphasia is the so-called territory of Wernicke (gyrus supramarginalis, gyrus angularis, and feet of the first two temporals).

That's what he says; it is explicit. Among other things, it completely excludes the middle parts of the first and second temporal gyri as well as any frontal cortex. But then came the war (World War I), and Marie softened his attitude in the face of new evidence.

Before we consider Marie's opinion after his observations during World War I, we should consider Dejerine's view as illustrated in his textbook of 1914.[6] This is of particular interest, since Marie and Dejerine were such implacable opponents. (They were not even able to agree, in the great debate of 1908,[7] which questions to debate). There are several notable things in Dejerine's picture of Wernicke's region. First, there is a vague suggestion of what we eventually must have; that is, an awareness that there is no sharp boundary on one side of which a lesion causes aphasia and on the other side of which it does not. What we will eventually need is a picture that indicates the *probability* of a language deficit as being higher or lower. Dejerine indicated more heavily in his picture the places in which a lesion is most certain to cause aphasia; and then he has a dotted line that takes in considerably more territory: most of the parasylvian area including the second temporal gyrus. But there is another feature of note. He did not include, within the dotted line, the inferior Rolandic region, although it is part of what Wernicke called the primeval first gyrus. Why was this excluded by Dejerine? It probably reflects a usage that has reappeared from time to time ever since; that is, to call by the term "language area," not an area within which a lesion disturbs language, but within which a lesion disturbs language without disturbing something else even more, such as making the person hemiplegic. It is nearly certain that a lesion in the inferior Rolandic area will cause aphasia. Well, then, why isn't this included in the "language area"? It is because there is also paralysis. So some people have tended to use the term "language area" to mean "language and only language." This is a serious misconception, because it happens

that almost all cerebrocortical tissue serves more than one function. So here is a rather peculiar convention, appearing clearly for the first time; it has reappeared from time to time ever since.

Now we consider Marie and Foix (1917).[8] Before World War 1, people who had aphasia were usually older folks with strokes. But during the War, neurologists saw many young men with shrapnel wounds, free of any uncertainty as to whether the patient's deficit was the result of an aphasiogenic lesion superimposed upon generalized atherosclerosis or some previous, silent stroke. What Marie and Foix ended up with is illustrated in their paper of 1917 (as reprinted in Lhermitte and Gautier[2]). This picture shows the zone within which one can expect a lesion to cause difficulty in comprehension of language. (We note parenthetically that, according to Marie, if you have trouble talking it is not necessarily a sign of aphasia—it is the failure to *comprehend* language which Marie considered definitive). The region outlined by Marie and Foix definitely takes in most of the second temporal gyrus as well as the angular gyrus and the supramarginal gyrus. It also includes the inferior Rolandic cortex, which Dejerine wanted to exclude for the terminologic reasons previously mentioned. There are two details about the region indicated by Marie and Foix which are peculiar; first, they left out Broca's area—there is a funny little dent anteriorly because Marie had very strong feelings about Broca's area. He believed that it "plays no special role in aphasia," an extreme view which is agreed to by hardly anyone else before or since. There is a similar funny little dent at the top of the language area. If you have had much experience with aphasia patients you know that this dent is misleading, since lesions there do occasionally cause aphasia.

Next we consider Lewandowsky's textbook of 1923[9]; an important authority in those days. In his picture, Wernicke's area comes a short way into the second temporal gyrus; but for some reason, he leaves out part of the angular gyrus. In fact, he leaves out most of the parietal operculum. But what is most important about his picture is that it shows us something that no one else bothers to show us. He has opened up the Sylvian fossa and shows the language zone extending medially into the Sylvian fossa to include the insula and then back out underneath the frontal operculum to include Broca's zone. This resembles what Wernicke said, but which no one else has bothered to indicate in a picture before or after Lewandowsky. If there is a *confluent language zone* (as Wernicke said and as Lewandowsky illustrated forty years later), where does the posterior part end and the anterior part begin? Perhaps the division is somewhere in the middle, half-way across the insula? It is apparent that when the actual anatomy is exposed, and the Sylvian fissure no longer looks as if it were a bottomless chasm, a sharp division between anterior and posterior language areas can be drawn only arbitrarily. This is certainly worth remembering—that there is no big gap between the posterior and anterior language areas, as suggested by so many other pictures.

