Localization of Temporal Lobe Foci by Ictal EEG Patterns

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Summary: Identifying patients whose complex partial seizures originate in temporal neocortex rather than in hippocampus is important because such patients have less favorable outcomes with standard anteromesial temporal resections. We reviewed scalp-recorded ictal EEGs of 93 epilepsy surgery candidates who either underwent intracranial EEG monitoring (n = 58) or who were referred directly for temporal lobectomy (n = 35). We defined seven patterns of early seizure discharges, grouped patients according to their seizure pattern, and correlated these with the site of seizure onset determined by intracranial EEG. Categorization by seizure pattern was also compared with brain magnetic resonance imaging (MRI) findings and intracarotid amobarbital (Wada) testing. An initial, regular 5- to 9-Hz inferotemporal rhythm (type 1A) was most specific for hippocampal-onset seizures. Less commonly, a similar vertex/parasagittal positive rhythm (type 1B) or a combination of types 1B and 1A rhythms (type 1C) was recorded. Seizures originating in temporal neocortex were most often associated with irregular, polymorphic, 2- to 5-Hz lateralized activity (type 2A). This pattern was commonly followed by a type 1A theta rhythm (type 2B) or was preceded by repetitive, sometimes periodic, sharp waves (type 2C). Seizures without a clear lateralized EEG discharge (type 3) were most common of temporal neocortical origin. These associations between type of seizure pattern and probable site of cerebral origin were statistically significant. MRI and Wada testing did not have as much specificity as ictal patterns in differentiating among seizure origins. We conclude that the initial pattern of ictal discharge on scalp EEG can assist in distinguishing seizures of temporal neocortical origin from those of hippocampal onset. This information can be used to identify patients for invasive monitoring. Key Words: Electroencephalography—Epilepsy—Seizures—Temporal lobe—Localization.

Localization of the epileptogenic focus is the principal goal of long-term monitoring in a presurgical evaluation. EEG recorded noninvasively from scalp and sphenoidal electrodes provides much of the information needed in this effort (1–3). Most epileptologists believe that the ictal EEG is necessary for definitive localization because bilateral interictal epileptiform discharges are commonly observed in patients with strictly unilateral seizures (4), and even unilateral spikes cannot always distinguish between mesial or lateral temporal or extratemporal foci. Although the spatiotemporal characteristics of the EEG during complex partial seizures have been described by some investigators (5–9), few have attempted to categorize these ictal patterns on the basis of seizure origin. Risinger et al. (10) reported that a lateralized ≥5-Hz seizure discharge within 30 s of seizure onset was highly correlated with seizure onset in the ipsilateral mesial temporal lobe. This ictal abnormality was rarely falsely lateralizing, but approximately half of their patients did not have this EEG pattern.

Recently, it has become evident that a significant group of patients with temporal lobe epilepsy have seizures that originate from neocortex and not from the hippocampus. The syndrome of “neocortical” temporal lobe epilepsy is only now being defined, and the ictal patterns associated with temporal neocortical onset have not been well described or validated. Early results, however, suggest that standard anteromesial temporal lobectomy does not have as favorable an outcome in such patients as in those with mesial temporal lobe epilepsy (11). Distinguishing neocortical from hippocampal temporal lobe epilepsy early in a presurgical evaluation is important, because patients with neocortical sei-
zture onsets probably require invasive monitoring to define their epileptogenic zone more clearly so that the surgical resection can be appropriately tailored.

We believe that additional localizing information can be extracted from the ictal EEG and used to make presurgical decisions. We propose that surface-recorded partial seizures can be categorized according to the pattern of the early ictal EEG discharge and that this classification is frequently diagnostic of seizure origin. These findings were presented previously as abstracts (12–14). The intracranial EEG correlates of these scalp patterns will be the subject of a separate publication.

