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Commentary

In Vivo Imaging of Human Anatomy in Temporal Lobe Epilepsy

Frank Morrell[1]

Temporal lobe epilepsy is the most important cause of medically intractable seizures. Complex partial seizures, the main clinical manifestation of temporal lobe epilepsy, arise primarily from the mesial temporal structures, the amygdala, and the hippocampus [1, 2]. This form of epilepsy is also the most readily treatable with surgery: in most series, recovery is reported in 60–85% of cases [3]. The pathologic entities associated with temporal lobe epilepsy are varied and include tumors, vascular malformations, heterotopias, and hippocampal sclerosis [4–6]. Hippocampal or mesial temporal sclerosis is by far the most common pathologic finding, occurring in approximately 65% of cases [5, 6]. In those cases without evidence of alien tissue, hippocampal sclerosis is thus the overwhelmingly predominant pathology; furthermore, its location coincides with the electrophysiologically defined origin of most complex partial seizures [1]. Finally, there is a high correlation between the presence of hippocampal sclerosis in the excised surgical specimen and the likelihood that the patient will benefit from the surgical procedure [5].

Unfortunately, in many cases of temporal lobe epilepsy, the routine diagnostic studies may not clearly indicate the side of the disease. Clinical manifestations may be ambiguous with respect to lateralization, and electroencephalographic discharges may often appear bilaterally. Accordingly, it is clear that any method of preoperative identification of hippocampal sclerosis would be of significant clinical value.

Because previous reports of MR detection of hippocampal sclerosis have been controversial [7–13], the articles by Bronen et al. [14] and Ashtari et al. [15] in this issue of the *AJNR* are of special importance. Both studies make use of substantial advances in MR technology or in quantitative computational evaluation. Both provide strong positive evidence for MR detection of hippocampal sclerosis and the atrophy associated with it. They support the earlier observations of Jackson et al. [16, 17] and of Jack et al. [18], and emphasize the importance of quantitative and objective measures.

In the study by Bronen et al. [14], despite the promise of an earlier, more truly volumetric examination [19], a "gross estimate" of hippocampal size derived from the product of height × width was used in an apparent effort to derive a quantitative measure that might be available to radiologists who do not have access to the computer facilities for accurate volume calculations. These measures were compared with cell counts of hippocampal subfields taken from the body or midportion of the excised hippocampal formation. The comparisons yielded fairly high correlations for the CA_3, CA_4, and dentate gyrus cell fields, but not for the CA_1 cell field (which is the most consistently affected in mesial temporal sclerosis). Furthermore, in the blinded portion of their study, correct identification of the side of hippocampal sclerosis was achieved in only five of nine (observer 1) or six of nine (observer 2) patients. To be sure, the raters became more accurate in the second phase (nonblinded) of their study; nonetheless, it is necessary to attempt to specify possible sources of uncertainty that could have contributed to the outcome.

One of the sources of error may have been in the "corrected" height measurement used to approximate a 90° hippocampal angle for coronal images. The authors used the plane of the inferior aspect of the *left* hippocampal formation to provide a correction factor for both right and left hippocampi. Evidence from autopsy studies and from properly positioned MR images indicate that the long axis of the left hippocampus differs from that of the right (H. Damasio, personal communication). Thus, correcting only for the left side would introduce a systematic error in volume calculations for the right side. In a similar vein, age-related or developmental differences in the "hippocampal angle" may have contributed

This article is a commentary on the articles by Bronen et al. and Ashtari et al.

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to the failure of volumetric measurements to correlate with disease in children with temporal lobe epilepsy, although a shorter duration of illness may be the more important factor [20]. These problems may be overcome in *prospective* studies, in which proper positioning of the patient can be carried out to begin with. It will still be necessary to take into account left–right differences in the angle of the long axis of the hippocampus in order to resolve some persisting discrepancies. For example, the control patients of Ashtari et al. [15] showed a relatively smaller right hippocampus while the opposite difference has been reported in other studies [19, 21, 22].

None of these comments or criticisms diminish the significance or importance of the contributions of these papers to the diagnosis of temporal lobe epilepsy. Rather, in fact, I suggest that the quantitative approach of these investigators and of other recent neuroradiologic research [23, 24] represents a potential contribution to neurology of far greater importance than that of diagnosis in epilepsy. The importance of the hippocampus and its subfields to epilepsy has, perhaps inadvertently, resulted in the realization of the enormous resolution of MR imaging for normal as well as pathologic anatomic detail. Hippocampal involvement in Alzheimer disease and in memory disorders of other sorts has similarly spawned extremely valuable MR investigation [23, 24]. Obviously, however, the principle that detailed brain anatomy can be demonstrated in the living human being applies to all other regions of the brain as well when the proper orientations and conditions for imaging them are discovered.

The situation today is perhaps analogous to that for neurology in the 19th century. Indeed, modern neurology may be considered to have begun with the emergence of pathologic anatomy in the 19th century. The emphasis on autopsies coupled with the discovery of the aniline dyes and silver precipitation techniques that allowed microscopic examination of nervous tissue made possible the first systematic correlations between the symptoms of neurologic disease and disorder of specific sites in the brain and spinal cord. Together with the developing science of experimental neurophysiology, the observations established the main principles of localization of cerebral function—principles that formed the foundation of neurologic diagnosis, but also, to a great extent, the foundation of how we understand normal brain function.

However, the problem with pathologic anatomy, or "silver-plated neurology" as it was sometimes called, was that the autopsy most often took place months or years after the occurrence or first appearance of the neurologic symptom. Recovery processes, the development of intervening symptoms, or further deterioration limited the precision with which causative inferences could be drawn between the autopsy-documented lesions and the behavioral manifestations.

The potential "great leap forward" that these techniques imply may not be immediately obvious to radiologists, who, after all, provide images of the living human body on a daily basis. For the neurologist, however, who is more used to awaiting the (now nearly vanishing) autopsy before obtaining the information necessary for true clinicopathologic correlation, the availability of high-resolution MR for in vivo, then and there, delineation of anatomy is an exciting prospect. The correlation of such human observations with those of experimental neuroanatomy will certainly lead to far-reaching advances in the understanding of complex behaviors, including memory, attention, language, and cognition. The anatomically oriented and interested neuroradiologist of today should be a major participant in research at the frontiers of brain science.

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