■Special Lecture

Cortical Stimulation and Language

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INTRODUCTION

Since the pioneering studies of Penfield and collaborators (Penfield and Rasmussen 1949, 1950 ; Penfield and Jasper 1954 ; Penfield and Roberts 1959) cortical stimulation has become a widely accepted method for mapping of cortical language areas (Fedio and Van Buren, 1974 ; Ojemann, 1978, 1979 ; Lesser et al., 1984, 1986). From cortical stimulation evidence, Penfield and Roberts (1959) described three language areas which they labelled anterior, posterior and superior language areas. They correspond approximately to Broca's area, Wernicke's area and the supplementary motor area. In this paper we are going to summarize some of the findings related to cortical language areas we observed during stimulation of the human cortex in patients who were candidates for epilesy surgery.

MECHANISMS FOR SPEECH ARREST ELICITED BY CORTICAL STIMULATION

Speech arrest during electrical stimulation of the cortex can be produced by the following five mechanisms.

1. Speech arrest due to stimulation of a positive motor area.

Stimulation of the primary motor area 4 can produce muscle contraction of muscles that participate in the production of speech as for example the tongue, lips, etc. Under these circumstances the patient very frequently has speech difficulties which may range from slowing and distortion of speech to complete speech arrest.

2. Speech arrest due to stimulation of a negative motor area.

Stimulation of the inferior frontal gyrus immediately in front of the primary motor area of the face (corresponding to Broca's area in the dominant hemispere) and of the supplementary motor area produces a negative motor response. (Lueders et al., 1983 ; Lüders et al., 1989a). A negative motor response is defined as inability to produce or to sustain a voluntary motor contraction during cortical stimulation at a stimulation intensity which otherwise produces no effect. Negative motor responeses can be elicited from both (dominant and non-dominant) inferior frontal gyri and also both supplementary motor areas. Stimulation of a negative motor area frequently produces complete speech arrest but when using lower stimulus intensities or in certain cortical areas even at the highest stimulus intensity

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only slowing of speech with distortion of word pronunciation may occur. Stimulation of a negative motor area produces, however, no actual language disturbance. The patient knows exactly "What he is going to say and how he is going to say it" but when he tries to speak he is unable to do so. In addition, there is no difficulty of comprehension of verbal material.

3. Speech arrest due to stimulation of cortex that elicits other non-motor positive symptoms.

Speech slowing, interruption or even speech arrest may be secondary to experiencing some other symptoms elicited by the cortical stimulation which distracts the patient. For example, cortical stimulation may produce vivid visual or auditory hallucinations which attract the full attention of the patient or even produce a strong secondary emotional response. This may produce speech disturbances that are unrelated to an alteration of a language area due to cortical stimulation. The presence of such positive symptoms can be detected easily by just asking the patient to report any signs or symptoms when stimulating the same cortical area at an identical stimulus intensity but without having the patient perform any speech or language task. In addition, it is frequently possible to get the patient to perform the speech related task appropriately if the stimulus is applied repeatedly and the patient asked to neglect the positive symptoms elicited by the stimulation (the vivid hallucinations in the example given above) and to make special efforts to concentrate on the speech task. This last maneuver allows the observer to exclude the possibility that stimulation of a given cortical area produces both, namely a language disturbance and also another positive symptom.

4. Speech arrest due to loss or alteration of consciousness during the cortical stimulation.

Cortical stimulation may produce complex partial seizures with different degrees of alteration of consciousness. During these episodes alteration of speech and frequently a total and prolonged speech arrest occurs. Monitoring of the EEG while stimulating is helpful to detect the occurrence of epileptiform discharges which are usually seen in patients who experience alteration of consciousness while stimulating. It is important to stress here, however, that afterdischarges are usually asymptomatic (even when stimulating an eloquent area of the cortex) unless they spread from the stimulating electrode to numerous adjacent electrodes and therefore engage a more extensive cortical area in the epileptogenic process. It is best to only use stimulation intensities that are subthreshold for epileptogenic discharges (including afterdischarges limited to the stimulated electrode) to avoid confusion between symptoms elicited by the spread of the epileptogenic process and symptoms due to electrical interference at the stimulated electrode. We have also observed isolated cases in which stimulation produced a short alteration of consciousness which did not outlast the duration of the stimulus and was also not associated with any afterdischarges once the stimulus was discontinued (no clear analysis of the EEG is possible during the electrical stimulation due to the stimulus artefact). In these cases the patient also had a complete speech arrest, but the fact that he was totally amnestic for all events happening during the stimulus indicated tht he most probably suffered a short alteration or loss of con-

sciousness. In addition, during these episodes the patients are also unable to perform repetitive motor movement such as pushing a button. This indicates that the interference of function is certainly not limited to verbal processes and therefore that the cortex stimulated is not a specific language area.