**METHODS**

We reviewed scalp-recorded ictal EEGs from 93 patients with complex partial seizures due to temporal lobe epilepsy. Focus localization was determined by intracranial monitoring before resective surgery (n = 58) between 1989 and 1993, by combined clinical features that strongly suggested mesial temporal seizure onsets (n = 15), or by magnetic resonance imaging (MRI) that demonstrated a temporal lobe tumor (n = 17). Three other patients were referred directly to surgery because medical complications precluded intracranial EEG. We excluded patients whose seizures were shown by invasive monitoring to originate outside the temporal lobe.

Three hundred ninety-one seizures were categorized by consensus between the authors. A minimum of two seizures (maximum of nine) were reviewed for each patient. Twenty to 27 channels of EEG were recorded on VHS tape at a 200-Hz sampling rate (Telefactor Beehive) during long-term monitoring in the epilepsy units of West Haven VA Medical Center (WHVAMC) or Yale-New Haven Hospital (YNHH). International 10–20 placements with the addition of three inferior temporal electrode locations bilaterally (F9,F10,T9,T10,P9,P10) were used most commonly. We analyzed ictal segments of EEG from either paper printouts or directly from the screen of a high-resolution video monitor (Telefactor Beekeeper). Longitudinal bipolar montages with a bandpass of 1–70 Hz were always inspected, but we made our final categorization from EEG displayed with a common Oz reference with a narrow bandpass (3–12 Hz). The occipital midline reference was chosen because of its relative inactivity in most temporal lobe seizures from either hemisphere. Narrow bandpass filtering was used to accentuate EEG activity in the frequency range of most temporal seizure rhythms (7,9) and to minimize movement and muscle artifact, which commonly obscure ictal recordings. Ictal patterns were classified based on the frequency, spatial distribution, morphology, and evolutionary characteristics of the earliest definable EEG ictal waveforms. Seizures accompanied only by diffuse and irregular slowing or by attenuation of background activity were placed in a separate category.

In 58 patients, we were able to correlate the pattern of scalp EEG ictal discharge with the site of seizure onset determined using 64 channels of intracranial EEG. A combination of bilateral depth electrodes and subdural strips was used in most patients. Thirteen patients with clinical data strongly suggesting nonmesial temporal epilepsy had subdural grid or strip electrodes only. Scalp electrodes recorded simultaneous surface ictal data in 24 patients. The location of intracranial electrode contacts was verified by reconstructed MRI scans. Details of these techniques were reported previously (15–17).

The principal distinction we made was that between seizures of hippocampal origin and those of regional or definite temporal lobe neocortical origin. Seizures were classified as “hippocampal” when the electrographic onsets were localized to appropriate contacts of the mesial depth electrode. Typically, ictal activity did not spread from these hippocampal contacts for many seconds. Regional seizure onsets (18), in which ictal discharges were simultaneously recorded from all temporal contacts of the depth electrode (including those anterior to the hippocampus and amygdala) and from anterobasal temporal subdural electrodes, were presumed to be of nonhippocampal origin and were classified as neocortical. Typically ictal activity in these seizures spread rapidly to involve all of the ipsilateral temporal lobe. Seizures that clearly originated in temporal neocortex and did not initially involve hippocampal contacts were also classified as neocortical. We used Fisher’s exact t test to determine if the correlations among ictal patterns and seizure origins were significant.

Ictal EEG categories were also compared with brain MRI findings and intracarotid amobarbital (Wada) testing of lateralized memory function. Qualitative assessments of hippocampal and temporal lobe atrophy were made in all but 1 patient (n = 92). Quantitative volumetric measures of hippocampal symmetry were available in 68 patients (19,20). Patients were deemed to have inadequate hemispheric memory (a Wada test failure) when they remembered fewer than 2 of 6 (WHVAMC) or 4 of 10 (YNHH) visual stimuli 10 min after amobarbital.
injection, as measured by a combination of free and multiple-choice recognition recall.

