Epileptogenic Aphasia

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Disturbances of speech and language related to epileptic seizures within the dominant cerebral hemisphere are well documented in the literature. In general, these reports deal either with children and adolescents (Landau and Kleffner, 1957; Gascon, Victor, Lombroso, and Goodglass, 1973; Shoumaker, Bennet, Bray, and Curless, 1974; De Pasquet, Gaudin, Bianchi, and De Mendelaharsu, 1976; Cooper and Ferry, 1977) or adults with histories of long-standing seizure disorders (Bingley, 1958; Alajouanine and Sabouraud, 1960; Serafetinides and Falconer, 1963; Hamilton and Mathews, 1979).

Little attention, however, has been directed toward adults with no history of seizures, who present with speech and language disturbances which may be correlated to seizure activity within the dominant temporal lobe. The purpose of this paper is to focus additional attention on seizure-related speech and language disturbances in patients with no history of seizures and acquaint aphasiologists with some of the presentations of this phenomenon.

Patients presenting seizure-related communication imapirments may be dichotomized along two dimensions. The first differentiates between two groups of patients with regard to etiology. One group presents seizures related to a focal lesion within the temporoparietal region of the dominant hemisphere. This group includes patients showing post-infarction epilepsy or seizures resulting from cortical irritation by a neoplasm. A second group shows no such focal lesion. Rather the epileptiform discharges are attributed to some nonspecific cause. The second factor by which patients may be differentiated is the nature of their pre-seizure communicative abilities. The patients most frequently encountered by clinical aphasiologists are those who present a baseline aphasic impairment with seizure-related exacerbations. The nature of these exacerbations is highly variable, ranging from momentary fluctuations in attention to profound communicative impairments. Conversely, some patients exhibit no communicative impairment prior to the onset of a seizure. Furthermore, in some of these patients, aphasia is the only clinical feature observed. This latter group of patients who have no demonstrable speech and language impairment during the inter-ictal period may be said to present "epileptogenic aphasia."

Four patients will be discussed in this report. The first displayed aphasia secondary to a CVA with seizure-related exacerbations of her communicative impairment. The remaining three patients exhibited epileptogenic aphasia. One of these patients had a left parietal glioblastoma, while the other two showed no specific cortical lesion. None of the three patients showed any communicative impairment prior to the onset of seizure activity, and all three responded favorably to anticonvulsant medication. Discussion of the patients' aphasic impairments and correlated EEG recordings follow:

Patient 1. This patient was a 62-year-old female who experienced a left temporoparietal CVA in July, 1978. Her past medical history included a previous left CVA in 1976 with a mild residual right hemiparesis and communication impairment. On admission, EEG revealed left hemisphere status epilepticus which was treated with Dilantin and later sodium valporate.

Initial speech and language evaluation revealed a moderate-severe impairment of auditory comprehension with the patient able to follow approximately 50% of simple commands. Oral expression was marked by word-finding difficulty and considerable perseveration. Spontaneous verbal output was generally intelligible and meaningful, but inappropriate. A treatment program focusing on improving the patient's auditory comprehension and verbal expression was initiated, which included activities such as pointing to an object named, from a response field of five, following single stage commands related to body parts (e.g. raise your hand, shrug your shoulders), and confrontation naming of common objects.

Although the patient's performance in treatment fluctuated significantly and she frequently required extensive cueing and/or modeling in order to respond accurately, she consistently attempted to respond in an appropriate manner. However, throughout the first five weeks of treatment, she continued to experience occasional focal seizures. These episodes lasted from one to two minutes during which time the patient remained alert and maintained eye contact, but failed to acknowledge any auditorily-presented verbal stimuli and was unable to respond to treatment activities in an appropriate manner. Verbal output was limited to undifferentiated mumbling, and no meaningful gestural expression could be elicited. Immediately upon the cessation of seizure activity, the patient was able to resume active participation in treatment activities.

