

Profiles Of Aphasia: Test Interpretation
Regarding The Localization Of Lesions

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INTRODUCTION. The study of aphasia has intrigued us for well over a century because it offered the physician a possible mechanism for localizing lesions in the days before the existence of brain scans and CT scans, and it has always held the promise of teaching us more about how the brain worked, and how it was organized. More recently, the study of aphasia has become crucial to those of us who have the responsibility for attempting to rehabilitate patients whose life has been changed by this massive interference in the brain's capacity to process communication.

In spite of our best efforts to understand it, the brain evades our scrutiny, hiding in its protective box of bone and refusing to function if we probe into it. It is therefore necessary to study the brain more indirectly, through the use of tests. We put the patient into some kind of standard environment, present certain stimuli and require various types of responses. We then compare these results to those obtained from a standard reference sample and attempt to make interpretations about the functioning of a given brain at one point in time. A particularly difficult area of test interpretation is localization of brain functions and lesions.

Why should the clinical aphasiologist be concerned with the localization of a patient's lesion? You will remember that Wepman used to say that he wasn't particularly interested in where patients' brain damage was since it had little importance to the recovery process. However, this may no longer be true. It is conceivable that lesions in one location may have a different prognosis than those elsewhere in the brain. Possibly some day we will be able to develop specific treatment approaches for aphasias caused by lesions in certain areas in the brain.

From a more practical standpoint, our tests can sometimes localize lesions that cannot be discerned through the standard diagnostic techniques. In some cases, these standard techniques are not available, especially in the case of CT scans, or these methods may successfully distinguish one apparent lesion but miss some minor secondary ones. Sometimes findings from these more conventional methods are negative because the lesions are too diffuse, superficial, or transient. Occasionally a diffuse lesion presents unusual types of symptoms and the patient is thought to have psychiatric problems rather than aphasia. And, most commonly of all, a patient will have one verified lesion and after treatment for his acute medical problems will be discharged to the care of a speech pathologist or clinical aphasiologist for long-term treatment of the aphasia, only to have subsequent CVA in such a benign way that it doesn't call obvious attention to itself, but is discernible on examination of later test results. For these reasons it is important that we be alert to changes in test results which suggest either an extension of the pathology to different zones in the brain or a discrepancy between conventional localizing methods and a patient's symptoms. Such observations are important to the patient and his physician.

Let us turn to the brain itself and briefly review how it is organized to carry out communicative processes.

The organization of the brain is best understood if viewed from the phylogenetic or ontogenetic standpoint. Early in its development the brain is reasonably plastic and has considerable flexibility as to how it might organize itself. However, as time passes, nature must commit more and more of the circuits of the brain for specific functions. Initially, primary areas that are important to survival are committed first, and the brain develops areas for sensing and differentiating critical information in the environment.

These first areas to develop are the auditory areas in the temporal lobe, the visual areas in the occipital lobe, the tactile proprioceptive areas in the parietal area, and the basic motor areas in the frontal lobe. As the brain matures, it begins to discriminate more complex messages through each of these modalities and adjacent areas become responsible for coding and decoding more complex signals. Finally, in man at least, the brain attempts to integrate this information from each modality and combine all of the messages being received into a composite of what is happening in the environment outside of the brain. This apparently takes place in the general area of what the neurologist refers to as the angular gyrus area or what Pribram has referred to as the posterior intrinsic system.

Turning to consideration of output systems in the brain, Geschwind has hypothesized that information from the posterior part of the temporal lobe is generated and conducted anteriorly to the motor programming areas of the motor strip for gestural and graphic output.

Of course, this model must necessarily be an over-simplification of what really goes on in the brain during a communicative act. However, there is considerable evidence that this is a reasonable approximation of the organization of the brain. If so, lesions in specific areas of the brain should have a predictable result as far as test profiles are concerned. Let's examine some test results obtained from patients with lesions of known locations. What we will use as a point of departure is the Porch Index of Communicative Ability. This test offers the advantages of having high test-retest stability and interscorer reliability. It has a binary choice, multidimensional scoring system which is very sensitive to small differences in patients and performances and it holds content constant throughout all of the subtests. Therefore, it is possible to make inter-subtest and inter-modality comparisons and interpretations which aren't possible when various modalities are tested with different stimuli and different content.

The method by which we are localizing lesions in the brain is fairly straightforward and probably over-simplified. Through the use of the literature and through the analyses of actual cases on which we have localizing information, we have constructed a topographical representation of where certain communicative activities are localized in the left hemisphere. These general zones are shown in Figure 1. The middle cerebral zone includes some primary auditory functions, some basic verbal output programming and the frontal lobe speech areas commonly referred to as Broca's area. Lesions in this zone tend to produce what is commonly called Broca's aphasia, although in our experience these patients consistently have some demonstrable auditory dysfunction in addition to nonfluent speech.

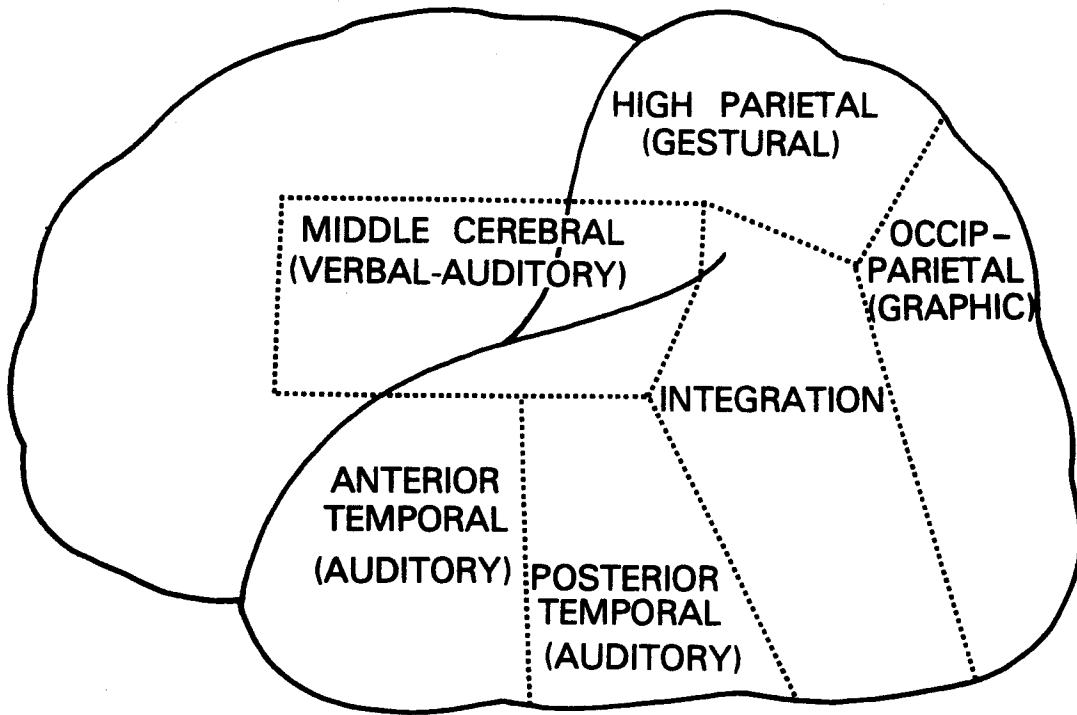


Figure 1. Cortical Zones Suggested by PICA Profiles and CT Scans.

