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Ictal Speech Disturbance and Cerebral Dominance.

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Ictal Speech Disturbance and Cerebral Dominance

Itsuo KAWAI and Hiroshi OHASHI

I INTRODUCTION

Ictal speech disturbance is treated as a symptom of epilepsy and may be divided roughly into aphasic fit, speech arrest and speech automatism.

An aphasic fit is defined as a paroxysmal disorder of expression, and also of comprehension of language. A speech arrest is an ictal epileptic manifestation during which the subject is unable to utter a single word, although his "internal language" is completely intact. Neither disturbance brings clouding of consciousness and both are retained in the patient's memory. These definitions, as references, are originating from the articles of Penfield17), Bingley4), Gastaut and coworkers8)9). Serafetinides and Falconer18) define speech automatism as utterance of identifiable words or phrases which are linguistically correct but for which the patient is subsequently amnestic.

The problem of the relationship of these ictal speech disturbances to lesions or cerebral dominance has been examined by many researchers. All authors are of the same opinion with respect to the fact that aphasic fits occur during temporal lobe seizures and are associated with epileptogenic lesions of left hemisphere in cases with right-handedness. However, the exact percentage is a matter or controversy. There are also differences of opinion concerning the problem of cerebral dominance for speech arrest and speech automatism.

The authors, therefore, collected 43 cases with ictal speech disturbances during the past 8 years and examined the problems as follows:

Three types of ictal speech disturbances and their relation to the laterality of the focus of cerebral disorder.

II MATERIAL AND METHOD

About 43 cases with these symptoms, the interictal EEG were examined at least twice in every case and the laterality of abnormal findings was confirmed. The criteria of laterality utilized was as follows: spike or sharp wave focus, unilateral dominant spike or sharp wave, unilateral slow wave corresponding with brain damage or tumor in same region.

Laterality was thus confirmed in 36 cases of the total number studied. Ad-
ditional 7 cases which showed independent foci or no lateralities or normal findings were excluded.

The determination of handedness was made according to the articles of Bing-ley and also of Hécaen et Angelerque. It should be noted that many left-handed people in Japan are right-handed in using pens and chopsticks.

Two hundred and forty three right-handed patients showed unilateral abnormal EEG findings during 8 years past. In these cases, 136 were of abnormalities on the left and 117 cases showed abnormalities on the right.

On the basis of these control numbers, quantitative relations to cerebral dominance in right-handed individuals were examined statistically by t test. Two left-handed individuals out of 36 patients could not be statistically tested because of the small number. These 2 cases were not included in this report.

III Results

Table I presents the relevance of ictal speech disturbances to the lateralities of foci of cerebral disturbance.

On the basis of this evidence, the incidence of aphasic fits in left hemisphere is statistically significant (P<0.01).

The incidence of speech automatism in the right hemisphere is also significant (P<0.05).

The laterality with regard to speech arrest is not significant.

In addition the authors studied the ratio of combination within three types. They did not found any case combined with aphasic fit and speech automatism. There were two cases combined with speech automatism and speech arrest of

<table>
<thead>
<tr>
<th>Side of epileptic dysfunction</th>
<th>Left</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aphasic fit</td>
<td>13</td>
<td>0</td>
</tr>
<tr>
<td>Speech arrest</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Speech automatism</td>
<td>3</td>
<td>10</td>
</tr>
</tbody>
</table>

Table II.

<table>
<thead>
<tr>
<th>Speech disturbances</th>
<th>Aphasic fit</th>
<th>Speech arrest</th>
<th>Speech automatism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ant. temporal</td>
<td>7</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>Mid. temporal</td>
<td>3</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>Post. temporal</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Central</td>
<td>0</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Front-central</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Centro-occipital</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Unilateral hemisphere</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Summation</td>
<td>13</td>
<td>8</td>
<td>13</td>
</tr>
</tbody>
</table>
which one was left abnormal and the other was right abnormal by EEG. It is difficult to confirm whether the combined form with aphasic fit and speech arrest exists or not.

Three types with ictal speech disturbances in relation to EEG localization were presented in Table 2. The incidence of temporal foci is most frequent in aphasic fits (sensory aphasia is represented in left postero-temporal region in two cases and in left anterotemporal region in one case). It is noteworthy that 4 cases with speech arrest are represented in central region.