RESULTS

Type 1 ictal EEG patterns

Almost all seizures in 15 patients were accompanied by a progressive buildup of a regular 5- to 9-Hz EEG rhythm of uniform morphology that persisted >5 s with the same morphology and was localized principally to the subtemporal and temporal electrodes on one side (Fig. 1). When this was the first definable ictal activity, we categorized the seizure as type 1A. In the middle and later portions of this type of seizure, slower and more diffuse rhythms were common. Thirteen patients had seizures only of this pattern. In 2 other patients, the EEG in most seizures showed a regular theta rhythm of predominant positive polarity which was distributed broadly over the midline and frontal-central parasagittal electrodes (Fig. 2). In a longitudinal bipolar montage, this discharge commonly appeared as frontopolar and posterior activity due to voltage cancellation in channels with frontal-central electrodes. We designated this pattern type 1B. Four patients with predominantly type 1A or 1B ictal patterns had some seizures in which the 1B morphology was followed by type 1A (Fig. 3). We termed this combined seizure pattern type 1C. Patients with type 1 seizures did not have other seizure patterns.

Seven of the 17 patients with type 1 EEG seizures underwent intracranial EEG monitoring. Six of the seven (86%) had seizure onsets recorded exclusively from hippocampal electrodes. Only 1 patient had ictal onsets recorded from subdural electrodes and not those in the hippocampus; in this case, the active electrodes lay on the parahippocampal and fusiform gyri. Four patients (24%) proceeded directly to surgery because MRI had demonstrated lesions that proved to be tumors. All tumors were in the mesial temporal lobe and did not involve inferior, middle, or superior temporal gyri. Six other patients (35%) had surgery without invasive monitoring because combined clinical information (excluding seizure categorization) strongly suggested mesial temporal seizure onsets.

Eight of the 17 patients (47%) had MRI scans that showed atrophy of the hippocampus on the same side as the scalp EEG seizure, and 3 patients (18%) had both temporal lobe and hippocampal atrophy. Ten of 12 patients (83%) studied in this manner had quantitative asymmetry of the hippocampi. Seven patients in this group (41%) did not demonstrate adequate memory ipsilateral to the seizure pattern on intracarotid sodium amobarbital testing (Table 1).

Type 2 ictal EEG patterns

The early EEG ictal discharge in 53 patients was characterized by irregular EEG rhythms in the 2–5-Hz range that were lateralized to one hemisphere but less often were localized only to the temporal electrodes. These rhythms exhibited little stability, and changes in morphology or frequency occurred every few seconds (Fig. 4). We termed this seizure pattern type 2A. It was the exclusive seizure pattern in 36 patients. In 17 other patients, the type 2A seizure pattern was followed, usually within 30 s, by a regular, higher frequency lateralized theta rhythm reminiscent of the type 1A pattern (Fig. 5). We designated this combined pattern type 2B. Fifteen of the 53 patients had seizures in which the type 2A or 2B pattern was preceded by a 1–2-s burst of irregular sharp waves or a run of periodic (1–3 Hz) sharp waves lasting several seconds (Figs. 6 and 7). These “ictal-onset potentials” could vary between a more burstlike and a more periodic character in the same patient from seizure to seizure. We termed this a type 2C pattern. Patients with type 2B or 2C seizures could have some type 2A seizures, but no patient with type 2 seizures also had type 1 seizures.

Thirty-two patients with type 2 seizures underwent intracranial EEG monitoring. Seizures with temporal neocortical onsets were observed in 27 patients (84%), whereas hippocampal-onset seizures were noted in 5 (16%). No patient with type 2C seizures had hippocampal-onset seizures documented by depth EEG. Eight of 53 patients in this group (15%) were offered surgery for presumed mesial temporal lobe epilepsy without invasive studies, and 3 other patients (6%) proceeded directly to surgery because medical complications precluded invasive monitoring. Ten other patients (19%) had surgery because their MRI scans showed tumors. Brain MRI showed hippocampal atrophy ipsilateral to the EEG ictal discharge in 14 patients (26%) of this group. Nine (17%) had hippocampal and temporal lobe atrophy, and 16 of 37 patients (43%) had quantitative hippocampal asymmetry. Twelve patients (24%) of the 51 who had sodium amytal testing showed inadequate memory on the same side as the EEG ictal pattern.