The second zone of interest is the anterior auditory zone, which handles basic auditory processing and relatively shorter auditory stimuli than does the posterior auditory zone adjacent to it. We would expect patients with anterior temporal-lobe lesions to have more difficulty in handling shorter auditory stimuli than longer ones, which are decoded in the posterior part of the temporal-lobe. Conversely, posterior temporal-lobe lesions would produce breakdowns in complicated auditory processing while shorter messages might be relatively spared.

A third zone is the high parietal area. This is the zone that generally is described as handling proprioceptive-tactile input and is visualized in our model as being primary to the formulation of gestural responses. Immediately adjacent to this zone is the occipital parietal zone which is a combination of gestural and visual processing and therefore implicates graphic output. A final zone is the integrative zone which the neurologist refers to as the angular gyrus area but which is probably more extensive than that. This is the zone that Luria refers to

as a tertiary zone and which Pribram referred to as the posterior intrinsic system. This zone is responsible for combining and integrating information from adjacent zones; therefore lesions in this area tend to not involve primary processing, but rather more complicated integrative tasks.

Using this model of how communication is processed in the left hemisphere, we analyzed the PICA tasks to see which subtests might have these functions. Figure 2 shows a hypothesized schematic of how these PICA subtests might be localized in the brain. Moving through the zones that we have discussed, we have hypothesized that the middle cerebral zone handles the primary verbal output and some primary auditory processing. If this zone were intact but the rest of the cortex were no longer functional, a patient might have good imitative abilities on test XII, but would have very little other functioning intact, as is the case in diffuse transcortical type of aphasia. In some of the other zones the localization of

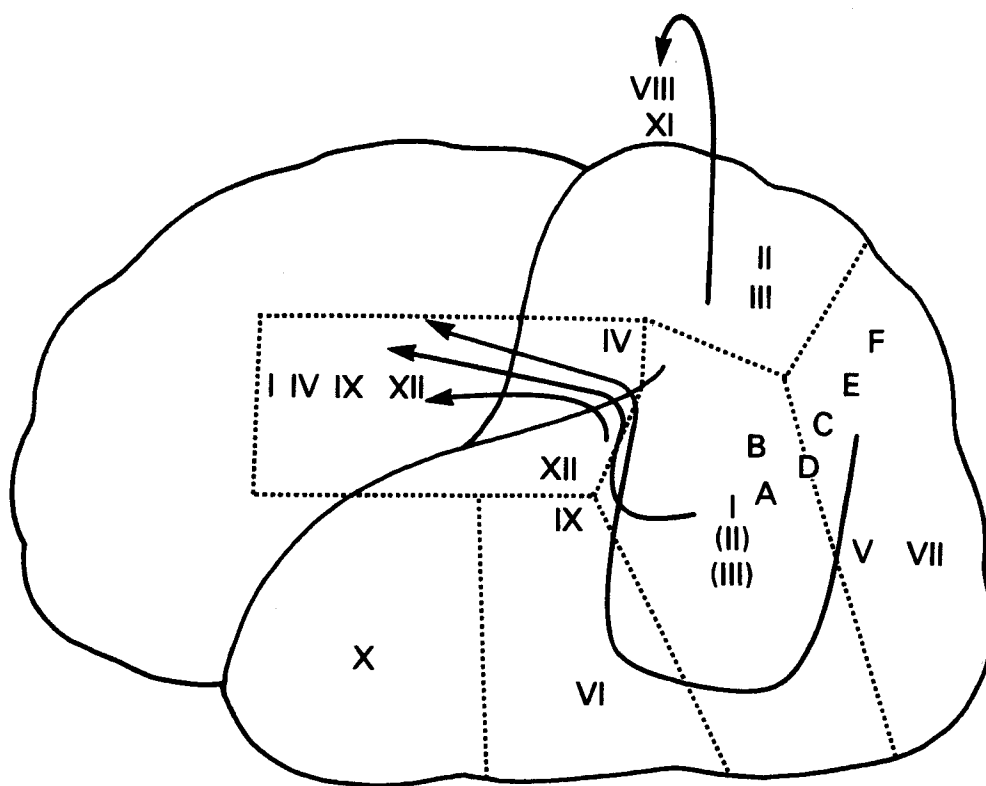


Figure 2. Hypothesized Localization of PICA Subtests.

tasks is as follows: Test X, the shorter auditory task, is localized anteriorly in the temporal lobe while Test VI, the more complicated auditory task, is visualized as being processed more posteriorly in the temporal lobe. Subtest II and III, which are gestural tasks, are localized in the high parietal area, while graphic tests C-F are visual-gestural

types of tasks which one would suppose are located in the parietal-occipital area. Reading tasks V and VII implicate the occipital lobe and more complicated tasks which involve multiple processes such as Test I and A especially would be integrative in nature. Finally, visual matching tasks would generally not involve linguistic coding and therefore could be handled in the right hemisphere. Therefore depressions in these more primitive processes would suggest that the right hemisphere is affected in addition to the left hemisphere.

Let us now examine some PICA profiles to see how well this model holds up. In order to simplify our discussion, we will restrict our comments to the Ranked Response Summary graphs. This graph displays the 18 subtests in order of difficulty, from the most difficult test for aphasic patients, on the left, to the simplest, on the right. Response level is established on the ordinate, going from the lowest response level (1) to the relatively normal performance level (15). The diagonal line on the graph represents the average performance of a large random sample of aphasic patients. Figure 3 shows a typical profile for a patient with a middle cerebral lesion. As mentioned above, the classic profile landmarks are depressed