Patient 2. This patient was a 54-year-old male who was admitted to the George Washington University Medical Center (GWUMC) following a seizure. Past medical history included a left parietal glioblastoma which had been biopsied and irradiated approximately three months earlier. The patient's speech and language were reported to be normal prior to this episode.

An initial speech and language evaluation was conducted prior to the achievement of adequate blood levels of Dilantin. This evaluation revealed a severe Wernicke's aphasia (Figure 1) with profound impairment of auditory comprehension for single words and simple commands. Oral expression was marked by a moderately severe impairment. Confrontation naming elicited

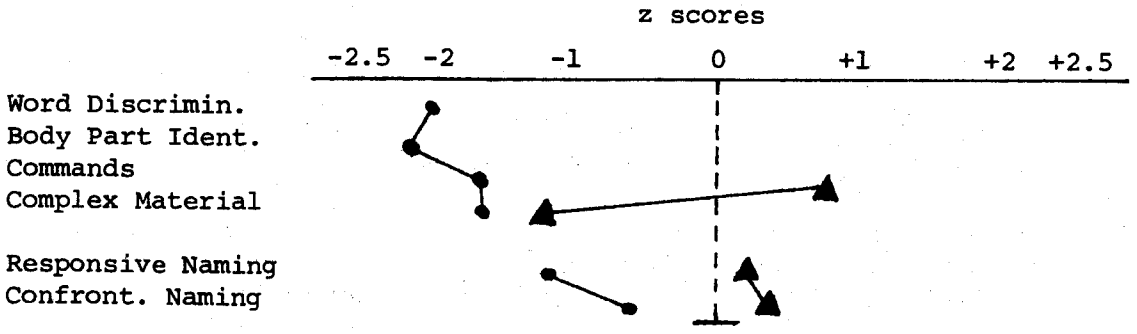

Figure 1. Language profiles of Patient 2. The circles show the patient's performance on selected language tasks prior to the achievement of an adequate Dilantin blood level. The triangles show a partial resolution of the patient's language deficit following the attainment of a therapeutic Dilantin blood level.

numerous neologisms, verbal paraphasias, and perseverative responses, and spontaneous expression consisted of fluent, English jargon. An EEG performed on the same day as this evaluation revealed epileptiform discharges over the left posterior temporal region (Figure 2).

A reevaluation (Figure 1) was administered the next day, after the patient's Dilantin blood level had reached an adequate therapeutic level (20 µgm/ml). At this time, he presented a mild-moderate Wernicke's aphasia. Auditory comprehension of sentences containing 5 or more elements was mildly impaired. Confrontation naming was marked by increased response latency and only occasional verbal paraphasic errors. Spontaneous expression was functional, but somewhat reduced in efficiency. Following the second evaluation, the patient's speech and language performance continued to improve, and he left the hospital with no apparent communication impairment.