5. Speech arrest due stimulation of a language area.

To establish that a speech arrest is due to stimulation of cortex selectively involved in the processing of verbal material we have to exclude all other four possibilities for speech arrest mentioned above. In other words, during the stimulation the patient should have no positive symptoms (motor or non-motor), there should be no negative motor response, and he should be able to perform without difficulty non-verbal tasks. In previous studies all these different causes for speech arrest were not always taken in to account before assuming that the speech arrest was produced because of interference with a cortical language area. It is possible, therefore, that in some cortical regions in which speech arrest occurred because of interference with function other than those specifically related to language.

CORTICAL LANGUAGE AREAS

In the following few paragraphs we are going to discuss the three language areas described by Penfield and Robertson (1959) and also the basal temporal language area which has been identified lately by electrical stimulation studies.

1. Superior language area or supplementary motor area

Dinner et al. (1987) found that stimulation of the supplementary motor area produced speech arrest in 5 out of 6 cases. In all cases, however, the speech arrest was due to a negative motor response involving muscles that participate in speech. Receptive language disturbance were not detected but no systematic testing was carried out. These results suggest that the supplementary motor area is most probably important for the performance of voluntary movements (including those necessary for the generation of speech) (Lüders et al., 1989 a) but cast some doubts about whether it is actually a specific language area. In the studies of Penfield and Robertson (1959) no systematic testing was performed to exclude the possibility of a negative motor response as the cause of the frequent speech arrest they observed in the supplementary motor area.

2. Anterior language area or Broca's area

It is interesting to notice that stimulation of the inferior frontal gyrus very frequently also produced a striking negative motor response involving not only muscles that participate in speech but also extensive other somatotopic regions. There were, however, isolated electrodes in Broca's area which produced speech arrest which could not be explained by a non-specific negative motor effect. In addition, some of the language electrodes in Broca's area also produced a significant receptive language deficit similar to the results obtained when stimulating the posterior language area (Lüders et al., 1986a) These findings seem to suggest that Broca's area, similar to the Supplementary Motor Area, is a cortical center for organization of voluntary motor movement. Broca's area, however, has in addition a specific language function. The predominantly expressive character of the aphasia produced by lesions of Broca's area may well be related to the significant nega-

tive motor response which can be elicited by stimulation of that region.

3. Posterior language area or Wernicke's area

Stimulation of Wernicke's area produced speech arrest which consistently was due to a selective language impairment. Usually a mixture of receptive and expressive aphasia was produced by stimulation in that area. No negative responses occur in that region. In other words, Wernicke's area is exclusively a language center and most probably does not participate in the organization of voluntary movements.

4. Inferior language area or basal temporal language area

Recent studies have demonstrated that stimulation of the basal temporal region, particularly the fusiform gyrus, produces speech arrest which, the same as the speech arrest in Wernicke's area, is due to a specific language deficit (Lüders et al., 1986b, Lüders et al., 1989 b). In other words, stimulation produces no negative motor response and the language deficit elicited by stimulation tends to consist of a mixture of comprehension related and expressive verbal deficits.

TYPE OF LANGUAGE DEFICIT ELICTTED BY CORTICAL STIMULATION

Stimulation of a language area does not elicit a positive effect (namely speech) but always a negative effect. At many electrodes the effect of cortical stimulation is quite dramatic with complete speech arrest and a total receptive aphasia. In other words, in spite of the relatively small area that is affected by the electrical stimulus (less than 9 mm^2) an extensive interference with both express and comprehension of language can be produced. This certainly contradicts the usual clinical observation that global aphasia is almost always the expression of a relatively extensive cortical lesion. We have already discussed in a previous paper the possibility that the stimulus actually could affect a more extensive region around the electrode but discarded this possibility as unlikely (Lüders et al., 1987). It seems, therefore, that the relative stronger intensity of the deficit produced by electrical stimulation is related to the acuteness of the insult. The acute and reversible effect of stimulation do not give the brain enough time to compensate whereas with a chronic lesion, such as a stroke, compensation can occur over time.