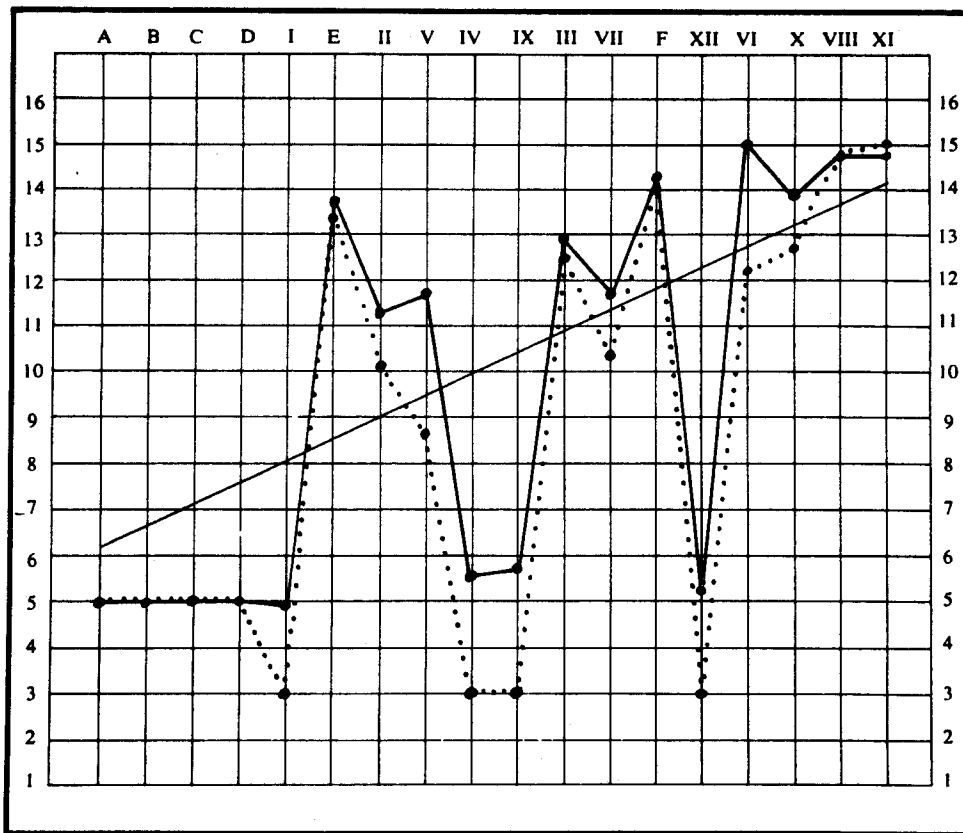


Figure 3. Left Middle Cerebral Zone Lesion

verbal ability and mild auditory defects. Later in the recovery process, such a patient will show gradually improving verbal ability, particularly in the easier and shorter verbal tasks at the right of the graph and there will be diminishing of the auditory dysfunction on subtests VI and X, while graphic tasks will improve as the patient acquires some skill with his non-dominant hand. An example of this type of patient is shown in Figure 4.

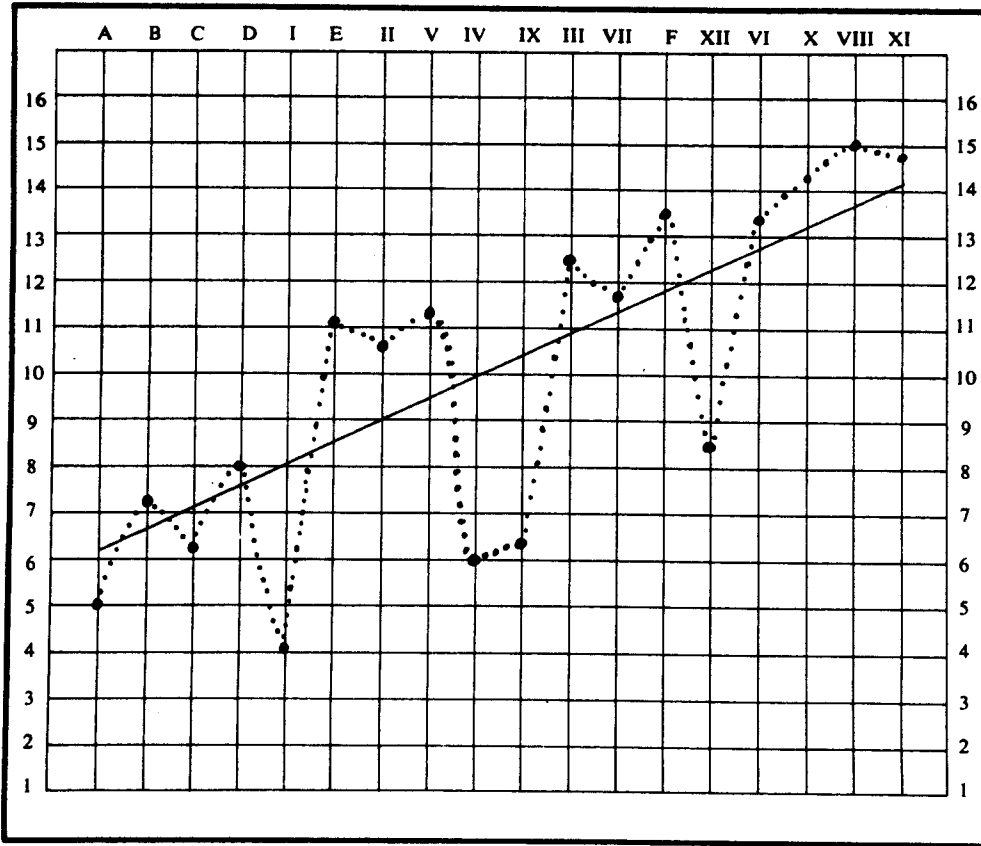


Figure 4. Left Middle Cerebral Zone Lesion.

Figure 5 shows two profiles of a patient who had a serious left hemisphere lesion incurred in a motorcycle accident. The trauma resulted in the removal of the anterior part of the temporal lobe in the left hemisphere. Initially because there were widespread contusions and edema, the first pattern at two months post onset is seriously depressed in all modalities with only some imitative speech remaining intact. This suggests that at this early stage he had what would best be described as a transcortical diffuse extensive lesion of the left hemisphere. At twenty-one months post onset, he had made extensive improvement in some of the modalities but the persistent involvement of the temporal lobe is still impressive,

particularly on the shorter auditory stimuli in Subtest X. Two other observations one might make from this second profile is that, because the visual matching tasks VIII and XI are better than the auditory VI and X, the lesion is now restricted to the left hemisphere. Secondly, because test XII, imitative speech, is at normal levels, we would assume that the middle cerebral zone is intact and that this lesion is now restricted to the left temporal lobe.

