Revision of Broca Aphasia and the Syndrome of Broca's Area Infarction and its Implications in Aphasia Theory

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Before an audience this experienced in the analysis of aphasia, any speaker is well advised to proceed with caution and attempt to develop facts before launching into conjecture. Most of the following discussion is rooted in neuropathologic and CT scan observations which document the lesions that set the stage for the clinical syndromes. Special emphasis is placed, we believe, upon the evolution of the syndromes over time, instead of attempting to recount the fine details of speech and language abnormalities which have been well described by speech pathologists and neurolinguists, to whom clinical neurologists owe a debt of gratitude. (It is discouraging to review the neurological literature when one sees how simplistic and to some extent misleading have been the clinical characterizations of the speech and language abnormalities by us neurologists.)

Attempts at neuropathologic and CT scan correlation are limited to the small number of cases which have been subjected to careful speech and language testing, followed over a time course of change lasting weeks, months, and years and then finally come to CT or neuropathologic examination. In all, only a few hundred cases comprise the material from which such correlations can be made, even when one stretches the literature survey back into several languages over a period of almost a century.

Broca's original cases are illustrative of this point. In his two particularly well known cases, the clinical characterization was confined to observations that the patient's comprehension appeared to be normal but that the speech was so disturbed that the observer gained the impression that the patient had lost the ability to use the speech apparatus for normal speech and language communication. Reading and writing were either not originally within the skills of the patients or not tested in detail. From the neuropathologic point of view, it is remarkable that Broca's original contributions stress the importance of a lesion in the third frontal convolution of the left hemisphere, whereas lesions actually spread in continuity along the entire Sylvian fissure, including the insula and the inferior parietal lobule. Earlier authors suggest that Broca was attributing the syndrome encountered to only a part of the lesion, because of the dogma of his teachers, who taught that lesions began as a small focus and spread slowly outward. With the long history of clinical deficit in his two cases, it was possible for him to claim that the remainder of the lesions encountered at autopsy was subsidary, secondary, and later in development. We would not accept these views today, since we now believe that ischemic events which lead to infarction of the type seen in Broca's cases tend to occur in an estremely short period of time and do not simply make gradual evolution in size and scope typical of abcess, brain tumor and other types of abnormalities. This point of view was not prevalent in the day of Broca and for this reason, he may, perhaps, be forgiven for failing to emphasize the possibility that the entire lesion, from front to back, deep and superficial, may have been the basis for the clinical syndrome.

Broca did not lack for challenges to his view that the inferior frontal infarction produced the entire syndrome. Challengers have made their cases known from the earliest days after Broca's initial publications and have continued to the present day. However, most were confined to observations on one or only a few cases at best, and could be set aside as being exceptions to the rule rather than representing examples of syndromes which contradicted Broca's thesis. However, when one masses together the available cases in the literature and spreads them along a temporal continuum, it is apparent that the clinical deficit in Broca area infarction cases tends to disappear in a short time, while those with larger infarctions tend to be more persistent. Further, the cases described clinically as examples of Broca aphasia months or years after the onset of the stroke have with virtually no exceptions proved to be examples of large areas of injury consistent with those in Broca's original publication. The traditional literature shows virtually no examples of cases of infarction confined to Broca's area in which the speech and language deficit has taken the form of Broca aphasia when the patient has been examined years later. Instead, such cases are usually remarkably free of deficit, save for some disturbance in pronounciation and a mild abnormality in the use of the oropharynx for skilled motor function, which may qualify the patient for the term "oral dyspraxia."

Since cases with focal Broca area infarctions without Broca aphasia have been known in the literature for a long time, it might be asked why the theory that Broca aphasia is due to Broca area infarction has survived to the present day. One explanation is that a few Broca area infarction cases showed only extremely superficial infarction. When such cases were viewed from the lateral surface of the brain, the infarction looked impressive, but on cut section in a coronal view, infarction was confined strictly to the superficial layers of the grey matter and did not disrupt the underlying brain. A few such cases, reported early in the period of the original discussions of Broca aphasia, served to provide a documented explanation for all succeeding cases of Broca area infarction who underwent rapid remission of the syndrome.

A considerable number of such cases of Broca area infarction can be found in the literature. By contrast, it is difficult to come upon many cases showing a fully developed Broca aphasia syndrome months and years after the onset of stroke and studied later by autopsy. Many or most of the cases alleged to be examples of Broca aphasia have been described in clinical details only, and few, if any, well documented autopsy examples can be found in the literature. Enough of these cases exist, however, to have permitted authorities in the early years of the century to speculate that the initial syndrome labelled total aphasia eventually changes to one of Broca aphasia, so that years after the event, it might be impossible clinically to decide which patient with Broca aphasia earlier had shown a clinical picture of total aphasia. This specific point has been made by Liepmann and by other authorities who have (or had) considerable experience in examining such patients.