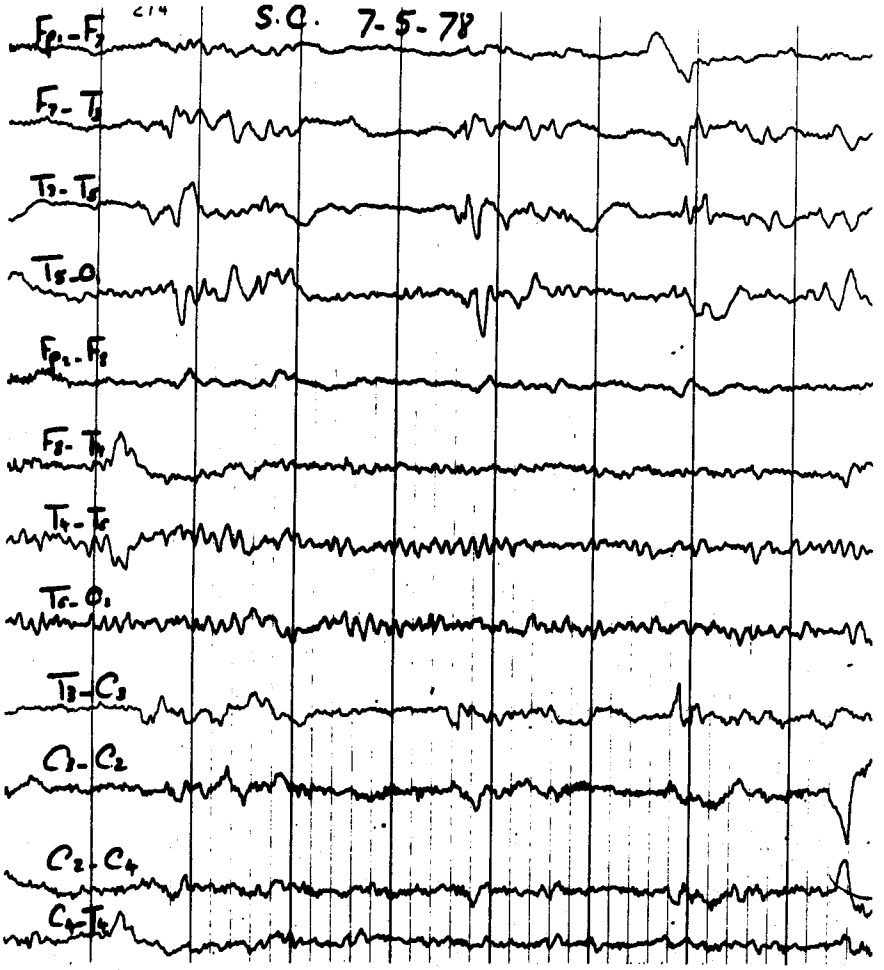

Figure 2. EEG of Patient 2. High amplitude slow spike-wave discharges arising from the left posterior temporal region may be seen on channels two, three and four.

Patient 3. The third patient was a 58-year-old woman who was admitted to GWUMC following an episode of transient aphasia. An EEG performed on admission revealed some slowing over the left posterior temporal region. CT scan revealed some diffuse cortical atrophy, but no discrete lesion. She reported no previous seizures.

The patient was referred for a speech and language evaluation prior to the initiation of treatment with anticonvulsant medication. Initially, the patient showed no speech and language impairment. She conversed

appropriately with the examiner, giving specific answers to questions and relating incidents in a fluent, grammatical manner. After approximately 10 minutes, however, the patient abruptly began producing fluent English jargon and neologistic spelling. Auditory comprehension was severely impaired. The patient appeared to be alert and had no other clinical features of seizure activity. This episode lasted approximately 4 minutes, after which her language production gradually returned to normal.

Subsequent episodes of aphasia were observed during EEG recording and correlated with high amplitude slow spike waves originating over the left posterior temporal region and spreading to other temporal placements ipsilaterally. Figure 3 shows the EEG patterns before and during an episode of aphasia. The seizures ended abruptly at which time the patient's communicative abilities improved dramatically. In the immediate post-ictal