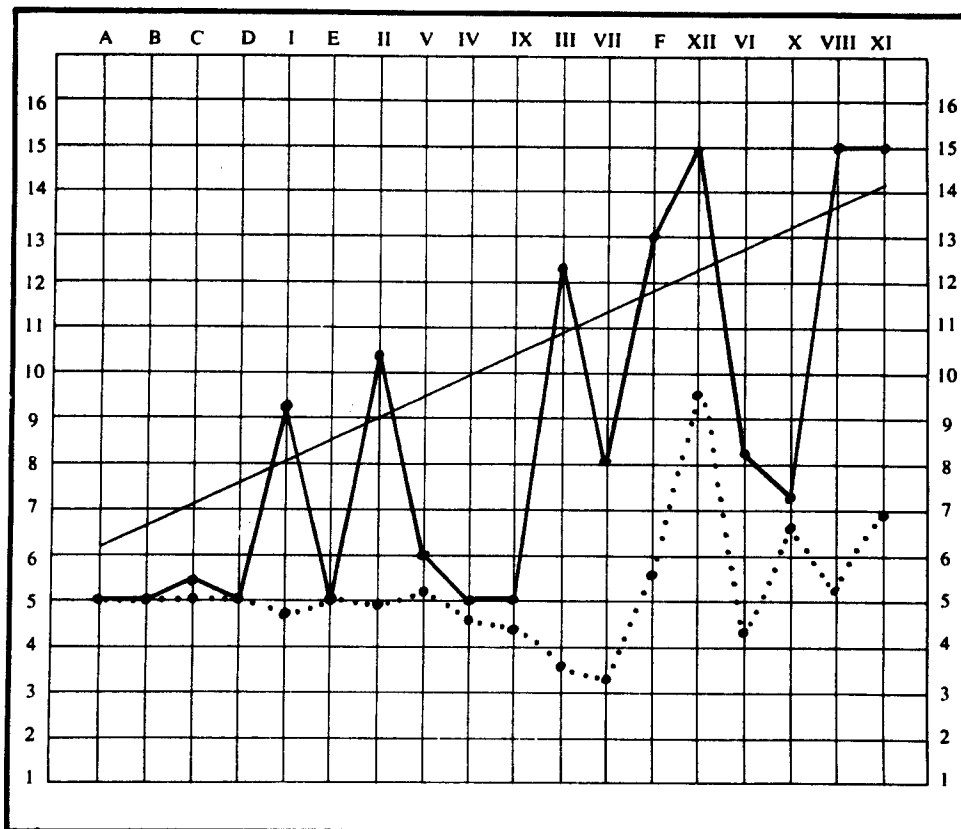


Figure 5. Left Anterior Temporal Zone Lesion.

Figure 6 is an example of a high parietal lesion on the left side. This patient had been struck on the head in that area and was described as having cerebral contusions. This lesion had a serious effect on his gestural processing ability, although the rest of his communication returned to near normal levels by the second month post onset as shown by the solid line. We can contrast this patient with a second case which demonstrates a patient with a CVA in the parietal-occipital area (Fig. 7). In addition to the parietal symptoms of reduced gestural ability on Test II and III, the patient demonstrates the depression of graphic ability.

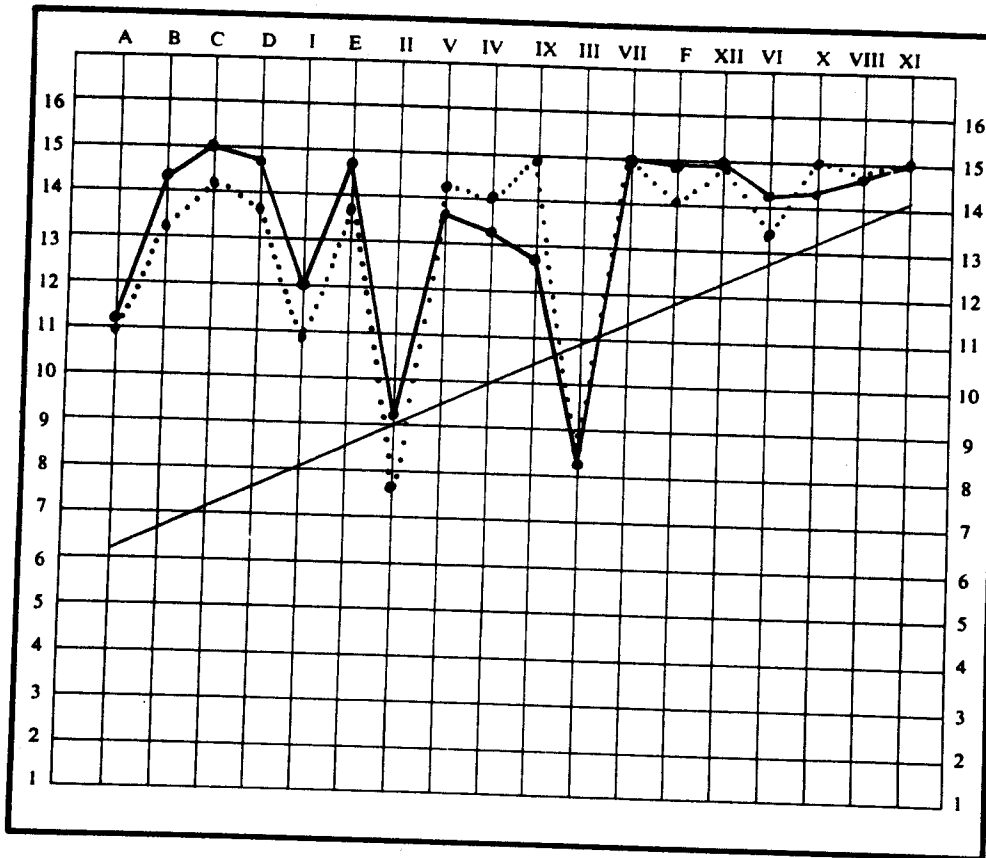


Figure 6. Left High Parietal Zone Lesion

The profiles in Figure 8 probably represent a change in the size of the lesion over time. This patient had a left posterior CVA which in the lower profile obtained at one month post onset implicated both the temporal and parietal areas of the brain, resulting in a large depression of auditory and verbal scores. As the edema diminished and the temporal lobe returned to a near normal level of functioning, the profile improved as shown by the solid line graph. Because this patient's lesion was in the posterior intrinsic integrative zone, all of the more complex communicative tasks were involved to some degree, although there were no longer significant depressions suggesting basic breakdown in any of the primary input or output systems. This saw-tooth curve with no dramatic peaks or depressions is very typical of a posterior intrinsic system lesion.