The lesions were clearly observed in 6 cases which suffered from head injury, meningioma or vascular lesion by history. In these 6 cases are included 3 cases with aphasic fits with lesions in the left temporal region, 2 cases with speech arrest with the lesions in the central region and one case with speech automatism of which the lesion was in the temporal region.

IV Discussion

The present results are in complete accordance with previous studies in so far as aphasic fits occur with a marked left hemisphere dominance in right-handed patients. However, Hécaen et al do not discriminate between aphasic fit and speech arrest. In the latter cases, right lesions are also frequently found. Only Bingley treated this problem statistically. The present study also reveals significant left hemisphere dominance in right-handed aphasics.

Alajouanine et al reported that seizure-patterns appeared on left side in 4 cases with aphasic fits. Penfield and his coworkers assumed that aphasic fits occurred in relationship to left temporal seizure-patterns by cerebral stimulation. Bilateral seizure-patterns with aphasic fits were noted in the other reports.

Ajmone Marsan and coworkers reported that there were certain cases in which the scalp EEG did not change during the aphasic fit while seizure-patterns did appear in the records when depth electrodes were utilized.

It seems that the side of focus is not relevant to the incidence of speech arrest in right-handed individuals as Penfield said though there are a few reports which state that left hemisphere dominance is necessary for this symptom. Penfield and coworkers found that speech arrest is caused by a neural discharge involving the inferior Rolandic area or the supplementary motor area of either hemisphere. The present study revealed that most of the cases showed a central spike focus. One of these was a patient who suffered from a meningioma in the supplementary motor area.

In this study the relationship between the symptoms and the cerebral regions showing paroxysmal dysrhythmia was investigated. Both neuropsychologically and EEG findings were consistent with aphasic fit and speech arrest. A careful analysis, however, of EEG findings tended to show an anterotemporal focus in those cases with any kind of aphasic fits. There is an interesting report
from Brain\textsuperscript{5} that EEG findings vary from the anterotemporal focus to postero-temporal focus depending on expressive or comprehensive aphasic reciprocity.

There exist completely different reports as to speech automatism: two reports\textsuperscript{31,4} insist that it is related to left hemisphere dominance, while two other reports\textsuperscript{41,59} suggest that right hemisphere dominance is necessary. In this study the phenomenon occurred significantly more with right hemisphere foci in right-handed patients.

Driver and his coworkers reported a remarkable case with speech automatism\textsuperscript{7}. Their patient suffered from temporal lobe epilepsy with speech automatism reproduced by electrical stimulation of the right amygdalar area after surgical exposure of the cortex. At the same time, right temporal sharp activity was recognized. The patient had a single attack with speech automatism although the postoperative course was uneventful. The authors suspected from Jackson's theory\textsuperscript{13}, that this phenomenon related the function of dominant hemisphere released by the dysfunction in the minor hemisphere.

Speech automatism seem to be provoked when the pathological excitations do not spread to speech areas in the confusional state.

The question of why there are different opinions as to speech automatism and cerebral dominance is an interesting one. First, ictal paraphasia or Jargon aphasia has been occasionally mistaken for speech automatism (all ictal paraphasia present in the dominant hemisphere according to Hécaen\textsuperscript{10}). Second, it may be that some workers have not discriminated between simple vocalization and this phenomenon. Third, automatism does not always appear in temporal lobe epilepsy. It seems possible that speech automatism springs from other regions and occasionally leftward regions distant from the temporal lobe.

For this third reason, the ratio of right hemisphere dominance with regard to speech automatism was not found to be particularly high in the present study.

V Summary

Ictal speech disturbances in right-handed 34 epileptics in relation to cerebral dominance were examined.

1) The incidence of aphasic fit in left hemisphere is statistically significant (P<0.01).
2) The incidence of speech automatism in right hemisphere is also significant (P<0.05).
3) The laterality with regard to speech arrest is not significant.
4) There were no cases combined with aphasic fit and speech automatism.
5) Most cases with aphasic fits were among those in which temporal foci were found. Some cases with speech arrest were among those in which a central foci were found.
6) The symptomatology and physiological mechanisms of speech automatism
were discussed.

REFERENCES


(Aug. 31, 1975, received)