Next we come to Henry Head (1926).[10] He studied British soldiers instead of French soldiers; and he concluded that there are four kinds of aphasia. These four types of aphasia are shown in a picture (IV-5), which Penfield and Roberts[11] based on Head's verbal description. This picture looks somewhat like Wernicke's primeval first gyrus, except that it stretches out posteriorly into the angular gyrus; it includes the parietal and frontal opercula. In fact, we are beginning to realize that whenever people try to present a comprehensive view of the matter, they commonly show a picture of the entire parasylvian region.

Well, then, one might ask, why is there so much confusion about the language zone? One answer is that many people are unduly influenced by certain textbook errors. Fulton's textbook of neurophysiology[12] was the *only* American textbook of neurophysiology for many years. In that book it says (on page 395) that Wernicke's

area is the angular gyrus of the left hemisphere. Where did he get this strange idea? We don't know. But he does say on the next page that there is an alternative view he attributes to Pierre Marie; namely, Wernicke's area is the angular gyrus together with the base (i.e. the posterior third, or foot) of the first and second temporal convolutions. *That* is what Fulton ascribed to Marie: but is not what Marie[5] said (in 1906), because it does not include the supramarginal gyrus. How is it that someone with the reputation of being as scholarly as Fulton leaves out the supramarginal gyrus (besides not mentioning the Marie and Foix picture of 1917)?[8] We may never know how this came about; but we do know that vast numbers of people have read this, and believed it, because it was in Fulton's textbook. One thing seems clear: Fulton did not rely on Schilder[13] (1923), who said that Wernicke's area was the posterior one-third of the first two temporal gyri.

Let us look at another famous authority, MacDonald Critchley.[14] This is what (on page 16) he says:

> In the dominant hemisphere the inferior parietal lobule (supramarginal and angular gyri) together with the posterior third of T_1, make up what is often termed by continental neuroanatomists, "Wernicke's area."

In other words, Critchley leaves out entirely the second temporal gyrus. He gives a definition of Wernicke's area which is no more accurate than what Schilder said, or what Fulton said, or what Fulton said that Marie said, which is *not* what Marie said.

Let us consider now our first teacher, J. M. Nielsen.[15] According to Nielsen, Wernicke's region has shrunk, and consists of a tiny raisinlike area in the middle third of T_1. When this picture was shown to some medical students (after they had seen the previous pictures), one of them said, "It looks like the side pocket of a pool table." But there is a really important part of the Nielsen scheme: what he called the "language formulation area." This is a region in which a lesion causes comprehension troubles, and it includes the posterior part of the *inferior* or third temporal gyrus. Why did Nielsen believe that this was an area in which a lesion causes language deficit? First of all, he believed it because Mills asserted this fact on the basis of several cases. Furthermore, Nielsen also saw people who had aphasia from lesions there. And the reason we emphasize this at the present time is because we also have had a couple of aphasic patients whose lesions were in the posterior part of the inferior temporal gyrus.

A somewhat different picture appears in the book of Percival Bailey.[16] This picture obviously came from Marie and Foix,[8] because Broca's area is left out. Furthermore, Bailey's picture has the other funny little dent in the superior aspect. What is notable about Bailey's picture (which is done with dots instead of just drawn lines) is that his picture suggests that the edges are not sharp; they seem to fade out, instead of having the sudden discontinuity of a sharp line beyond which nothing happens. This is a crucial concept.

Next we can look at a picture from World War II instead of World War I (and from Russian soldiers, rather than British or French soldiers). The data, from Luria,[17] are quite informative. The picture indicates that 95% of people with a lesion within the first temporal gyrus are going to have what Luria calls phonemic acoustic perception deficits. In the posterior part of the inferior temporal gyrus, the probability is 37%. The probability is essentially zero in the frontal pole and in the occipital pole. In the parietal operculum, it is 53%. It is important to note this because there are some people such as Lewandowsky[9] and Nielsen,[15] whose pictures totally omit the parietal operculum. It is also noteworthy that Luria shows the probability to be 19% for phonemic acoustic deficit with a lesion of Broca's area.

