THE SURGERY OF EPILEPSY

LIMITATIONS OF THE CONCEPT OF THE CORTICO-ELECTROGRAPHIC "SPIKE" AS AN INDEX OF THE EPILEPTOGENIC FOCUS

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Most neurosurgical procedures directed against epilepsy have been fashioned upon the generally accepted hypothesis that a hyper-irritable cortical focus ("epileptogenic focus," "firing point") is a prime factor in the pathogenesis of epileptic seizures. The hypothesis itself is based upon the pioneer work of Hughlings Jackson (1861–83), Fritsch and Hitzig (1870), Ferrier (1873), Munk (1880), Gowers (1881) and Talbert (1899), and from it follows logically the concept that the alleviation of epileptic seizures may be expected from (a) identification of the hyper-irritable focus and (b) its extirpation.

Unfortunately, the number of individuals who have obtained complete relief from epileptic attacks following surgical procedures does not conform closely to theoretic expectations. Even when grossly apparent lesions, such as cortico-meningeal scars, cysts of the cortex, and circumscribed atrophic areas are encountered at operation, only about one-fifth of patients subjected to operation can be freed of their seizures (Penfield and Erickson6). When, on the other hand, grossly apparent lesions are not in evidence and the surgeon is under the necessity of demonstrating the hypothetical firing point by physiologic means only, the results thus far have proven similarly disappointing.

This disparity between expected and actual clinical results is commonly accounted for by (a) citing the various practical difficulties encountered in identifying the cortical firing point; (b) assuming incomplete extirpation of the epileptogenic focus; (c) supposing the existence of more than one firing point; and/or (d) citing the inadvisability in certain instances of extirpating the cortical focus because of the likelihood of impairing crucial functions, such as speech and eupraxia. The fundamental concept of the cortical epileptogenic focus is, of course implicitly contained in all four of these accounts.

In consideration of the first three explanations (a, b and c above) the realization of better results would appear to reside in the improvement of methods for locating and precisely defining the limits of the cortical firing point. To this end, clinicians have striven toward more accurate observation and description of the convulsive seizure peculiar to each patient. Improved types of cortical stimulators have been designed, affording increasing control

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over voltage, amperage, wave form, frequency and pulse-duration for threshold determinations of cortical tissue. Roentgenography, including pneumoencephalography and cerebral angiography, has been extensively utilized and during the past 15 years electroencephalography has been employed to the same end. In connection with the latter diagnostic procedure, the concept has been evolved that the cortico-electrographic "spike" may be considered an indicator of the epileptogenic focus.

The assessment of spikes in the electroencephalograms of patients with convulsive disorders has been the particular object of several recent investigations (Jasper, Walker et al., Penfield and Jasper). The general viewpoint relative to post-traumatic epilepsy has been summarized by Walker et al.: "In post-traumatic epilepsy focal *spikey* waves or spikes about the site of the injury indicate a convulsive diathesis." In an extensive series of cases, Walker et al. showed that areas of after-discharge of spike and associated phenomena could be localized electrocortically following infraliminal sine-wave stimulation. Of these findings they say:

"These observations seem to indicate that certain cortical areas adjacent to cerebral scars may be excited by infraliminal stimuli producing a state of local epileptic hypersynchrony. Although these phenomena have many characteristics of cortical after-discharge, their lower threshold, more localized activity and longer duration suggest that they are different, at least in degree. That areas capable of such activity are potentially epileptogenic seems highly probable. That they are the foci, from which spontaneous epileptic attacks originate, lacks demonstration, but it seems a reasonable inference in those instances in which the electrocorticographic attack is associated with an aura identical to that in the spontaneous attacks. If the focus is some distance from the scar, another and perhaps primary focus may be located nearer the injury."†

Inferentially, then, the spike *per se* is regarded as evidence of an epileptogenic focus, and from this it would follow that excision of the spike zone may be expected to have a salutary effect on the patient's seizures.‡

Another view of "spikes" and "spikey" (or "sharp") waves is given by Jasper. While he concurs in the notion that clean-cut spikes of brief duration are evidence of a focal zone of discharge, he considers that spike-like waves or "sharp" waves are the conducted end-result of spikes originating at a distance. The evidence supporting this view is convincing.

It is clear that caution must be exercised in differentiating between the two types of cortical potential change which may casually be included in the generic term "spiking." In this connection, Jasper and Droogleever-Fortuyn, by stimulating midbrain and diencephalic structures, have demonstrated spikes formed with a dome simulating the familiar "petit mal" seizure pattern. These are, to be sure, not spikes in the sense that they are

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* Italics ours.
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‡ This inference was made rather explicit by Quadfasel and Walker in a description of the delimitation of an epileptogenic focus. Their report was made relatively early after completion of an operative series and for this reason no statistical data were given. On the basis of clinical impression, however, the authors were inclined toward belief that the results were beneficial.