The next example shows two profiles obtained on a patient who had a CVA in the left posterior hemisphere (Fig. 9). The first test at two weeks post onset (shown by the dotted graph) is generally saw-tooth in nature, suggesting an integrative zone lesion, although at this point there is

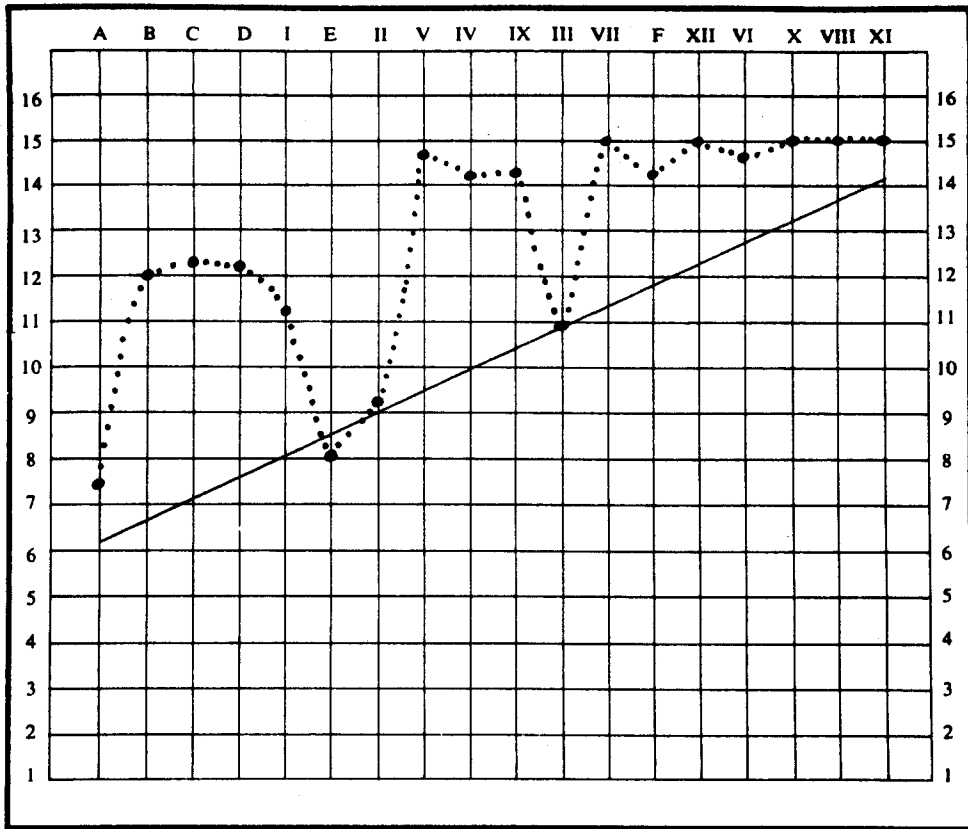


Figure 7. Left Parietal-Occipital Zone Lesion.

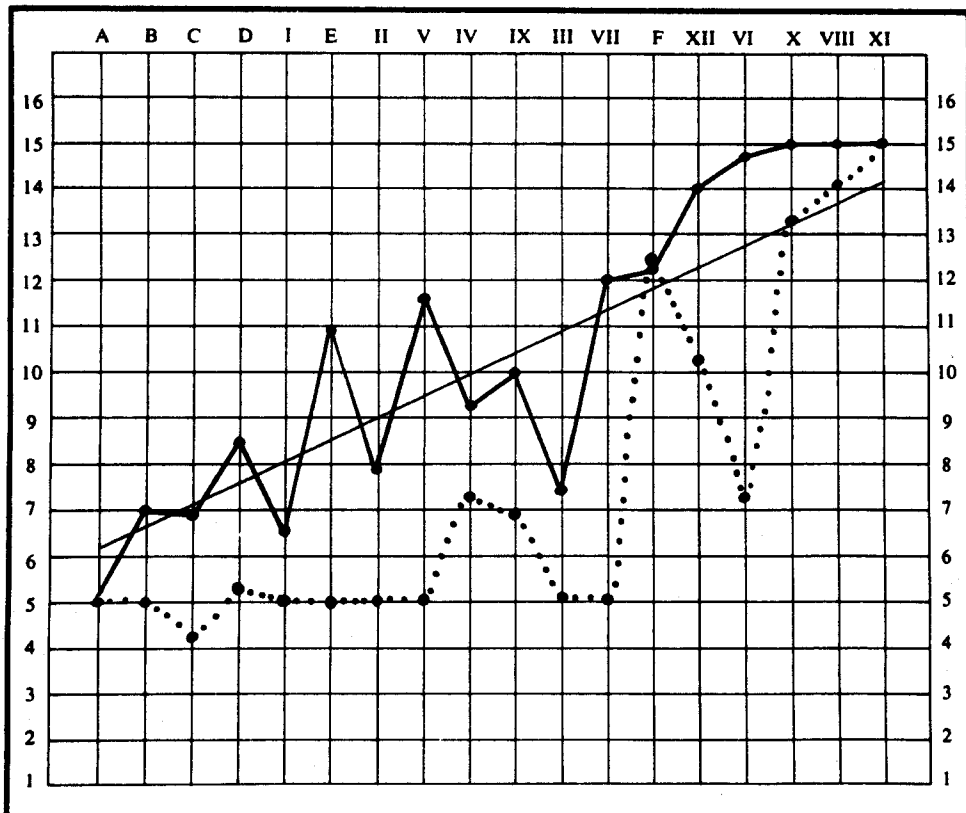


Figure 8. Left Posterior Integrative Zone Lesion.

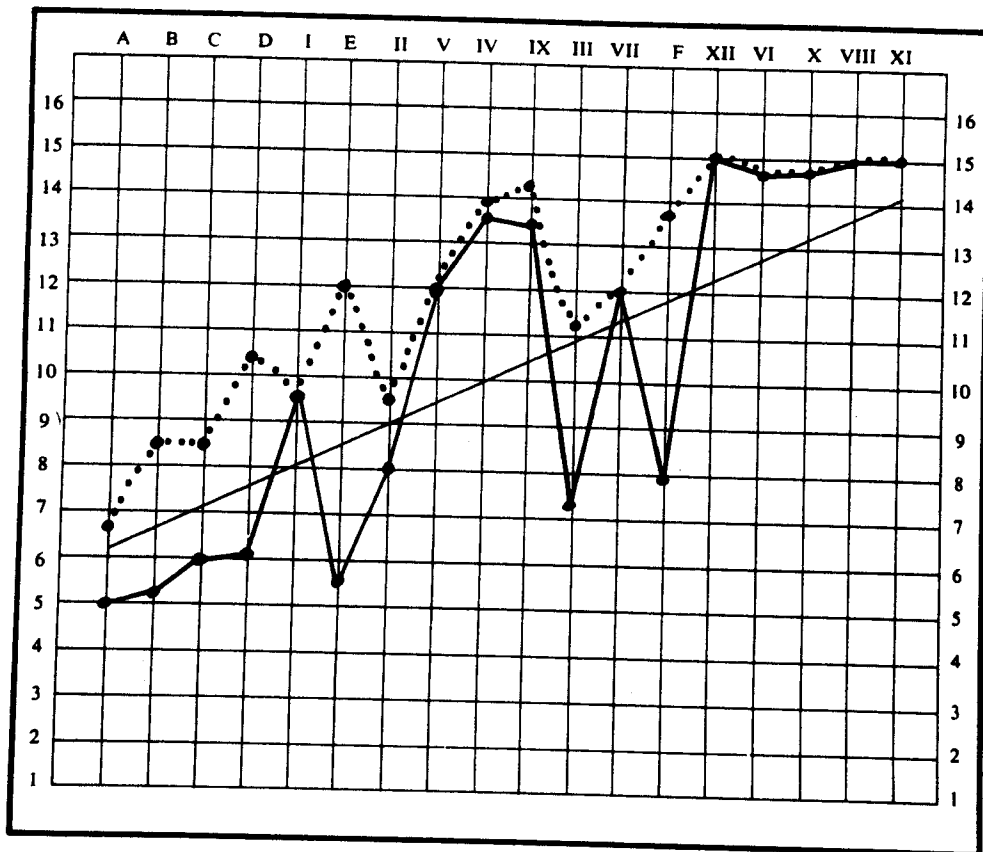


Figure 9. Left Posterior Integrative Zone Lesion.