To summarize these points, it seems fair to say that at the present time the available literature indicates that a person whose infarction is confined to the Broca area experiences an initial period of mutism with some disturbance in language function, followed quickly thereafter by the emergence of speech which is remarkably free of agrammatism, that the person

rapidly recovers the ability to write well, and in the months and years that follow, may show only a residual facial-lingual-palatal-pharyngeal paresis, mild ideomotor facial dyspraxia for skillful movements, and only slight hesitancy in speech, taking the form of speech dysprosody or speech dyspraxia. A persistent disturbance in the proper use of grammar appears to be so rare as to be considered an exception to the rule. Few, if any, cases exist that show the Broca aphasia syndrome later one. One such case, kindly sent to me by his grandson, was reported by vanGehucten, whose patient experienced a deficit persisting for a full year. However, most cases that are described as examples of Broca aphasia months or years after the onset of the stroke regularly appear to show large infarctions which involve far more than the Broca area, and spread into the posterior portions of the operculum, involving most of the insula and even the inferior portion of the parietal lobe. These cases conform to the central territory of the upper division of the middle cerebral artery. Variations in the arterial collateral from the anterior cerebral artery may limit the degree to which the infarction spreads above the level of the operculum into the upper frontal and central regions. Any spread of infarction into the frontal and central regions appears to contribute to the presence of severe hemiparesis in such cases, but such motor deficits need not occur in order for the language disturbance to be present. In virtually all of these cases of larger infarction, the initial deficit appears to be quite profound in speech and language function. The patients are usually unable to speak at all in the first days and weeks, have impairment in comprehension of spoken and written language--particularly where grammatical contingencies are important for speech comprehension--and when they begin to speak, show the characteristic effortful speech and simplified grammatical structure as well as poor communication by means of writing.

A variety of clinical causes underlie the minor ('baby') Broca syndrome. These include embolus to the Broca area along the ascending frontal branch of the upper division of the lateral cerebral artery. A few other causes include anteriovenous malformation in the region, small putaminal hematoma placed near the subcortical region underlying the Broca area, a few cases of fragment or gunshot wounds, one example of an umbrella poked through the orbit which entered the skull, one of a stone which penetrated the skull and pressed on the Broca area, and an occasional case of subcortical infarction which involves occlusion of the lenticulostriate branch of the middle cerebral artery. It is probable that some of these cases in the past were given a label such as subcortical motor aphasia or pure word mutism. These clinical entities are extremely difficult to characterize, since most of the cases in the literature were only clinical descriptions or conformed to many of the features now labelled as minor ('baby') Broca aphasia. One thing is certain--most were not examples of pure subcortical injury to the brain, but instead involved cortical infarction or bleeding into the Broca area itself. It is probable that terms such as "pure word mutism" or "subcortical motor aphasia" will decrease in usage in future years, since they describe neither the unique pathology nor unique clinical syndromes.

The major syndrome of total aphasia leading later to full Broca aphasia also usually is due to embolic infarction of the upper division of the middle cerebral artery, but occasionally has been reported as a result of abscess, large putaminal hemorrhage, large hemorrhage from arteriovenous malformation and even post epilepsy, but the descriptions of the post-epileptic aphasic deficits are few and have not been characterized in any great detail. More work is needed in this area.

A number of unsettled questions still exist on the subject of the big Broca aphasia syndrome and the baby Broca syndrome. If the big Broca syndrome begins as total aphasia, can a lesion exist large enough to preclude any such change from total toward Broca aphasia? It is suspected that such cases exist in those total occlusions involving the complete middle cerebral artery territory who present early with verbal stereotypes and fairly accurate pronounciation but undergo virtually no change in the comprehension of spoken and written language. Such cases may be rare, and examples of complete middle cerebral artery territory infarction, front to back, top and bottom, superficial and deep are only scantily described. More studies on such cases are urgently needed.

Another question relates to how deep lesions such as hematomas alter the aphasiologic picture. Huge putaminal hemorrhages appear to produce a picture of total aphasia and in most instances undergo an evolution toward being Broca aphasia. However, a small one may produce no disturbance in language at all and even produce echolalia, the opposite of what would be expected classically from interruption of the nearby arcuate fasciculus. The fine details of language testing in huge putaminal hemorrhage is a bit lacking at present, so we are unable to determine if there are any unique qualitative features or if the time course of evolution produces a slightly different syndrome from that due to surface embolism. Any difference in such syndromes would help us considerably in determining what aspect of the brain's grey and white matter helps mediate the changes toward normal.