Type 3 ictal EEG pattern

Twenty-three patients, one fourth of all patients in the study, had clinical seizures without a distinct EEG ictal discharge. In these patients, clinical seizure activity was accompanied by an EEG showing an interruption of normal background activity and
A type 1A seizure pattern: steady evolution of a regular 6- to 7-Hz left subtemporal and temporal ictal rhythm. In this and in Figs. 2–7, the top EEG traces provide an overall view of seizure morphology, and those below show shorter epochs of selected channels at an expanded time scale. Epochs displayed are denoted on top traces by underscores. In Figs. 1–7, the EEG is displayed with a common OZ reference and 3–12-Hz bandpass filtering. Vertical time scale at 1-s intervals; calibration markers = 100 μV.
FIG. 2. A type 1B seizure pattern with regular 5-Hz vertex and parasagittal ictal rhythm.
FIG. 3. A type 1C seizure pattern. After muscle artifact, a 5-Hz right frontal and parasagittal rhythm evolves within 12 s into a 6- to 7-Hz right subtemporal and temporal ictal rhythm.
often diffuse irregular slowing. We termed this a type 3 seizure pattern. Nineteen of the patients underwent intracranial EEG monitoring. Four (21%) had seizures of hippocampal onset, and 15 (79%) had neocortical onset seizures. Only 1 patient (4%) proceeded directly to surgery for presumed mesial temporal lobe epilepsy. Three patients (13%) had tumors. Unilateral hippocampal atrophy was demonstrated by MRI in 6 patients (26%), and 1 patient (4%) had temporal lobe atrophy as well. Eight of 19 patients (42%) had quantitative hippocampal asymmetry, and inadequate memory was observed from one hemisphere in 8 patients (35%).

Statistical comparisons of seizure types and origins
Type 1 seizure patterns were most commonly observed in patients whose seizures originated in the hippocampus. Type 2 and 3 patterns were noted mainly in patients with temporal neocortical seizures. These associations were statistically significant according to Fisher's exact \( t \) test (two-tailed). A significance level of \( p < 0.001 \) was obtained for a 2 \( \times \) 2 comparison of type 1 versus type 2, and \( p < 0.005 \), was obtained for type 1 versus type 3. There was no significant difference between the origins of type 2 and those of type 3 seizures: \( p < 0.75 \).

MRI and sodium amobarbital test results correlated with invasive monitoring
Fifty-seven patients had both intracranial monitoring and brain MRI scans. Unilateral hippocampal atrophy was noted in 15 (26%). Eight of the patients (53%) had seizures that originated in the ipsilateral hippocampus, and 7 of them (47%) had neocortical-onset seizures. Twelve patients had both hippocampal and temporal lobe atrophy on one side, but only 3 of these patients (25%) had seizures from the hippocampus; 9 (75%) had seizures of neocortical onset. Seventeen patients had normal brain MRIs, and 16 of these patients (94%) had neocortical seizures. Twenty-three of 47 patients had quantitative hippocampal asymmetry. About half of these had hippocampal onset seizures (11 of 23); the remainder had neocortical seizures. In our group of patients, hippocampal atrophy was not a specific indicator of hippocampal onset seizures. However, a normal MRI scan was highly correlated with seizures of temporal neocortical origin, and combined atrophy of the temporal lobe and hippocampus was reasonably supportive of the same.

In terms of sensitivity, ipsilateral hippocampal atrophy was more common in patients with hippocampal seizures (8 of 15 or 53%) than in those with neocortical temporal lobe seizures (7 of 43 or 17%). Atrophy of both the hippocampus and temporal lobe was evident in \( \sim 20\% \) of patients in both groups (3 and 9, respectively). MRI scans were normal in 16 patients (38%) with neocortical seizure onset but in only 1 patient (7%) with hippocampal onset. Quantitative hippocampal asymmetry was noted in 79% of those studied who had hippocampal seizures (11 of 14), but in only 36% of those with neocortical seizures (12 of 33).