definitely implication of the posterior intrinsic lobe, as demonstrated by the depression of test VI below test X. At the time of the second test, at two months post onset, most of the auditory problems had resolved and the profile improved significantly. The continuing depression of test A and I suggest that the integrative zone is still implicated and the marked depressions of II and III suggest that the high parietal area was still involved. These findings conformed exactly to those of the CT scan.

The final series of profiles demonstrate various forms of bilateral brain dysfunction. The first example (Fig. 11) shows a classic multiple CVA bilateral patient who demonstrates the three cardinal PICA signs of bilateral brain damage. These are as follows: 1) visual auditory reversal in which either VIII or XI, the visual matching tasks, are below either of the auditory tests, VI or X; 2) verbal scores are disproportionately high considering the rest of the profile; and 3) graphic tests are disproportionately low considering the rest of the profile.

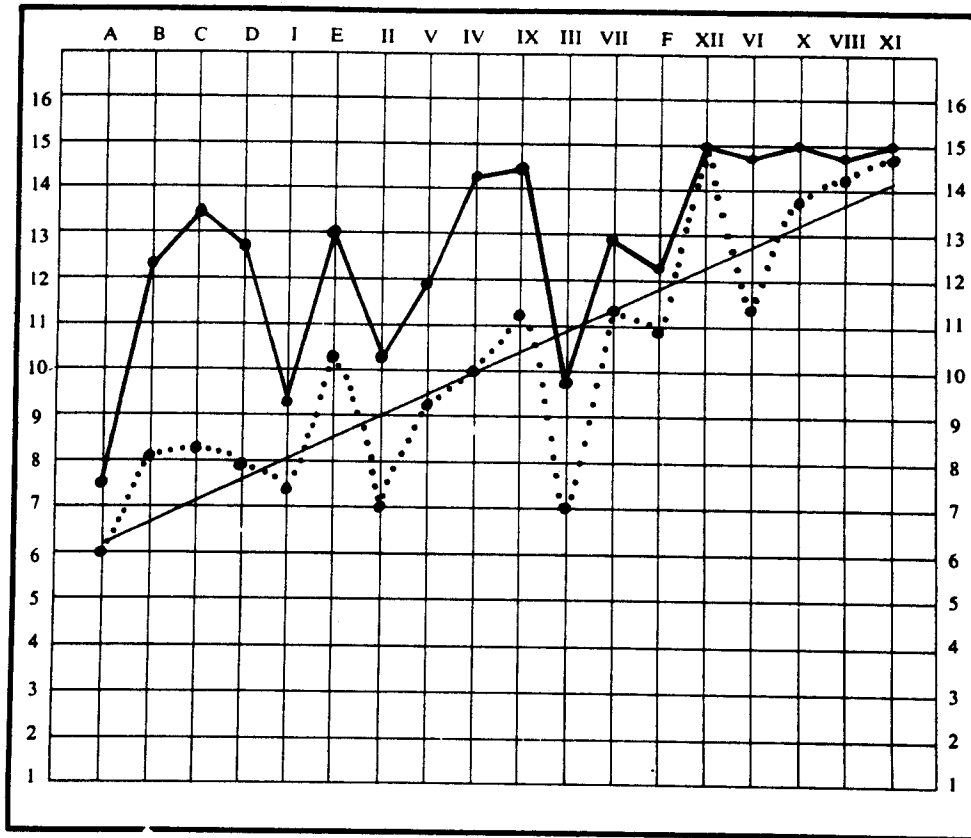


Figure 10. Left High Posterior Integrative Zone Lesion.

A second variation of bilateral brain damage is shown in Figure 12. This patient had serious head injuries that resulted in generalized contusions and edema and upon first testing at one month post onset (shown by the dotted line) he not only manifested bilateral brain symptoms, but, because of the high peak on test XII (imitative speech), he demonstrated that, although the middle cerebral zone was still intact, his cortex was non-functional and he demonstrated what is commonly referred to as a transcortical aphasia. On second testing at two months post onset, much of the edema had subsided and his second profile shows marked improvement in all modalities although he showed a bilateral brain dysfunction. Occasionally, if there is no primary tissue lost in the right hemisphere and the right hemisphere effect is due simply to edema or increased intracranial pressure, the pattern can improve and become essentially a left hemisphere pattern, as shown in Figure 13. This patient suffered a traumatic lesion to the brain and initially had a bilateral transcortical pattern, but a month