Another important question bears on how much behavior is completely spared that might be considered to be due to nondominant hemisphere function. Cases with total middle cerebral artery territory occlusions of the dominant hemisphere often articulate with normal voice early after onset and utter only verbal stereotypes. Such cases make one think that complete infarction of the dominant hemisphere may set the stage for physiological release of the articulatory skills of the nondominant hemisphere. By contrast, such skills appear to be held or suppressed when the injury to the dominant hemisphere is less complete, as it is in the cases of initial total aphasia or Broca's area infarction with the minor Broca syndrome.

Other questions also exist concerning the syndrome of Broca area infarction (baby Broca aphasia). The impact of bilateral lesions is only now being understood. We have seen four cases who have satisfactory articulation and good use of spoken and written language with CT evidence of bilateral Broca area infarction. The extent to which the patients show the big Broca picture appears to reflect the typography and size of the lesion in the dominant hemisphere; a large infarction in the dominant hemisphere seems to produce total Broca aphasia while a small one in the Broca area on the opposite side produces temporary mutism following which improvement toward prior speaking ability is seen. By contrast, when a small infarction in the Broca area of the dominant hemisphere is encountered, followed by a large infarction in the nondominant hemisphere also involving the Broca area and adjacent areas, the effect on speech and language is like that of a small infarct in the dominant hemisphere. These cases lead us to believe that the tissue in the region of the dominant hemisphere surrounding the Broca area may in some way share or carry a portion of the physiological effort involved in normal speaking and use of language. Perhaps part of the restoration of function or improvement in skills after a Broca area infarction represents some contribution from those regions of the dominant hemisphere that surround the Broca area rather than simply from the isolated activities of the Broca area of the

nondominant hemisphere. How large an infarct exists before the baby Broca syndrome becomes that of total aphasia resolving to Broca aphasia? So far, there is very little understanding of the spectrum of abnormalities from the minor to the major syndrome and the exact neuropathologic correlations. How small can the infarction be to produce even the baby Broca syndrome? A case with Rolandic infarction reported by Lhermitte and Roche-LeCour is instructive here, since the patient's initial mutism gave way to normal language function. The Broca area was completely spared. What are the coexisting motor deficits in such cases, and how do the changes in such motor deficits over time mirror the changes in the language deficit encountered? There exist about forty or so cases, in which changes in lingual, facial, oropharyngeal, and brachial pareses have been compared with changes in the language deficit. It has been our impression that the language deficit undergoes changes toward normal at a considerably faster rate than that of the sensorimotor abnormalities. Could the comparative time tables of the motor and language abnormalities be used in some predictive fashion? Still another question refers to the importance of the depth of the lesion. This issue has been addressed only by Kleist, who in 1934 was of the opinion that if the infarct of the Broca area was sufficiently deep, it might be able to produce a picture essentially identical to that of the Broca syndrome. This opinion has not been tested fully in modern times, but if confirmed, would have an important impact upon our understanding of the role of the white matter in such cases. Finally, the most important question of all is, can the knowledge that we gain concerning these syndromes be used therapeutically either to speed recovery or to see that recovery takes place at all?

The issue of the nature of the changes toward normal remain unsettled. It may be proper to use such words as recovery, restitution, or remission, but all of these terms imply that we have full understanding of the mechanisms at work that make the patient's performance improve. Whether or not there is a simple quantitative improvement of original performance or whether there is qualitative change in performance so as to compensate for or to reduce the importance of permanent aspects of the deficit remain something of a mystery. We use the term "amelioration" deliberately, to point up our lack of personal knowledge of the means by which the deficits seem to fade. We would speculate that performance improvements represent a qualitative change in the means by which the oropharynx is used for articulation and is not simply restitution or simple recovery of partially damaged tissue, but the matter needs serious investigation. Settling the question of the mechanism of change toward normal would go a long way toward providing insight into how the brain responds to injury in many areas. In this regard, it is instructive to notice that the material written by vonMonokov on the subject of diaschesis was drawn partly from study of cases of the Broca aphasia syndrome.