All 58 patients who had invasive monitoring underwent intracarotid sodium amobarbital testing. Sixteen patients (28%) did not demonstrate adequate memory on the epileptogenic side. Three (19%) had hippocampal onset seizures, and 13 (81%) had neocortical seizures ipsilaterally. A failed Wada test was thus strongly suggestive of seizures of neocortical origin in our group but was not very sensitive for either seizure type. Only 20% of patients with hippocampal seizures and 30% of those with neocortical seizures failed the Wada test.

**DISCUSSION**

We have defined criteria for three major patterns of scalp EEG ictal discharge associated with partial seizures arising in the temporal lobe (Table 2). The type of ictal pattern can predict the location of seizure onset in many cases. Type 1 patterns are highly associated with seizures that originate in the hippocampus (86%). This association was statistically significant as compared with either type 2 or 3 seizure patterns. Only 1 patient with this ictal marker had intracerebral recordings that demonstrated a neocortical seizure onset, and this was localized to the most mesial temporal cortex. Patients with type 1 EEG seizures more often had ipsilateral hippocampal atrophy on MRI. When type 1 patterns occurred in patients with tumors, the neoplasm was always in the mesial temporal lobe. In contrast, patients with neocortical onset

**TABLE 1. Clinical correlates of seizure categorization**

<table>
<thead>
<tr>
<th>Seizure pattern</th>
<th>Type 1 (n = 17)</th>
<th>Type 2 (n = 53)</th>
<th>Type 3 (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depth eeg seizure onset</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hippocampal, n (%)</td>
<td>6/17 (86)</td>
<td>5/32 (16)</td>
<td>4/19 (21)</td>
</tr>
<tr>
<td>Neocortical, n (%)</td>
<td>1/7 (14)</td>
<td>27/32 (84)</td>
<td>15/19 (79)</td>
</tr>
<tr>
<td>Direct to surgery:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>? mesial, n (%)</td>
<td>6/17 (35)</td>
<td>8/53 (15)</td>
<td>1/23 (4)</td>
</tr>
<tr>
<td>Direct to surgery:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tumor, n (%)</td>
<td>4/17 (24)</td>
<td>10/53 (19)</td>
<td>3/23 (13)</td>
</tr>
<tr>
<td>MRI (atrophy)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hippocampal, n (%)</td>
<td>8/17 (47)</td>
<td>14/53 (26)</td>
<td>6/23 (26)</td>
</tr>
<tr>
<td>Hippocampal and temporal, n (%)</td>
<td>3/17 (18)</td>
<td>9/53 (17)</td>
<td>1/23 (4)</td>
</tr>
<tr>
<td>WADA failure, n (%)</td>
<td>7/17 (41)</td>
<td>12/51 (24)</td>
<td>8/23 (35)</td>
</tr>
</tbody>
</table>

MRI, magnetic resonance imaging.
FIG. 4. A type 2A seizure pattern with irregular 2- to 4-Hz right temporal and parasagittal ictal rhythm in which morphology and frequency change every few seconds.
FIG. 5. A type 2B seizure pattern with irregular 3- to 5-Hz right temporal rhythm that evolves in 15 s into a regular 7-Hz temporal ictal pattern.
FIG. 6. A type 2C seizure pattern with run of periodic, 2- to 3-Hz left temporal sharp waves followed in 13 s by a 6- to 7-Hz temporal ictal rhythm.
FIG. 7. Ictal onset potentials at the beginning of type 2C seizures in 4 patients. The pattern of ictal onset can range from a short burst of repetitive potentials (bottom left) to a slow, periodic pattern (bottom right). More sinusoidal rhythms evolved later in these seizures, as shown in Fig. 6.