Reducing the intensity of the stimulus results in progressively less severe interference with language process which frequently can only be detected by careful testing using relatively more complex tasks as for example reading a more difficult paragraph, following a several step command, or solving more advanced arithmetic problems. In this context it is interesting to notice that most patients had considerable difficulty with naming objects even at relatively low stimulus intensities. On the other extreme, repetitive and automatic tasks like counting or reciting a well known rhyme was frequently performed well even at relatively high stimulus intensities. At some cortical areas even high intensity stimuli only elicited very mild aphasic defects. Presumably the location of the ellectrode in those areas was such that stimulation produced inactivation of only very limited cortical areas related to language functions.

Finally, we should address the question of why cortical stimulation only produces negative effects when stimulating the language areas. A language area represents

cortex that participates actively in the processing of language. This is an extremely complex task which must involve accurate fine tuning of the neuronal complex which forms the language area. Stimulation of such a neuronal set can certainly not reproduce the conditions to produce any reasonable output which may result in actual speech. It is difficult to understand, however, why there is not any vocalization or perception of unintelligible sounds or visual images when stimulating these areas. The total absence of any positive symptoms in spite of a complete speech arrest or total receptive aphasia leads us to suspect the existence of a protective mechanisms that nullifies any input or potential output if it is grossly distorted.

EXPRESSIVE VS RECEPTIVE LANGUAGE DEFICIT

The language deficit produced in all language areas was relatively similar regardless of whether we stimulated the anterior, posterior, or inferior language area. No specific language deficit was elicited in the supplementary motor area. This observation was in clear contrast with the clinical experience that lesions of the anterior speech area produce a predominantly expresive aphasia whereas lesions of the posterior language area produce a predominantly receptive aphasia. The results of electrical stimulation seems to suggest that lesions of a primary language area produces a predominant receptive aphasia unless the lesion is very severe (or acute) which would lead to a global receptive and expressive aphasia and total inability to process any verbal material. This is also true for primary language cortex located in Broca's area. It is possible that the predominantly expressive aphasia observed in patients with lesions in Broca's area is primarily related to a lesion of the negative motor area which is also located in the same region.

LANGUAGE DEFICIT PRODUCED BY LESIONS OF LANGUAGE AREAS IDENTIFIED BY ELECTRICAL CORTICAL STIMULATION

In our Lab we have usually avoided any resection of cortex which is eloquent by cortical stimulation. With this relatively conservative approach neurological deficits following cortical resection were almost exclusively limited to those cases in which a well defined complication occurred (like, for example, cortical infarct which extended into an eloquent area of cortex). Experience has shown, however, that resection of a cortical area that produces prominent symptoms when stimulated may not produce any deficit or only a very limited transient deficit when resected. This was shown first by Penfield and Jasper (1954) and is examplified best by the supplementary motor area. Stimulation of the supplementary motor area produces violent motor movements because of its predominant proximal somatic representation but its resection is not associated by any lasting motor deficit except a transient and very mild incoordination. We were also able to document a similar result after lesions or resection of the basal temporal language area. In the first few cases in which our stimulation results showed a basal temporal language area we carefully avoided resection of the portion of the fusiform gyrus which when stimulated produced language deficits. In one of the earlier cases, however, the patient suffered an infarct of the basal tem-

poral region (including the fusiform language area) following the temporal resection. We were surprised to see that the patient, in spite of an extensive infarction including the whole basal temporal language area, did not suffer any noticible language deficit even when tested quantitatively by a linguist. Following this case we have consistentntly resected the basal temporal language area. Resection of the anterior tip of the dominant temporal lobe produces in many cases a slight verbal memory deficit, and it is still unclear if this is related to the resection of the basal temporal language area. Extensive clinical experience clearly suggest that a lesion of Wernicke and Broca's area should produce severe language deficits. It is unclear, however, how extensive an area must be damaged to produce a clinically noticible deficit. The same may also be true for the basal temporal language area. In other words, a more extensive resection of that area may be associated with a verbal deficit can not be excluded.