How generalizable the syndrome of Broca aphasia is as a model for change in brain function after injury might be open to question. As an example of some of the conflicting views on the physiology of these syndromes, we might mention the interaction between written and spoken responses in the syndrome of big Broca aphasia. It is traditionally claimed that the disturbance in writing is as severe as the disturbance in speaking and is of the same type. Such claims have led to theoretical formulations that the mechanical act of writing is achieved by the actions of the superior frontal region near the area considered to be the homonculus for the hand, and that the instructions for such activities are mediated through the orofacial region and the Broca area below. In such a setting, it might be anticipated that severe damage to

the Broca area might obliterate the capability of writing with the hand, or, in any case, that spoken language should be equal to or better than written language. Against such a background, we were interested to discover in our own researches whether the ability to communicate and use language by writing was superior to the use of speech from onset to final resolution of the deficits. It might easily be objected that mechanical impairment in using the oropharynx to speak preclude demonstrating that speech was in fact superior to writing. For this reason, we waited until the patients were capable of repeating aloud the various spoken responses called for when they looked at printed words or looked at pictures. We then compared their written response to the picture stimulus with their spoken response to the picture stimulus. It was interesting to note that the written response was better organized, more accurate, and scored higher by impartial observers than was the spoken equivalent, even though in another test in the same test session, the patient had repeated aloud these required words quite easily. Their repeating aloud at least indicated that the mechanical capabilities of oropharyngeal and respiratory function were equal to the task of making the required sounds. Yet none of these sounds occurred in response to the language task of producing the sounds as words in response to pictures. Writing was superior to spoken naming in all of the cases thus tested. These data suggest that the use of the written mode for language may not be dependent upon the same interanatomic connections as speech, and may free us from some of the requirements that postulate that writing is dependent upon speaking.

This discussion brings us to the question of the impact of our findings on theories of higher cerebral function. We have indicated that it appears that Broca aphasia is a late syndrome which emerges from an initial picture of total aphasia and involves a lesion larger than Broca area. We have also indicated that the Broca area infarction produces a syndrome rather different from and far less significant than that of Broca aphasia, and appears to have its permanent effects on the use of the oropharynx for skilled motor acts, and that it is not necessarily and not characteristically a disturbance in language. Such cases have made the syndrome of pure word mutism or subcortical motor aphasia a bit harder to justify. The entire series of findings raises other suspicions, since we have discovered that the minor (baby) Broca syndrome has been precipitated by infarctions even outside the Broca area, along the operculum in the insula, in the subcortical white matter, and even high on the Rolandic cortex. Further, the recognition that the major (big) Broca syndrome involves damage fairly far posteriorally along the insula and operculum puts some strain on traditional connectionism as a theory of language function. It might even be speculated that the regions of the upper division, (the Broca area, sensory motor cortex, inferior parietal region, and the insula combined) work in a synergistic fashion for the formation and generation of grammatical structures in spoken and written language, and that they also mediate the comprehension of the grammatical aspects of seen and heard language. It is our current thesis that these regions work together in a synergistic fashion, so that small injuries at any point along the system are insufficient to shut the whole system down, but that large injuries may so damage it that a qualitatively changed state emerges. We have elsewhere used the metaphor of a soccer team, which, even though several players are sent off because of injury, can still preserve the essential functions of the team. However, if too many are sent off, the remaining players must undergo a radical change in their mode of play in order to function at all in an organized manner.

Such a metaphor opens the door to the possibility that the behavior we refer to as language could be the artifact of a series of individual activities, so that what we call language is in the whole greater than the sum of its parts. If so, we need not look for language to be in any particular spot in the brain, but rather expect it to emerge as the product of team or corporate action from individual components which themselves produce only components of language.

At this point, the discussion has wandered sufficiently far from fact and into theory that a change toward more scientific consideration seems in order. But before leaving the topic altogether, perhaps you will allow my speculation, in passing, that the syndrome of Wernicke aphasia, like that of Broca aphasia, may be a historical myth. Modern data support our revisionist view that pure word deafness is really the auditory form of Wernicke's aphasia, that alexia with agraphia is the visual form, and that when they coexist, the full syndrome of Wernicke's aphasia may occur. There is little evidence in the literature that the syndrome of pure word deafness is due to subcortical lesions. Instead, it appears to be due to lesions that would, on inspection of the autopsied brain alone, lead most viewers to consider that the patient clinically must have had fullblown Wernicke aphasia. The lesions in most instances of pure word deafness lie in the superior temporal plane exactly in the region currently described as Wernicke area. Cases of pure word deafness regularly have disturbances in speaking with paraphasic substitutions, so much so that the presence of paraphasic errors is now considered part of the syndrome of pure word deafness. This violates the theoretical structure of the syndrome, since it is supposed to be purely a disturbance of impaired comprehension of words heard.