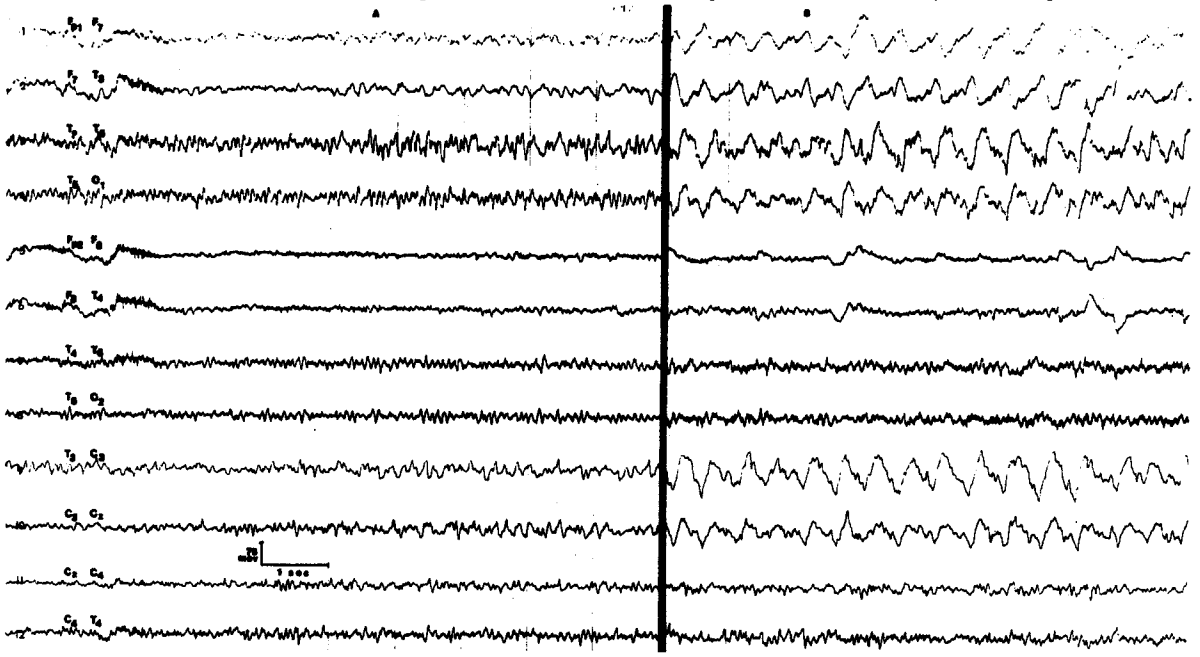

Figure 3. EEG of Patient 3. The left side of the figure(A) shows the earliest evidence of recruitment emerging from the alpha activity over the left posterior temporal area (channels three and four). The right side, (B) is taken 3 1/2 min later, revealing rhythmic high amplitude, slow spike-wave discharges over the left temporal region (channels 1-4, 9, 10). Normal alpha activity over the right posterior quadrant (channel 8).

period, the patient presented a mild aphasia marked by slightly reduced auditory and reading comprehension for longer, more complex units and occasional literal and verbal paraphasias in oral and graphic expression (Figure 4). Following the institution of treatment with anticonvulsant medication (Dilantin), no further seizures were observed, and the patient was discharged with no apparent communication impairment.

Patient 4. This patient was a 57-year-old female who was transferred to GWUMC from an alcoholic treatment center following the abrupt onset of severe communication difficulty. A history of a generalized seizure disorder was later obtained, but the patient was reported to have been free of seizures for at least 2 years prior to admission and had been taking no anticonvulsant medication. EEG revealed epileptiform discharges with a left temporal focus which were markedly attenuated with an intravenous injection if Diazepam (Figure 5). Anticonvulsant treatment was then initiated.