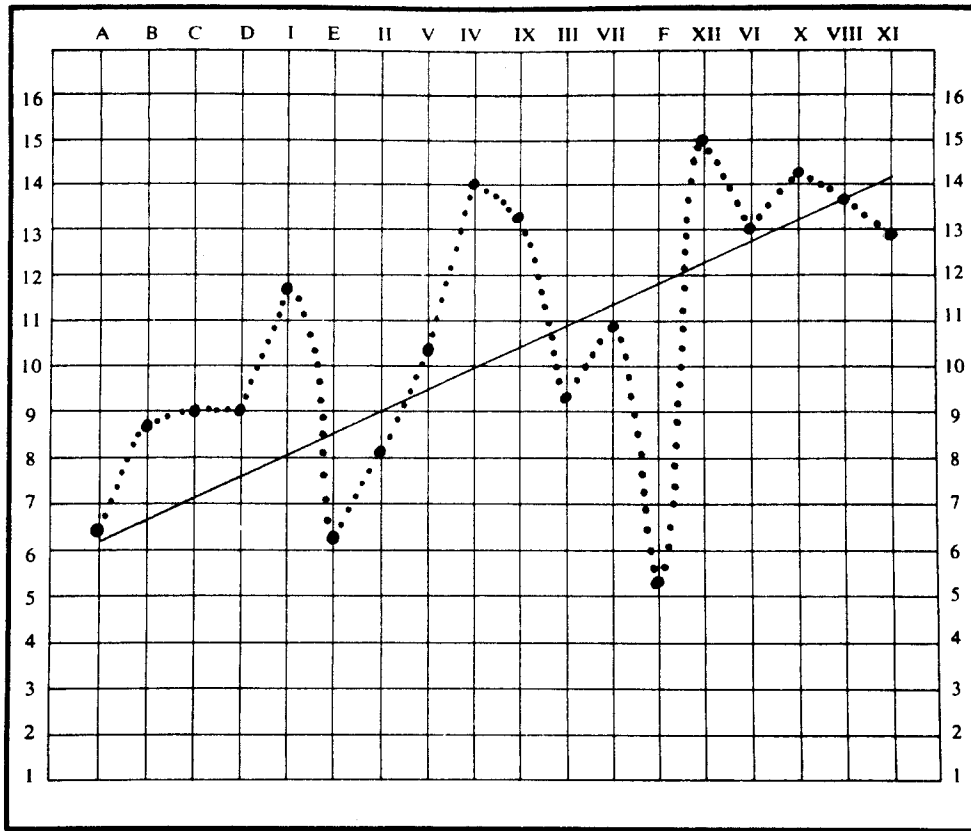


Figure 11. Bilateral CVA Pattern

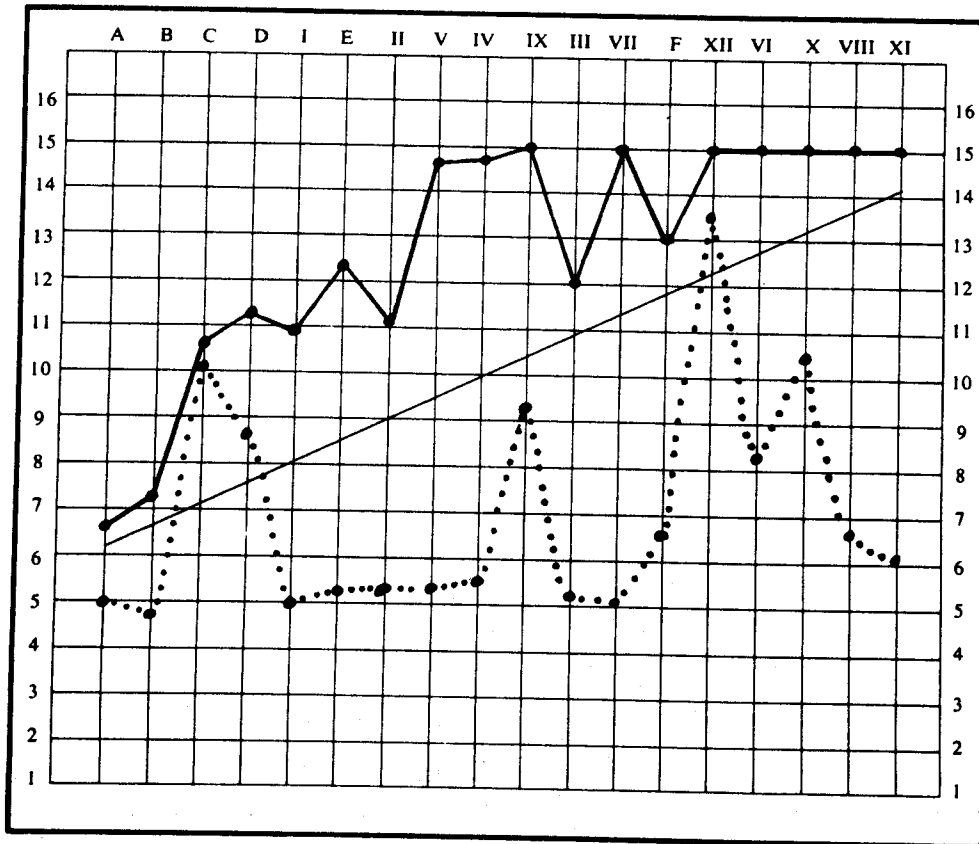


Figure 12. Bilateral Transcortical Pattern Traumatically Induced.

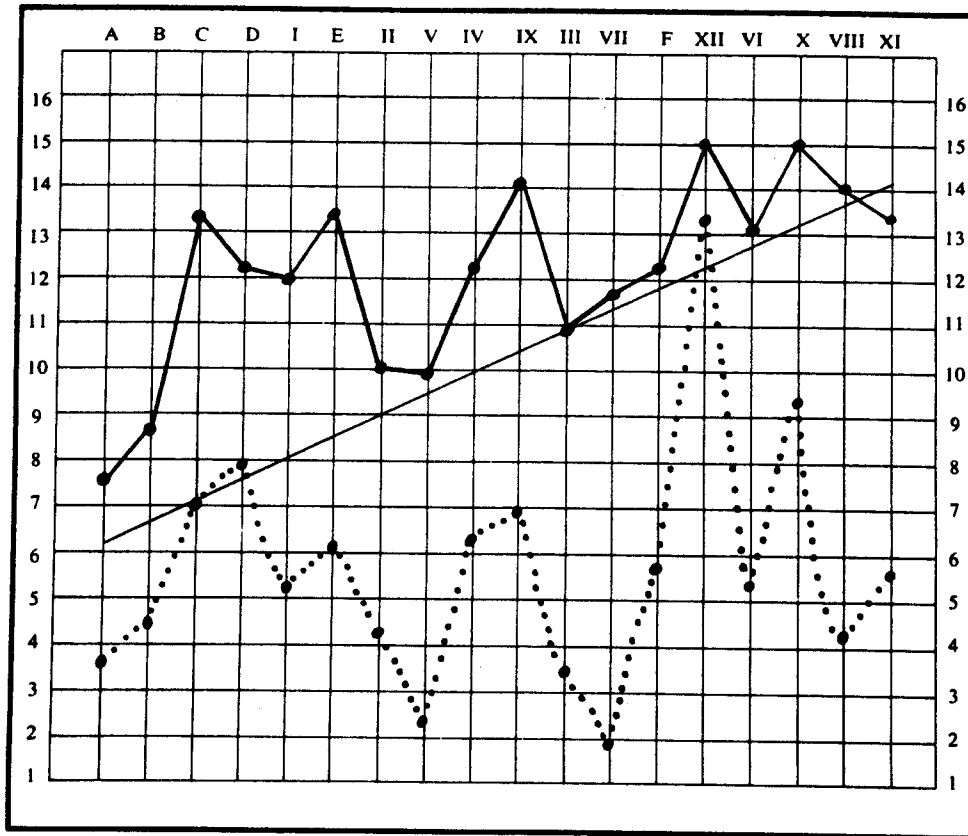


Figure 13. Bilateral Transcortical Resolving Pattern.

later the edema had cleared and the pattern was suggestive of a left hemisphere lesion.