With the picture of Luria, we have the beginnings of an appropriate map. It is the *probability* of a deficit which should concern us. What is the probability that a lesion, here or there, will cause a language deficit? And the answer is that it is very high in or near the first temporal gyrus, and fades out with different gradients (varying among individuals) toward the poles. And by the time it gets to any pole (occipital, temporal, or frontal) the probability is essentially zero.

Next we can consider some World War II soldiers from England. Sir Ritchie Russell[18] shows a picture labeled "the limits of the area within which a small wound will cause aphasia." This takes in Broca's area and the entire second temporal gyrus. Furthermore, the dent in the superior aspect, which looked rather peculiar in the picture of Marie and Foix (and of Bailey), is entirely missing. The work of Russell is widely respected. But surely it is unreasonable to show a distinct boundary beyond which a lesion will not cause aphasia, and within which it always will. We already know about the data of Luria. Furthermore, we know about the "language formula-tion area," which includes the posterior part of the third temporal gyrus. So why does Russell show a distinct line of demarcation? Probably because it is easier to draw a distinct line.

Next we consider a book by Penfield and Roberts,[11] a book which has been cited as widely as any on the subject, and rightly so. But the book is a little confusing, because it contains several different versions of Wernicke's area in one book. One of these pictures is the Penfield and Roberts version of the literature (figure IV-8 on page 81). This is not *their* version of Wernicke's area—it is their version of everybody else's version. They do include Mills' and Nielsen's inferior area. They show a "writing center," not because they believe in it (nobody believes in it), but because it is part of the literature. But a strange thing about this picture is the inferior Rolandic and parietal areas! There is just a big empty space! Another surprise is that "Wernicke's area" in this picture consists of the posterior half of the first temporal gyrus and the angular gyrus. This is again unique! We have no other picture of Wernicke's area consisting of the first temporal gyrus and the angular gyrus alone; there is only this picture, which purports to describe the literature up until this time.

Well, what do Penfield and Roberts themselves believe? What did they conclude from their own evidence on the basis of stimulating or excising the exposed cortex of the locally anesthetized human? First, there is the stimulation evidence. On page 135 (figure VIII-14)[11] the area in question includes the foot of the first temporal gyrus, the posterior half of the second, a bit of the third, and it takes in the angular gyrus and the supramarginal gyrus; but it does not include the inferior Rolandic area. Why not? Apparently, they excluded *that* area because stimulation there not only produced interruption of speech but also caused a behavioral deficit which they interpreted as a motor disorder. So they followed the peculiar convention of Dejerine.[6] But is this region not important for language? Of course! Picture VIII-14 is not a picture of their data (which is shown on page 122); it is a picture of a theory. The data picture (VIII-3) is particularly notable for showing something new; no one before this pointed out the role of the posterior part of the third (superior) frontal gyrus. It turns out that there is not only no dent in the superior aspect (as shown in the picture of Marie and Foix) but, in fact, there is a bulge.

Penfield and Roberts show another picture (X-4 on page 201)[11] from stimulation evidence; but, to our consternation, it is slightly different! The first temporal gyrus is not even included. Wernicke's area (as shown in this picture) omits the third or inferior temporal gyrus. Indeed, the mystery here (as on page 135) is that the first temporal gyrus is not included, although everyone else agrees that it should be included except for Fulton. Why have Penfield and Roberts left it out? It is not because stimulation there did not interfere with speech, as we can see by looking at

their data (picture VIII-3 on page 122). The data picture really looks a great deal like Bailey's version of Marie and Foix.[8] And it looks very much like the picture of Russell.[18] In fact, we are beginning to come to the conclusion that when we have a picture of the data, rather than someone's theory, that we will have a picture that looks pretty much like all the other pictures of the data.

Next is a Penfield and Roberts[11] picture of their excision evidence (Figure X-10).[11] They left out the first temporal gyrus again! This picture is not the same as those on pages 135 and 201, where they also left it out, because in this picture Wernicke's area goes all the way down to the inferior surface of the hemisphere.

In another picture (IX-23) from Penfield and Roberts, based on excision, the first temporal gyrus is finally included, which is a great relief, since otherwise it might suggest (as does X-10) that if excision of this gyrus were done, it would not cause aphasia, which, of course, it usually does.