Similarly, examples of the syndrome of alexia with agraphia almost invariably include speech disturbances of a similar paraphasic sort, so that it is difficult to maintain that the patient's disturbance is confined strictly to problems in reading and writing. If one sets aside the theoretical requirements that cases of Wernicke aphasia have combined alexia, agraphia, word deafness and paraphasia, it might then be considered that the usual syndrome of word deafness is merely the auditory form of Wernicke aphasia and the usual syndrome of alexia with agraphia is merely the visual form of Wernicke aphasia. Such division of the syndrome of Wernicke aphasia argues against a central organic entity. It also does indirect damage to the basic theory of connectionism and reduces the importance of the search for a supramodal language center, such as the one that is speculated to exist in the angular gyrus. Such a theoretical structure for the revision of Wernicke aphasia would bring it in line with current observations made on the syndrome of Broca aphasia as well. More data are needed on the subject, but the present data appear to be leading us in that direction.

Turning finally to methodologic consideration, it seems important to emphasize the change in patients with Broca aphasia, big and small, over time. It is difficult to study any given case in detail in a single session. For this reason, many cases have been set aside until a chronic state is reached, at which time it is assumed that the scores on tests will be more or less uniform over a long period of time. In waiting for such alleged stabilization to occur, the most interesting aspects of the evolving syndrome often are overlooked or are no longer present when the more static state has been reached. Further, a number of cases have been lumped together who have been studied at different points in their time course, and the averaged data produce a somewhat misleading picture. It seems a good plan to emphasize

test methods that will permit rapid documentation of the elements of the syndrome and then to repeat these tests over a considerable period of time. In this context, it has been our personal experience that five stimuli of the same class presented for spoken naming are usually insufficient to give a representative score and that upwards of twenty are needed. When twenty, say, picturable items, are used for spoken naming response, the numerical score achieved can be depended upon to reflect a change across time that is fairly consistent, or at least shows a fairly consistent trend.

It is our belief that our understanding of the language deficit is as much a function of the method used to define the deficit as it is of the deficit itself. Quite clearly the method used to define the deficit gives us the picture of the deficit as we currently understand it. In some ways, it may force the shaping of the performance along lines that follow the constructions of the tests, instead of having the tests designed to explore the nature of the performance. Particular mention needs to be given to tests that are of such great complexity that the low scores of the patient virtually defy analysis. Other tests are so simple that the patient soon passes to the upper limits and beyond, leaving us with the false impression that performance is normal. It is extremely difficult to adjust the level of difficulty of tests and still maintain the interanl consistencies required for the tests to be performed. So far, this problem has defied many groups of investigators who have attempted to deal with them.

Finally, it might be asked, at what point in time after onset of the deficit does treatment become valuable? It is my current belief that intervention at virtually any point would be helpful. We favor the initiation of speech treatment as early as practical, even at the patient's bedside. We have encouraged the speech pathologists to come to the wards and work at the bedside of the patient, even before the patient is well enough to be moved to the more traditional desk and chair setting in separate rooms, where speech pathology testing and treatment is usually carried out. The earlier the clinical deficit is analyzed, the clearer is the extent of the deficit and in the end the better will be the understanding of the syndrome.

Please let me express my appreciation at being given the opportunity to address so interested and dedicated a group. I hope the remarks have not proved, as Bastian observed in the last century, "unduly wearisome and difficult to follow."

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DISCUSSION

Q: I'm very interested in your comments on Andy Kertesz's work on patterns of recovery.

A: Well I don't have any objection to it. I think he has done a nice job but didn't encounter everybody in their earliest stages. That is the only remark that I have. I have no argument with him. In fact, I think he has documented a few cases of different syndrome than would be anticipated from anterior lesions--which I take as a very exciting development. They haven't been all motor disorders from lesions up front. Some would even pass for Wernicke aphasia. I like that. It makes me think even more of unrecognized so-called neuroplasticity.

Q: Can you tell us something about positron emission tomography?

A: In positron emission tomography the concept is that the radio emitter is sending out the equivalent of a radio beacon which is located in two planes. It is non-invasive and gives a cleaner concept of cerebral blood flow than we have had up till now. In the past, the use of Xenon inhalation studies has required that we scan for a transmission image.