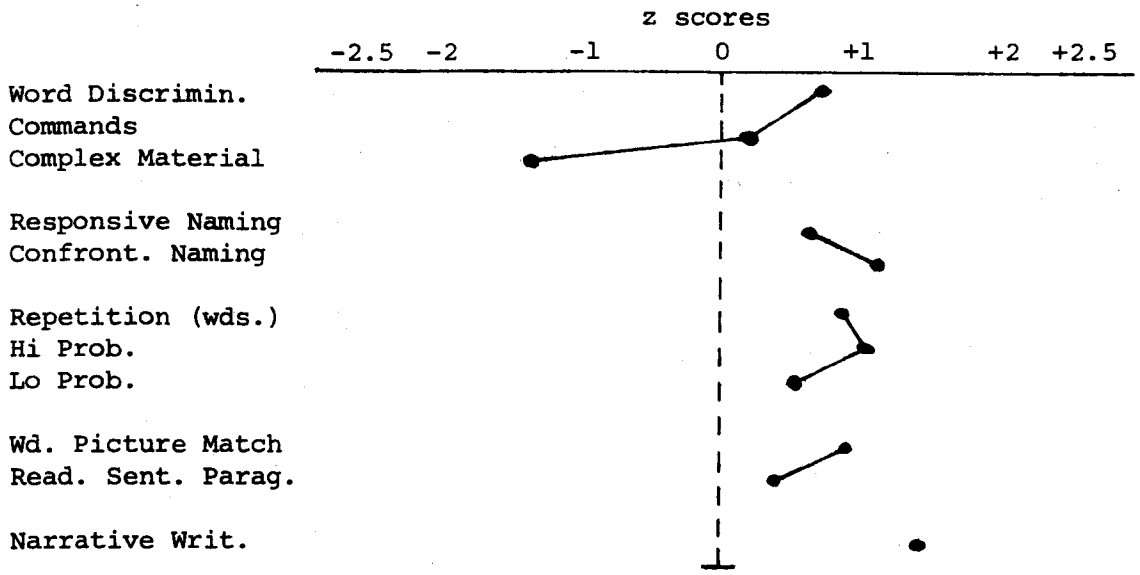

Figure 4. Language profile of Patient 3. Scores on selected tasks show the patient's language performance to be only slightly impaired following the remission of seizure activity.

