Preoperative Electroencephalographic Localization of Large Epileptogenic Zones in the Frontal and Temporal Lobes
Cosimo Ajmone-Marsan

ABSTRACT: The concept of epileptogenic zone is defined as a large area of cortical neurons arranged in concentric circles of variable degrees of epileptogenicity. This is particularly so in frontal lobe epilepsy since the interictal and ictal epileptic abnormalities are poorly localized, often absent and at times misleading in terms of localization. In temporal lobe epilepsy, the epileptogenic zones may be more restricted.

In the late forties, there were essentially three main “approaches” to electroencephalography. One school emphasized the morphological features of the changes in electrical activity. Another stressed the significance of background rhythms, with automatic analysis of their frequency and amplitude. The third, while taking into consideration these various parameters, placed its emphasis on the actual location of changes, especially those characteristic of seizure disorders. The latter was typical of the Montreal school, and developed to a high degree by Herbert Jasper.

In the last thirty years or so, important improvements have included the manufacture of recording equipment, with 16, 20, 32 or more channels, and the development of computer-aided identification, localization and quantification of epileptiform phenomena.

I wish to add my personal tribute to the recognition of the enormous contributions of the MNI school to clinical EEG in the field of seizure disorders.

The presentations by Gloor and Quesney (as well as their discussions in the Palm Springs Symposium) exemplify the clearly expressed concepts that cover the field of EEG in the preoperative work-up in epileptic patients. The concept of “large epileptogenic zones” should obviously imply the existence of discrete, truly “focal” processes. One should distinguish between functional-electrographic phenomena on the one hand and functional-structural situations on the other. Both may appear to be circumscribed to a relatively small region involving limited neuronal aggregates on the basis of both electrographic and/or imaging criteria, but the crucial confirmation of the true focality of a process will eventually be provided only by the results of surgical excision. In a Symposium on “Changing Concepts in Focal Epilepsy” 28 years ago, I discussed the significance of the results of surgical treatment to the problem. I outlined all possible types of outcomes following a limited cortical excision for treatment of seizures and listed probable explanations for failure. Those same arguments were re-elaborated in 1987 by Engel and Hauser.

These investigators emphasized the practical aspects of what constitutes good or poor results from the point of view of the patient’s welfare, the various philosophies involved in the evaluation of results and the problems in standardizing and reporting to allow comparison between different centers.

Permanent disappearance of seizures following a relatively limited excision of cortical tissue is strong evidence in support of the concept of the pathophysiology of focal or partial seizures. But even certain partial failures can be used as arguments to support the validity of the concept; for example 1) persistence of seizures but with a modified pattern; 2) persistence of one out of several different types of seizures that were present before operation; and 3) appearance of seizures with completely new patterns, especially several years after the surgical procedure. Also failures from inadequate preoperative work-ups, due to errors of interpretation of diagnostic data, of electrocorticographic findings, or to inadequate topographic excisions, should not require conceptual changes.

Certain forms of surgical failures are more difficult to explain, for instance, the persistence of a few seizures per year, with exactly the same pre-surgical pattern.
Rasmussen,6 has formulated a theory applicable to frontal lobe seizures, but of general validity in other forms of epilepsy, to define and better understand the concept of “epileptogenic area”. One is not dealing with a focal process of a limited neuronal aggregate susceptible to epileptiform activity in an “all-or-none” fashion and sharply demarcated from normal tissue. Rather, one should visualize the epileptogenic process as a much larger area of neurons arranged in concentric circles of variable degrees of “epileptogenicity”, maximal at a center characterized by the lowest excitability threshold, with progressive increase of threshold in the neurons at the periphery. Rasmussen reached these interpretative conclusions on the basis of the greater incidence of surgical successes following larger areas of excision.

Gloor1 also stressed that the concept of a small focal process is “not commensurate with clinical reality”.

We confirm Rasmussen’s conclusions in the sense that when the topographic diagnosis of fronto-mesial, or fronto-orbital or frontopolar processes has been made, and on such basis the excision has been limited to focal frontal sector excisions — the outcome has not been satisfactory.7,8

A related, puzzling paradox is that the electrographic abnormalities involving the frontal lobe may either be widespread, especially in the interictal phase, or almost absent from the scalp recording, thus requiring elaborate and often invasive techniques for their demonstration.

For temporal lobe seizures, the picture seems to be more direct. In our experience, even here, there has generally been a better outcome with extensive excision of temporal lobe tissue than with more limited ablations.9

In regard to the problem of large versus discrete epileptogenic zones, we may well ask should one make all possible efforts to pinpoint the center of a hypothetical “focus”, including patterns and directions of spread of ictal activities, with lengthy and costly procedures that are not always harmless? Or would it be sufficient to differentiate the primary involvement among different lobes and/or to identify the lateralization of the main process? And what do we do when we excise this “epileptogenic zone”? Do we eliminate the population of abnormal neurons, or do we simply interfere with its efferent pathways and with the spread of epileptiform activity to other structures? The numerous instances in which a typical aura is preserved would be more in favor of the latter explanation.

These are among the major questions that still remain to be posed and examined.

REFERENCES