We scan through the head and hope to see where the changes in the relative distribution of the agent are. In positron emission, the emitting agent is going into the brain, and is sending out a "beacon" that we record so that we can get a 3-dimensional image much better than we can with Xenon. The images are sharper, and they give a much clearer understanding of blood flow. In measruing blood flow I don't think we learn much about lesions. It has regularly been demonstrated that dead tissue may have blood flowing over and through it. Characteristically it does, because that's how the body's scavenging agent, called macrophages, get into the damaged region to remove debris. It has long been known that angiographic documentation of lesions is pretty hazardous because the extent of the brain injury may not be reflected in the extent of the blood flow that is apparent. If there is an embolus that blocks an artery for long enough to kill the territory, and the embolus later is pushed distally by the force of blood and reflow occurs, the angiogram will show you the blockage many centimeters upstream from where the damage was. Similarly, with positron blood flow studies we are looking at the response of the brain to the injury; consequently it does not localize the injury as well as CT scanning. So I think scanning and its variants are probably better for the purposes that we have in mind.

Q: I am curious about your reaction, based on your review of the literature, to the quality of behavioral descriptions on which you base your views on Broca's aphasia?

A: I will be glad to respond to the question of the apparent degree of completeness in the description of the big and little Broca's syndromes in the literature. The completeness of the descriptions of the little Broca cases in the literature are very gratifying, because almost every case described was written by the author to hammer away at the idea that Broca area infarction equals Broca aphasia. Many of these cases were described in gratifying detail from Tuke and Fraser in 1867 on up to the present day. Descriptions of the distance the patient walked after he got off the tram, how far the umbrella fell, what he said when he came in the door, how long he sat at the supper table, how long before he began talking; and so forth. These reports were done by physicians interested in arguing against the Broca thesis. So perhaps 20 or 30 patients are really nicely described--it's just wonderful to read Bryan Bramwell's case--the case that Charles Foix had. Many of them are nicely described and very gratifying. Not so gratifying is the detailed description of the case material in the <u>big</u> Broca cases. Many of them, unfortunately, were like the material Nielsen described, "obvious Broca aphasia, comprehends well, doesn't talk, etc." Very dissatisfying. The most that I could use them for was to show that this semiqualitative description was given of the patient's deficit 9 years after his original stroke. No changes occurred until he was seen in July 1888, at which time he was unable to write, he did not say anything, he appeared to understand everything that you said to him, etc. Most of the descriptions of the big Broca linguistic disturbance is, I would say, from 1930 to 1970-75. The work of Goodglass, Quadfasel, and the like in this regard is excellent. I would say that none of the work that we have done adds anything to those beautifully described clinical pictures. Goodglass and Quadfasel found they were unable to, or had a great deal

of difficulty, encountering patients with so-called pure Broca's aphasia in their study in which patients were seen two months after the onset of the deficit. Either the patient was normal by the time of referral, or they were so complicated that they had to call it more than Broca's aphasia. It is that latter remark that runs through the work of most 20th century aphasiology--the harder they look or if they look at all, cases which are called Broca aphasia regularly have difficulty understanding what they read, etc. There is a lot of literature on that, but no there are not a lot of nicely described cases of big Broca aphasia with pathology documented from the onset of the deficit through, say, ten years. The only ones we know are the ones that we have, and a few others.

Q: I find it interesting that once we've got J.N. Nielsen severely criticized and forgotten about, everybody got excited about the right hemisphere as a spare for recovery. Now in your paper with Levine, I think you were suggesting that recovery might be more a function of intrahemisphere happenings than interhemisphere happenings. Would you comment on that?

A: First I will comment on the question of how patients with bilateral Broca area injuries appeared to undergo change. We got the impression from our four cases, and the others that we could review, that the size of the injury in the dominant hemisphere is what predicts the nature of the improvement. So that it was not so important that the lesion was bilateral. That appeared not to be the issue in terms of whether they could talk again. Instead, the quality of speech, the language content of speech, appeared to be a reflection of the size of the left hemisphere injury. In other words, it was almost as if the right hemisphere's contribution to the speech and language deficit was miniscule, so that a small injury in the left side--in the broca's region say,--and a huge injury on the right side would be associated with recovery or improvement or amelioration of the deficit typical of a small injury on the left side. Whereas a large injury on the left side, with gradual improvement in Broca aphasia, followed by a small injury on the right would send them back down into mutism for a little while, and they would gradually recover again or improve or ameliorate their deficit. Yes, we did think that the topography of the lesion in the dominant hemisphere was the controlling factor in the nature of the acute changes and the qualitative characteristics of the syndrome. The bilateral infarct cases that we have depended upon all of these years--like the minor Broca aphasia or baby Broca cases--have been studied only acutely. Tuke and Fraser's case was a 14-year-old rheumatic who had bilateral infarcts within a 14-day period and who died, I think, in ten days. Ten days may not be quite right, but it was a short course; bilateral infarcts, mutism, died. And that is how many of them are studied.

Q: Since aging itself seems to produce changes in the brain, and since the initial infarct can occur at various ages, I am curious as to how you work out all of that?