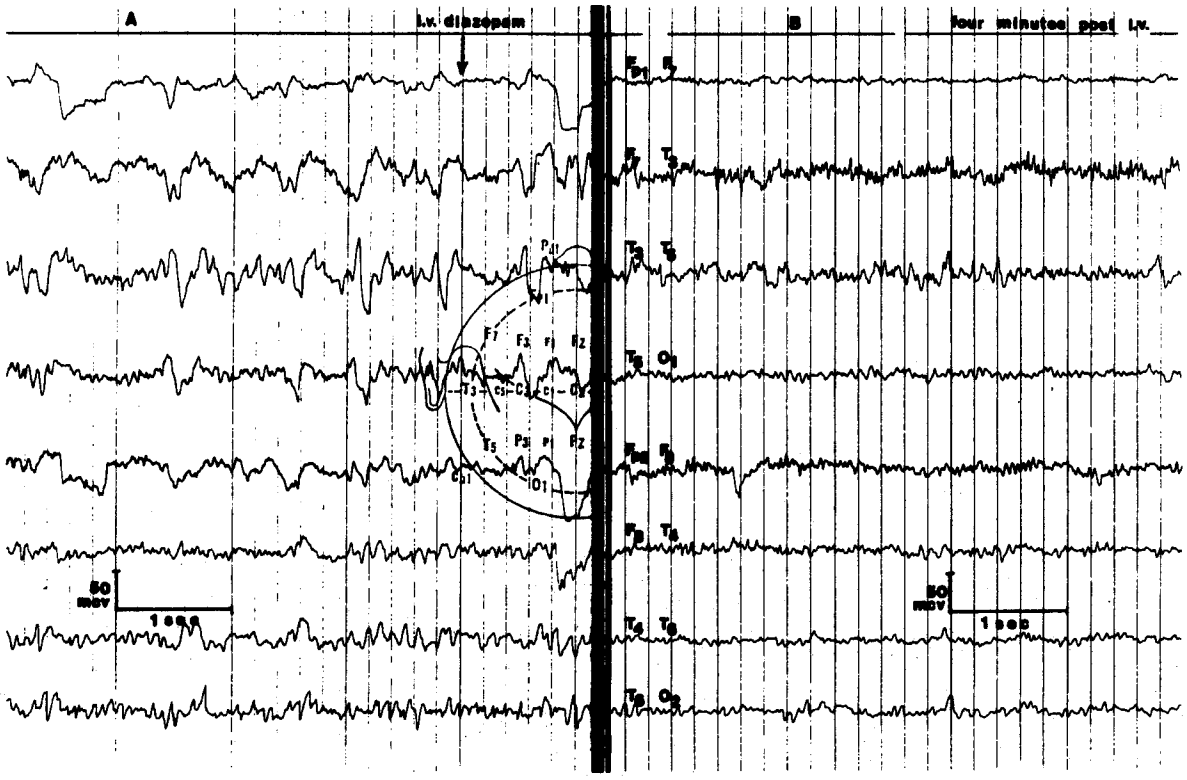

Figure 5. EEG of Patient 4. The left side of the figure (A) shows some of the recurrent sharp wave activity and associated rhythmic slowing over the left temporal region (channels 2 and 3). The arrow at the top indicates the time of injection of I.V. Diazepam. The right side of the figure (B), taken 4 min later, shows attenuation of the sharp wave activity.

On initial speech and language evaluation administered prior to attaining an adequate Dilantin blood level, the patient was observed to be having a focal motor seizure. Her head and eyes were deviated to the

right and her right arm was tonically flexed. She was unresponsive to any auditory verbal stimuli. Oral expression was limited to the automatized repetition of "nine-thirty, nine-thirty." The patient was next seen approximately 3 hours later. At that time she was markedly echolalic and comprehended a few simple commands. She was able to read some short sentences aloud, but with no comprehension. Response latency was significantly increased.

The following day the patient was markedly improved. Her Dilantin blood level had reached a minimum therapeutic level (14 μgm/ml) and EEG revealed an attenuation of the epileptiform discharges. At this time the patient was very talkative, exhibiting much extraneous verbalization; some echolalia persisted. She was able to follow approximately 90% of simple commands, making only occasional within-class errors. Confrontation naming was unimpaired, with the patient often providing unsolicited information. She appeared to be unaware of her excess and inappropriate verbalization. The following day the patient was discharged. At that time her Dilantin blood level had reached an adequate therapeutic level, and she presented only a minimal communication deficit (Figure 6). Auditory retention was mildly reduced, and auditory and reading comprehension were intact except for long, ideationally complex material. Conversational speech consisted of syntactically well-formed utterances produced in a fluent, well-articulated manner with only an occasional verbal paraphasia. Narrative writing was marked by spelling errors and the omission of function words.

