Temporal Lobectomy, with Special Reference to Selection of Epileptic Patients*

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It is now apparent that sclerosis of the infero-mesial aspect of the temporal lobe is the most common focal lesion in epilepsy and that it is also the most favorable from the standpoint of successful surgical treatment.\(^{14,30,35}\) These conclusions, which have evolved slowly, have aroused increasing interest in the subject of temporal lobectomy.

Historical Development

The Beginnings (1825–1939). Macroscopically detectable lesions (sclerosis or softening) in the hippocampus were first described in 1825 by Bouchet and Cazauvelli,\(^7\) according to Spielmeyer,\(^62\) in epileptic as well as non-epileptic psychopathic patients. Sommer,\(^41\) in 1880, was apparently the first to examine them microscopically. He found the changes to be restricted to the band of pyramidal cells of the hippocampus, an area now called Sommer's sector. His findings were confirmed in 1899 by Bratz,\(^8,9\) who noted that the end plate was affected as often as Sommer's sector.

Gowers\(^35\) questioned the relationship of the induration of the cornu Ammonis to epileptic seizures and with each epileptic patient sought answers to the following questions:

1. What is the seat of the discharge which thus produces the symptoms of the fit?
2. Is the seat of the discharge the seat of the disease?
3. How far does such discharge explain all the symptoms of the attack?
4. What is the nature of the morbid change which causes the discharge?

During the early 20th century there was little concern with sclerosis of the hippocampal formation until 1927, when Spielmeyer\(^62\) postulated these changes to be in the nature of ischemia brought about by vasospasm, which he regarded to be the result, not the cause, of seizures. He was not alone in his belief that the hippocampus was important only as related to olfactory function. Penfield, according to Malamud,\(^41\) commented in 1929 that "the changes in Ammon's horn reported by Spielmeyer are perhaps the farthest removed from the attention of those interested in the epileptic mechanism."

Bard,\(^6\) as early as 1929, conceived that emotions were built up in the hippocampal formation. Herrick,\(^32\) in 1933, suggested that the limbic lobe of Broca, or "rhinencephalon," served as a non-specific activator for all cortical functions influencing appropriately, in an excitatory or inhibitory manner, such functions as memory, learning, and behavior, as well as olfaction. Utilizing electrical recordings of potentials, Sugar and Gerard,\(^63\) in 1938, showed that the gray matter of Ammon's horn and cerebellum are the areas of the brain most liable to be affected by anemia, thus providing a physiological explanation for Spielmeyer's pathological findings in the cornu Ammonis.

More Recent Developments (1940–1949). On the basis of operative experiences, Penfield and Erickson\(^49\) asserted in 1941 that the focus of dreamy-state seizures was in the temporal lobe, and Jasper and Kershman\(^38\) suggested the role of the temporal lobe and subcortical structures in the production of automatisms.

Before 1947, epileptic patients were selected for temporal lobe surgery if a well-defined lesion was evident. The rehabilitation rate was low by present standards. The early concepts regarding sclerosis of Ammon's horn and adjacent structures prevailed; namely, that this abnormality was not related to the cause of epilepsy. Neuro-
logical surgeons considered the clinical seizure pattern and electroencephalographic localization alone to be insufficient indications for surgical intervention and also excluded those patients with focal epilepsy if they also had any serious psychiatric disorder.

In 1947–48, Fuster and the Gibbss,22,23 and Bailey and Gibbs,2,4 provided impetus for intensified study of the temporal lobe clinically and in the laboratory. Fuster and the Gibbss identified focal spike discharges in the anterior temporal area in the great majority of patients with psychomotor epilepsy provided that electrode placements were adequate and sleep studies were used. Bailey and Gibbs described temporal lobe surgery performed for psychomotor seizures on the basis of lateralization of the epileptogenic focus by EEG alone. They also included patients with psychiatric abnormalities.

The possible role of the hippocampal formation and its sclerosis as part of the "visceral brain" or limbic system was considered by MacLean29 to be important to an understanding of psychomotor epilepsy and the Papez Theory of Emotion.41 Thus, by 1949, the various lines of investigation of these seemingly distinct areas of medicine were beginning to assume meaning and unity but were still confusing because of differences of emphasis and terminology.

Developments Since 1950. A remarkable crescendo of activity has occurred since 1950. A comprehensive review made recently by us (Green and Scheetz),30 supplemented by pertinent reports during the 2 years since its publication, can be summarized under four headings: 1) terminology, 2) mechanisms, 3) epileptogenic lesions, and 4) surgical technique.

Terminology

Seizures may be classified in descriptive terms, or anatomically on the basis of their site of origin. This has led to controversy and confusion in the related terminology.

Since 1947 when Fuster, et al., determined that psychomotor epilepsy originated in the temporal lobe, both this type of seizure (the "uncinate fits" described by Jackson,26,30) and the temporal lobe seizures,12,15,18,38,49–56,62,64 have, in many instances, been indiscriminately classified as temporal lobe epilepsy. Experience has shown that a significant number of patients who have been thought to have psychomotor epilepsy, uncinate seizures, or temporal lobe epilepsy have had their epileptogenic areas and pathology in the opposite temporal lobe, in the insula, in the orbital portion of the frontal lobe, or in subcortical areas of the limbic system. Morrell46 has worked out a simpler classification, "limbic epilepsy," which includes the majority of these patients. Robb46 has made a new classification which is very complete and complex and de-emphasizes both "psychomotor epilepsy" and "temporal lobe epilepsy."

We suggest that these present avenues of classification should be maintained but that they should be combined in the diagnosis of each epileptic patient, for example, descriptive, anatomical, physiological, and pathological.

Mechanisms

The hippocampal formation is strategically related to the heart of the limbic system, connecting the uncus, amygdala, and the anterior temporal cortex to the mammillary bodies, septal nuclei, anterior thalamic nuclei, cingulate and orbital gyri, and the homologous areas of the temporal lobe. Green and Adey30 have suggested that the amygdala and hippocampus play rather complex roles in the visceral system of the brain, perhaps analogous to the basal ganglia and cortex in other areas. Extensive study of the limbic system and its hippocampal formation has continued with the growing realization that morbid processes in these structures are basically concerned in the pathogenesis of psychomotor epilepsy. MacLean40 emphasized that there may be a propagation of hippocampal discharges without alteration of neocortical activity and considered the limbic system to be intimately related to behavioral abnormality and psychomotor epilepsy in man.

A concept of the anatomico-physiologicoclinical functions of the limbic lobe was recently (1965) presented by White59 and Foltz.20 The authors have divided the limbic lobe into two almost concentric rings with free interaction between these two cortexfiber systems and the extra-limbic cortex. The "inner limbic ring" includes the amyg-