SUMMARY

Results of cortical electrical stimulation and its effect on language function are presented. Evidence is presented indicating that speech arrest can be produced by the following 5 mechanisms : positive motor response, negative motor response, other non-motor positive responses, loss or alteration of consciousness, and primary language deficits. Stimulation of the supplementary motor area and Baroca's area usually produces speech arrest due to a negative motor response. No primary language deficits can be elicited in the supplementary motor area. In Broca's area, however, there were limited cortical areas in which stimulation produced specific language deficits. Stimulation of Wernicke's area and of the basal temporal language area (fusiform gyrus) produces speech arrest that consistently is due to a specific language deficit. At all stimulation sites the language deficit produced by electrical stimulation was similar (various degrees of expressive and receptive aphasia depending on the stimulation intensity). The clinical observation that a lesion of Broca's area produces a predominantly expressive aphasia may be related to the coexistence in that area of negative motor sites. The predominantly receptive aphasias seen with lesions of Wernicke's area may be explained by the absence of any negative motor responses in that area.

REFERENCES

1) Dinner, D. S., Lüders, H., Morris, H. H., Wylie, E. & Kramer, R. E. : Human supplementary motor area (SMA) : Electrical stimulation study. Epilepsia, 28 ; 619, 1987.

2) Fedio, P. & Van Buren : Memory deficits during electrical stimulation of the speech cortex in conscious man. Brain and Language, 1 ; 29-42, 1974.

3) Lesser, R. P., Lüders, H., Dinner, D. S., Hahn, J. & Cohen, L. : The location of speech and writing functions in the frontal language area. Results of extraoperative cortical stimulation. Brain, 106 ; 275-291, 1984.

4) Lesser, R. P., Lüders, H., Morris, H. H., Dinner, D. S., Klem, G., Hahn, J. & Harrison, M.: Electrical stimulation of Wernicke's area interferes with comprehension. Neurology, 36 ; 658-663, 1986.

5) Lueders, H., Lesser, R. P., Dinner, D. S., Morris, H. H. & Hahn, J. : Inhibition of motor activity elicited by electrical stimulation of the human cortex, Epilepsia, 24 ; 519, 1983.

6) Lüders, H., Lesser, R. P., Dinner, D. S., Morris, H. H., Wyllie, E. & Klem, G. : Comprehension deficits elicited by electrical stimula-

tion of Broca's area. Epilepsia, 27 ; 598-599, 1986 a .

7) Lüders, H., Lesser, R. P., Hahn, J., Dinner, D. S., Morris, H. H., Resor, S. & Harrison, M. : Basal temporal language area demonstrated by electrical stimulation. Neurology, 36 ; 505-510, 1986b.

8) Lüders, H., Lesser, R. P., Dinner, D. S., Morris, H. H., Hahn, J., Friedman, L., Skipper, G., Wyllie, E. & Friedman, D. : Comentary: Chronic intracranial recording and stimulation with subdural electrodes. in Surgical Treatment of the Epilepsies. (Engel, J.), Raven Press, New York, pp, 297—321, 1987.

9) Lüders, H., Lesser, R. P., Dinner, D. S., Morris, H. H., Wyllie, E., Godoy, J. & Hahn, J. : A negative motor response (NMR) elicited by electrical stimulation of the human frontal cortex. in Frontal Lobe Seizures and Epilepsies. (Chauvel, P., Delgado-Escueta, A. V., Halgron, E., Bancard, J.) Raven Press, in press, 1989 a .

10) Lüders, H., Lesser, R. P., Hahn, J., Dinner, D. S., Morris, H. H., Wyllie, E. & Godoy, J. : Basal temporal language area. Brain, submitted for publication, 1989 b .

11) Ojemann, G. A. : Organization of short term verbal memory in language areas of human cortex and evidence from electrical stimulation. Brain and Language, 5 ; 331—340, 1978.

12) Ojemann, G. A. : Individual variability in cortical localization of language. J. Neurosurg., 50 ; 164—169, 1979.

13) Penfield, W. & Rasmussen, T. : Vocalization and arrest of speech. Archives of Neurology Psychiatry (Chicago), 61 ; 21—27, 1949.

14) Penfield, W. & Rasmussen, T. : The Cerebral Cortex of Man. McMillan, New York, 1950.

15) Penfield, W. & Jasper, H. : Epilepsy and the Functional Anatomy of the Human Brain. Little, Brown and Company, Boston, 1954.

16) Penfield, W. & Roberts, L. : Speech and Brain Mechanisms. University Press, Princeton, 1959.