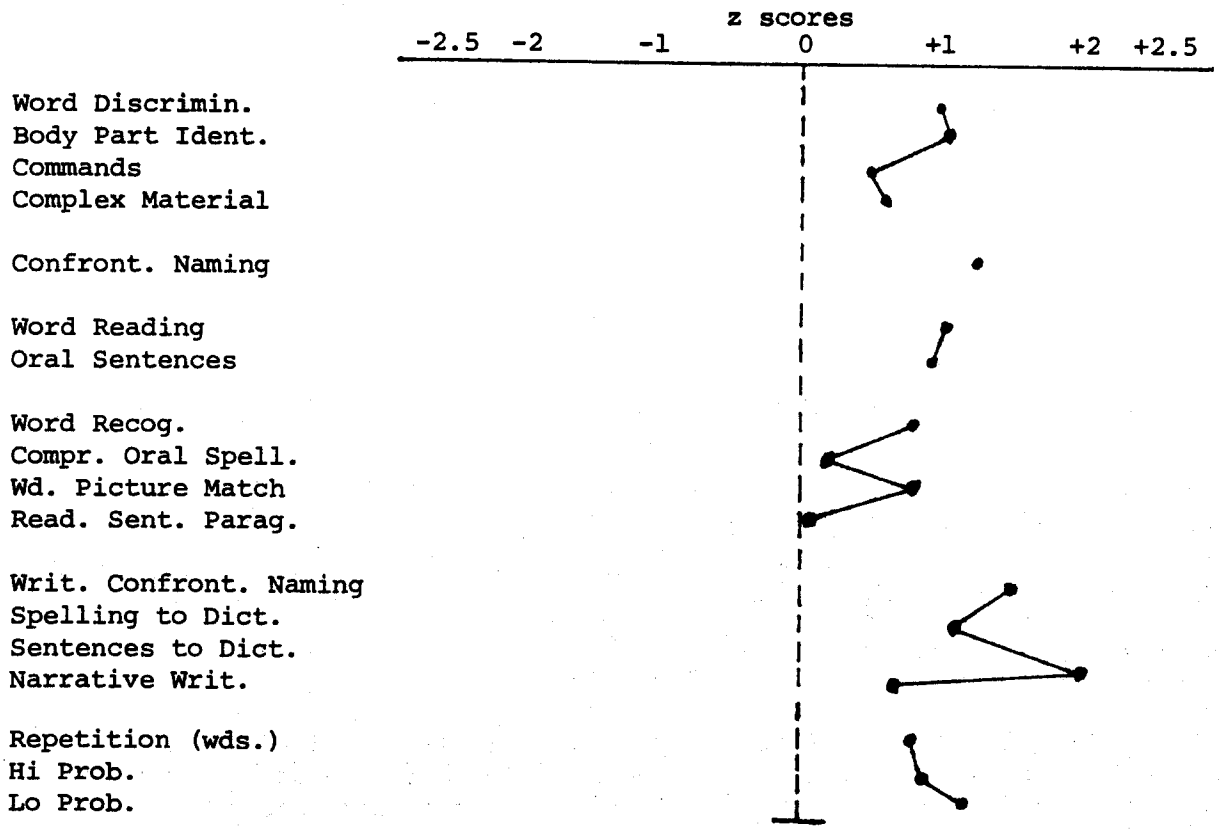

Figure 6. Language profile of Patient 4. The patient's scores on selected tasks show her language performance to be essentially unimpaired following the attainment of an adequate Dilantin blood level.

Implications

Historically, interest in seizure-related speech and language disturbances has been focused on individuals with histories of long-standing seizure disorders. Three of the patients discussed above had no previous history of seizures, and the one who did had been seizure-free for at least two years. In addition, three of the patients had no communication impairment prior to the onset of seizures, and two had no specific cortical lesion which might account for any speech and language disturbance.

These findings have significant implications both diagnostically and therapeutically. From a diagnostic standpoint, epileptogenic aphasia must be considered in a differential diagnosis of any communicative impairment which is of abrupt onset and is frequently fluctuating, particularly in the absence of a specific cortical lesion or other clinical signs of cortical dysfunction. It may be suggested that epileptogenic aphasia may be readily misdiagnosed as transient ischemic attacks or as psychotic reactions. With regard to treatment, clinicians must be sensitive not only to the seizure-related exacerbations of an aphasic impairment in patients with a known cortical lesion, but also to the variations in the communicative abilities of patients with epileptogenic aphasia. Careful monitoring of speech and language performance can also be employed, along with EEG, as a valuable tool in determining the effectiveness of anticonvulsant medications and the extent to which a patient's communicative impairment is seizure-related. The presence of seizure-related speech and language disturbances in patients with no history of seizure disorder is a phenomenon of great clinical significance and one that calls for further study.

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Q: The results and discussions presented in Clinical Presentations I, including right hemisphere damaged patients' PICA profiles and epileptogenic aphasia, may lead us to ask ourselves again what is aphasia. Ann, were your subjects really aphasic patients?

A: I think the purpose of our study was to bring to peoples' awareness that aphasic symptoms or behaviors that one sees may be caused by temporal lobe seizures. I don't think that we are out to name a new kind of aphasia. What we are trying to convey is that the cause of the aphasia was in fact temporal lobe seizures and not a T.I.A. or tumor or something of that nature. It was more the etiology rather than a different kind of aphasia. What we were seeing were some aphasic behaviors due to temporal lobe seizures.

Q: What is the typical length of recovery following the seizure?