The two final examples are clinically interesting. In the first case (Fig. 14), the patient had a meningioma that had produced hydrocephalus and had dramatically reduced communicative ability. One month after an A-V shunt was installed, the anterior temporal lobe became operational and the patient was able to process anything that involved short auditory stimuli, as shown in the dotted graph. By two months post surgery he had improved to the point where most of these functions had returned and he was left with mild problems in all modalities but a persistent mild bilateral pattern.

The final example (Fig. 15). illustrates a bilateral transcortical pattern in a patient with early Alzheimer's disease. At this point in the patient's illness, the neurologic examination and the CT scans were negative, and it was unclear whether this patient was simply having a

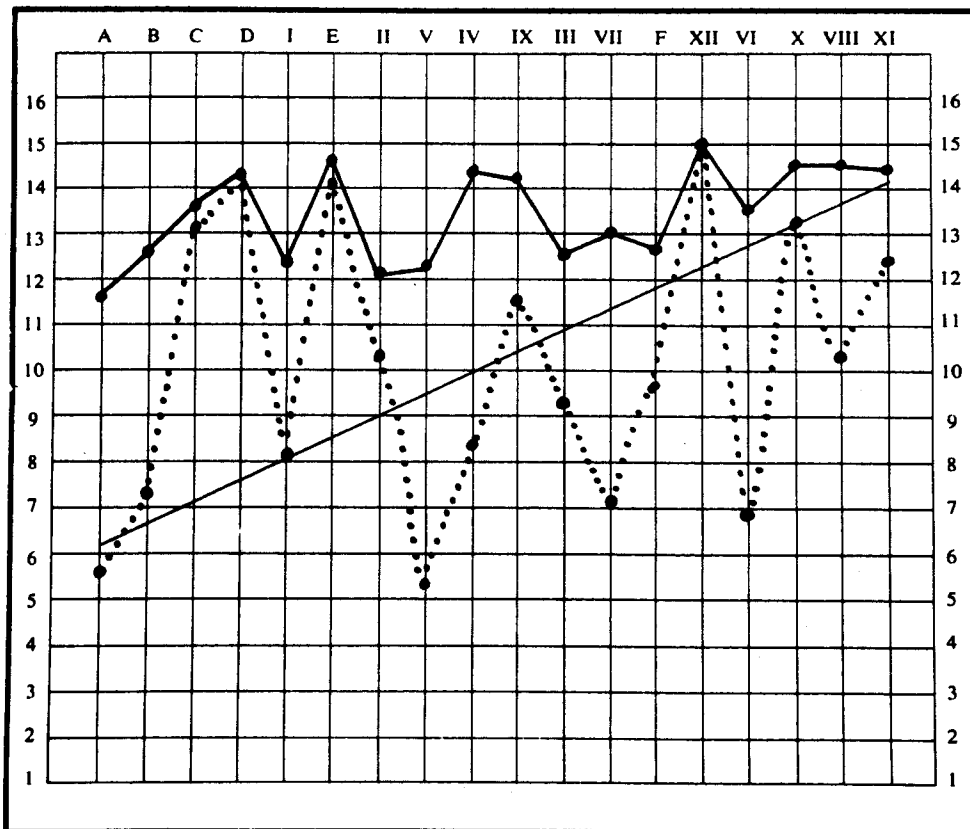


Figure 14. Diffuse Bilateral Pattern Secondary to Increased Intracranial Pressure.

psychiatric episode or if in fact he had some brain dysfunction. These tests showed clear evidence that he was having a diffuse bilateral loss of cortex. A short time later the neurologic symptoms became more prominent and CT scans subsequently verified the earlier PICA results.

In closing this discussion on localization of lesions, I want to stress some things. First, interpretations of profiles such as these are based on the assumption that the tester is a well trained experienced user of the PICA and that his scoring accuracy is at least at the 80% level in all modalities, as prescribed by our new standards of scoring competency that we have initiated last year. Secondly, it is important to remember that the localization of lesions is a joint effort involving the neurologist, neuroradiologist, neuropsychologist, and the clinical aphasiologist. Each has different approaches and insights he can bring to this very complex area. And finally, the models and hypotheses I have presented here are our initial efforts at localization with the PICA and this information must be considered as tentative. It is presented here to stimulate thought on the subject and therefore the methods should be used with discretion.

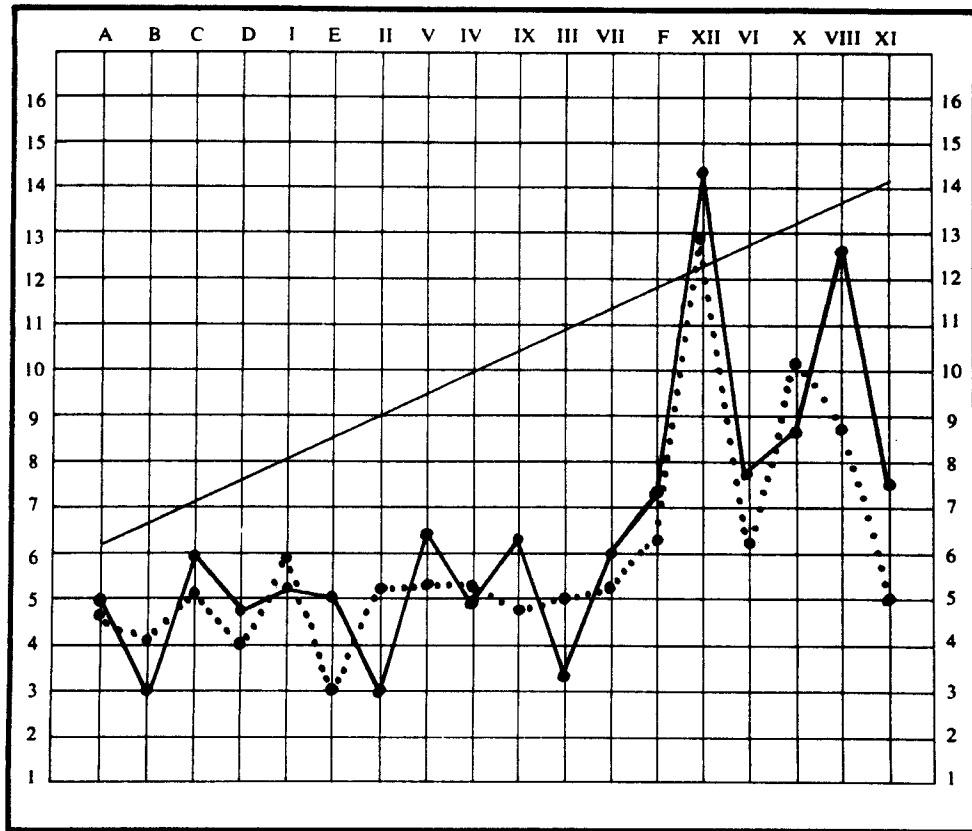


Figure 15. Bilateral Transcortical Pattern Due to Early Alzheimer's Disease.