We consider next a picture from Masland,[19] an eminent figure in contemporary neurology. His representation of the language regions does not include the supramarginal gyrus at all. What happened? He has there only a blank space in the very place where Henry Head[10] said a lesion causes semantic aphasia and where, according to Luria,[17] you have a 53% chance of losing phonemic acoustic perception. And it is the same place where, according to Geschwind[20] (as we will see later) a lesion causes "conduction aphasia." Small wonder that the student can easily become confused.

A student might also be confused by the book of two other eminent authorities, Espir and Rose.[21] They give, in three different places, three different definitions of Wernicke's area.

Let us look at a picture drawn by a linguist, Harry Whitaker,[22] until now, at least, a good friend of ours. This was written for linguists by a linguist; he learned some neurology and summarized it for the linguists who would like to know something about the neurology of language. So he went back, all the way back, and what he showed them looked very much like Wernicke's picture of 1881; it is mainly the first temporal gyrus. But Whitaker's picture[22] is a little bit different from Wernicke's picture,[3] because it does not creep across the sulcus and, therefore, it does not include *any* of the second temporal gyrus. What happened to all of the intervening data in the preceding hundred years? Here we find the linguists starting all over again, right from the beginning. Perhaps that is a good idea, if the neurologists are as confused as they sometimes seem. In Whitaker's favor, it might be said that at least he did not draw sharp lines around his labels.

Next we have a picture from another good friend, Norman Geschwind (or he was a good friend until now). This picture (Figure 4.1 in Ref. 20) is sometimes called the Boston version. What he shows us consists only of the posterior fourth of the first temporal. He just leaves out everything else we have mentioned so far, the past century of investigation. He has Wernicke's area so shrunk down and so pushed up in a corner that it looks like the corner pocket of Nielsen's pool table. Why does he do this? It is because he is not drawing a picture to represent data. It is intended to represent what he considers to be the preferable theory, or, at least, the theory to which beginners should first be introduced. This is what we might call "a picture for the people."

The simplication by Geschwind goes even further, later.[23] In a 1972 article his figure shows Wernicke's area just as shrunken as before, but now it is peculiarly reniform. He does this, apparently, to illustrate a physiological theory; he is not giving a picture of the data, nor a picture intended to have literal anatomical significance. The trouble is that, given this simplistic version, many people then carry it with them the rest of their lives. There are a lot of molecular biologists and quantum physicists and mathematicians who are educated, sophisticated people, and

who are now taking an interest in language; if you teach them this simple-minded version, how will they feel several years from now when they realize they have been talked down to?

We are nearing the end now, with a picture from Jason Brown.[24] His "Wernicke's area" is beautifully egg-shaped; but it seems a little small (although it is certainly larger than Geschwind's). One reason it is so small is that he redefines it as the area in which a lesion will produce jargon aphasia if you are past middle-age. This certainly is one way of solving the problem of where Wernicke's region is, to define it in a different way. But the picture does have the virtue of suggesting gradients; it seems to indicate that the further away the lesion is from the first temporal gyrus, the less likelihood there is of something happening.

We can conclude with a picture from Benson.[25] His Figure VII shows what he calls Group B aphasia; that is, those patients who have a comprehension deficit but are fluent. This is not quite a picture of the data, because the numbers indicate the centers rather than the entireties of the various lesions. But the picture does indicate, to some extent, the locations of the lesions that cause comprehension deficit. And these locations are where one would expect them to be, from all of the previous pictures, with the exception that there are no cases with lesions in the posterior end of the inferior temporal gyrus. But that is just a sampling error. If Benson had had a larger sample, he would have had some cases down there.

CONCLUDING COMMENTS

Is there *any* way to draw a satisfactory picture of Wernicke's region? One answer would be that we need a picture, resembling a topographic map, that shows a probability distribution; that is, a map which shows the likelihood at any particular locus of a comprehension deficit from a lesion at that locus. A probability distribution of the sort suggested here may provide one approach toward resolving the long-standing issue of topism versus holism. In order to construct the real thing, we will require a vast amount of quantitative information. But this requirement should not be a weighty argument against the probability approach. Indeed, we should welcome an approach that can make use of a vast amount of data which has heretofore been conveniently ignored by the simplified schemes with which we have struggled in the past.

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