A: So far, aging does not appear to be a factor in the improvement toward normal in the baby Broca cases. We have had them get well at 75 and 84 years of age. So, age, alone, even what we might call advanced age, doesn't seem so important. Age has not been a factor in the material

that we have in determining whether baby Broca infarcts are going to undergo improvement toward normal. They don't appear to take longer to start talking, and some young people with big Broca syndrome have been afflicted for many years, even though they were 14 or 15 years of age when they started. Whether or not the syndrome can change as the infarct changes is an interesting point. We had a case of a 36-year-old lady who was in a head-on collision. The accident fractured her mandible in two places, drove the mandible back toward the posterior pharyngeal wall, dissected both carotid arteries and occluded the left one. When she came into the hospital she had only a very minor speech deficit with a right facial paresis and we documented that she could do the very high level tasks. This lady was right on target on all of our quick tests and went to the angiogram suite, and we found the blockage, but before we could get her anticoagulated, and the surgery lined up, she extended the area of the infarction, and developed complete hemiplegia and a total aphasia. It was interesting to watch the change toward the big Broca syndrome when we knew from CT and angiogram what we had earlier. She is now coming to see me regularly and is doing well, apart from the fact that she speaks slowly and still has her hemiparesis (much better though). The acute effects that we saw in the emergency room were not global--that is the point that I felt was interesting. It has been said that the acute stroke so disorganizes the brain that you can't see the patient acutely because of diaschisis. Von Monakow coined the term. Most of his material comes from human studies of motor aphasia. As you go back through all the cases that he wrote in his lab book, there is case after case of baby Broca syndrome. I have been trying to find in his writings examples of so called diaschisis that would produce hemiplegia or hemianopia. I would think that nowadays he might not be as hep on the word "diaschisis" as he might be on "neuroplasticity." The traditional view of diaschisis was that the embolus broke up, passed on, and the brain was not really damaged anyway, and this is just a temporary derangement called diaschisis; but real infarcts make a permanent deficit that does not change. Now just shift over and say that real infarcts that are small (one gyrus wide will do), are what used to be called diaschisis. What used to be considered the permanent deficits are the big infarcts. Joe Bogen has been on to this for some time. He has wandered all around trying to find where Wernicke's area is--like the Holy Grail--he just keeps looking. It's apparent to him that everybody's description of where Wernicke's area is is largely professorial dictum--not based on clinical data but upon statements. I would say to get Wernicke aphasia and keep it, you have got a big dent in your head, and most of it runs from the superior temporal plane to the occipital poles. I think that makes big Wernicke aphasia and I think that little Wernicke aphasia is the auditory/visual form and that's what our data show and that's what we are writing up now.

Q: Would you care to share your views on apraxia of speech?

A: Not with the experts that I know are here. I would say that none of our work has done anything to change Darley's work. What I don't know and what I don't think many know is the topography of the lesions that used to be called pure word mutism and cortical dysarthria. Now pure word mutism used to be considered a subcortical injury--you know the old litany, that the cortex is really homebase. Everything lived there.

Messages came in and went out and one region was connected to another, and that is how you could have subcortical motor aphasia, transcortical motor aphasia, or cortical motor aphasia. Not bad, but as far as I can see, not right. Not bad though. It also follows the rule of three which I say are always bad in biology--right, left and middle, Father, Son and Holy Ghost, three strikes you're out. When I hear of three reasons or three layers or three structures, I know that it is almost always the human mind and not the biology that's responsible for the existence of the "three." I don't think that biology is set up in three part structures. In any case subcortical, cortical, and transcortical aphasia categories are not bad but I don't think right. I think most of the cases for which the label "transcortiçal motor aphasia" is used now, are examples of anterior cerebral artery syndromes which don't really qualify in the traditional terms. I think it is great history and a lot of fun tracking these cases all around, but most of the ones that are described are clinical, and they are not followed in much detail and there is no description of pathology. I would prefer that we enjoy reading these litanies at night. Copies of things like that should be in every bathroom and next to the tub just for enjoyment reading. It is not the stuff of the future, I don't think. At least it ought not to be. I would say that with the tools we have now, the old investigators would have gasped in amazement. They did the best they could. They analyzed cases well, but we have methods such as CT scanning now, plus much better behavioral methods. As a result, what was accepted clinically even 50 years ago is almost ludicrous now.

Q: Would you discuss the relationship between speech apraxia and oral apraxia?