A: That is highly variable, we found. Some patients took a couple days to get their anti-convulsant blood levels to a therapeutic level. In some it was dramatic and in a matter of minutes, the change occurred. So, I think it is highly variable. That is why I think speech and language documentation is so necessary when these seizure medications are given, to find out whether speech-language behaviors have reached a normal level or the best possible level that can be attained.

Q: Upon time of discharge from the hospital, did any of these people have residual speech-language problems?

A: Not as far as we know.

Q: Did you have the opportunity to see any of them in treatment?

A: No, we did not see any for treatment. However, the first patient that I described who did have aphasia because of a previous C.V.A., of course, her aphasia remained. She was in treatment at the time and continued, but the rest were not seen because they became so normal.

Q: Ann, do you know how those patients were performing on other than language?

A: No. No other tests, visual tests, or anything, were administered.

Comment: They had no other signs of clinical pathology. The one lady initially presented with a tendency but the others didn't.

Q: But you don't know how they would do on putting a puzzle together, or doing a coding test or anything like that?

A: At this particular point, no, becuase the neurologists were so hot on getting them fixed up and out of the hospital.

Q: I'm puzzled, too, and I guess I am asking the same kind of question Peg was asking...how can you talk about it as being aphasia if it is not differentiated from the rest of the behaviors and the proportion of the rest of the behaviors? But that's sort of trivial, maybe. I don't know. I guess I had thought what you usually saw in seizure activity was verbal automatisms. If I remember some of the research that has been recorded in England, but I can't remember now who did it. But I guess this is different?

A: I think the one patient who was repeated "9:30, 9:30" showed some of the automatisms, however, as her anti-convulsant blood level was increased, she broke out of that and was displaying more dysphasic-like behavior, like echolalia and comprehension deficits. She became more truly aphasic-like rather than having just the automatisms. The other patients---expecially the two I worked with closely---showed some repetitiveness of what they said, but it was too varied, I feel, to really call it automatisms.

Comment: The classical paper on epilepsy and aphasia was written by Serafetinides and Faleoner in England, as Dr. Darley suggested. They brought attention to the high incidence of automatisms and speech arrest; this is well known by most neurologists as a form of focal epilepsy.

It is very difficult to examine patients at length during seizure, because most of them last for a few minutes if not seconds. Everyone of us have seen patients like this except we have not been able to subject them to a series of needed tests as Dr. Darley suggested. It is very unlikely that you will be able to put them through a complete battery during a focal epileptic seizure. So this answers that point.

The other one is the unusual nature of fluent English jargon or neologistic jargon, as this would be. I think this is what makes your presentation interesting, because most of the classical symptoms that occur with focal seizure are mutism, speech arrest, repetition and perseveration, as was the case in Sarafetinides and Faleoner.

Q: I have a question for Dr. Kertesz, which is a continuation of what he said. How variable can an automatic comment be?

A: That's a good question and I'm not sure I can answer it. I would think that the repetitive kind of behavior could be classified under automatisms. That's a good observation. I just don't know myself. I haven't seen any fluent jargon during epileptic seizures. In my experience, the variability of automatisms is limited. One usually sees meaningless utterances or sometimes a single word is being used. So, I think the variability is relatively low. In this particular case, I thought the variability was more in keeping with jargon speech. Moreover, the recovery from postictal speech disturbance does depend a great deal on the nature of the lesion. If they have a glioma or a tumor it is likely that there is a longer postictal defect. If they have a scar that is an old stroke and following that they have seizures they usually recover much faster. So I think the nature of the lesion is a major variable.

Q: Ann, did you have an occasion to examine the patients when they were no longer on the seizure medication and, if so, how did their speech compare to the premedication phase?

A: No, I did not. One reason is that neurologists do not want to take patients off seizure medication because of potential reoccurrence. One of our patients with the glioma was put on dilantin prophylactically. He stopped taking the medication and when he came in with the seizures and aphasia his blood levels of anti-convulsants were practically nonexistent. Prophylactically he was on anti-convulsants and having stopped suddenly he demonstrated an onset of epileptogenic aphasia.