A: I would say that it is frequent that in baby Broca cases there is lack of skill in the use of the oropharyngeal, laryngeal, labial, and lingual movement along with difficulty in talking. Although it is said that patients can lick a crumb from the lip, to cough, sniff, close one eye, pull back one side of the mouth, wrinkle the nose, kiss, puff, blow, etc. to spoken commands, there also are errors that occur in principle--dyspraxic errors, and incorrect movements, not just approximations to correct movements. I believe that most of us could recognize differences in the acoustical quality of the speech of the paretic as opposed to the dyspraxic dysarthrias. In the paretic dysarthrias that come from bilateral capsular infarct, there is a nasal quality to the voice and, within the limits of oral pharyngeal movements, the patient's movements are in principle correct. We have used X-Y scattergram plots to analyze the errors made by these patients. To some extent their precision is not bad; they make labiopalatal movements when they should make a labiopalatal movement, (Editor's note: Labiopalatal movements are extremely difficult.) but accuracy is quite poor and I would say those patients regularly have a disturbance in nonvocal oropharyngeal-lingual-palatal-facial behaviors to dictated commands. Now what I don't know is something I would like to know. We have not had enough cases studied in detail over time to know whether or not the nonvocal movements improved compared to the vocal ones. The problem is, we haven't been able to find a common basis upon which to quantify the two. When we ask a patient to engage in an oropharyngeal praxic response that doesn't involve talking, we don't have a way of quantifying the comparative difficulty of doing that

movement; say wrinkling the face, or blowing a kiss, compared with making a vocal response. So I don't know whether we are yet sharp enough quantitatively to say "all evidence of orophrayngeal-facial-lingual-palatal dyspraxia has disappeared and yet he still has trouble talking." I don't know that. I would like to know, and I think it would be of some interest to try to develop short batteries to ask that question but I think the question would be relevant only in the setting of patients who are improving. I will say that the persistence of easily documented oropharyngeal-lingual-facial-palatal dyspraxia; sniffing, coughing, winking, etc., in the face of almost complete return of speech, is routine. We routinely see cases in which the use of praxic nonvocal responses is demonstrably impaired when the patient's talking seems to pass most tests. However, these patients often continue to have trouble coming up with words, though. Many of them will say to me "Gee I can't think anymore," or "I'm terrible if I take sleeping medication." So I know that the improvement is papered-over.

Q: Would you comment on spontaneous recovery? Some say it runs its course after three months or six months, but your slides seem to indicate that it goes on much longer than that. Is that true?

A: I have no patience for the idea that spontaneous recovery stops at three months. These are not crops that grow in the field. It's just that the apparent rate of change begins to slow down, but if you place the patient in a favorable environment, they speed right up again. Give them a little fertilizer or whatever it is you do to interact with them. Place them in a setting where they can get most things correct. I don't think that testing for aphasia (this is just a bias of mine) should be oriented toward failure. I'd say the definition of the deficit is the length the examiner has to go to get the patient to do it right. If you were failing, you would quit too. In fact, if you don't get promoted, tenure, and all that, you quit. You say, "these guys are terrible, I can't stand them, I'm working too hard and look what it's getting me." And if you were being tenured, promoted and earning $200,000 a year, you'd say, "this is just wonderful." For a year. And then you'd say "These guys are terrible, working me hard and I don't get anywhere." They give you another $100,000, and "Terrific, wonderful." So I would say that the patients who like to come in and be tested are those for whom the contingencies are arranged for success. We used to run the Boston Test backwards and that was a lot of fun, because they would get most of them right early. In fact, for a long time we used to have a joke with the Eisenson by running the reading questions without the text. What did Jimmy do all morning?--you remember that stuff. "Swam, ate, worked with grandpa," that sort of thing. They got pretty good scores but had no idea what the text was.

Q: Are you trying to tell us that potential for improvement is equated with spontaneous recovery?

A: Let me answer the question two ways--I would say there is a change toward satisfactory performance that occurs in the environment, and I believe it is possible to accelerate that change and reach a higher level by properly generated speech therapy or properly designed testing to show the patient the way. I believe that patients do improve. Let's just say that their adjustment to the environment goes on in the open world even

if we don't do anything. But the rate of improvement seems to slow down. If you then bring them into the lab and set the contingencies for good performance, they undergo a change, and their scores begin to improve dramatically. This satisfies me that treatment or special kinds of testing makes for higher performance and might get it there faster. Treatment is not required for anything to happen, but I believe that treatment can make it happen faster and go farther. I would say that the real question is "What do we do?" I don't think we have a basic understanding at the moment (at least I don't) of what the mechanisms are that are involved in improved performance. I just don't know. It's a basic question. I'd like to know the answer.