Seizures, almost without exception, did not have type 1 patterns. On the other hand, the type 1 seizure pattern was not a particularly sensitive indicator of mesial temporal lobe seizures. Although 40% of the patients with verified seizure onsets in the hippocampus had a type 1 scalp pattern, 33 and 27% of them had type 2 and type 3 ictal EEG patterns, respectively.

Type 2 ictal patterns are a very specific indicator of neocortical onset seizures: Eighty-four percent of patients in this category and every patient with a type 2C pattern had seizures arising from temporal neocortex. This association between ictal pattern and anatomical origin was also statistically significant when type 2 EEG seizures were compared with type 1. The type 2 pattern was also reasonably sensitive, since almost two thirds of all patients with documented neocortical onset seizures had such an ictal EEG marker. Patients with nonmesial temporal lobe tumors, who were not monitored invasively but for whom cortical seizures would be most reasonable, also had predominantly type 2 ictal discharges. The type 3 seizure pattern is reasonably specific for seizures beginning in neocortex onset seizures as well (79%), although it is not very sensitive (35%).
TEMPORAL LOB ICTAL EEG PATTERNS

TABLE 2. Scalp EEG characteristics of temporal lobe seizures

<table>
<thead>
<tr>
<th>Type of temporal lobe seizures</th>
<th>Description of earliest ictal rhythms</th>
<th>Most likely origin</th>
</tr>
</thead>
<tbody>
<tr>
<td>1A</td>
<td>Inferotemporal rhythm of 5-9 Hz that is regular for at least 5 s and often longer</td>
<td>Hippocampus</td>
</tr>
<tr>
<td>1B</td>
<td>Vertex rhythm of 5-9 Hz that is regular for at least 5 s</td>
<td>Hippocampus</td>
</tr>
<tr>
<td>1C</td>
<td>Seizure rhythm type 1B followed by type 1A</td>
<td>Hippocampus</td>
</tr>
<tr>
<td>2A</td>
<td>Temporal and/or frontocentral rhythm of 2-5 Hz that is irregular or regular for only brief periods</td>
<td>Temporal neocortex</td>
</tr>
<tr>
<td>2B</td>
<td>Seizure type 2A followed by a regular, inferotemporal, 5 to 9 Hz rhythm (type 1A)</td>
<td>Temporal neocortex</td>
</tr>
<tr>
<td>2C</td>
<td>Seizure type 2A or 2B preceded by irregular or repetitive sharp or slow waves</td>
<td>Temporal neocortex</td>
</tr>
<tr>
<td>3</td>
<td>Unilateral or diffuse arrhythmic change in background</td>
<td>Unilateral or diffuse arrhythmic change in background</td>
</tr>
</tbody>
</table>

Therefore, a major benefit offered by our EEG classification of scalp-recorded ictal EEG patterns is significant specificity, i.e., a particular pattern highly associated with whether seizures originate in the hippocampus or temporal neocortex. This information can be important in presurgical evaluations in which the principal issue is localizing seizure onsets based on multiple lines of evidence. Other noninvasive tests frequently used in this regard did not have as much specificity in differentiating these two seizure origins. Unilateral hippocampal atrophy is purported to be a good indicator of mesial temporal lobe epilepsy (20), but only half of our patients in whom this finding was demonstrated on MRI had seizures that originated in the hippocampus. Combined hippocampal and temporal lobe atrophy or, in particular, a normal MRI scan had greater specificity in our series, but only for neocortical seizures. The same was true of a unilateral Wada test failure, which was a specific, but relatively insensitive, indicator of neocortical-onset seizures.

Fifteen of our patients were offered standard temporal lobectomy without invasive monitoring because a diagnosis of mesial temporal lobe epilepsy was reasonable, based on a variety of convergent clinical data other than EEG seizure pattern. It is reassuring that patients with type 1 seizures were more than twice as likely to be in this group. We do not assume, however, that all these patients who bypassed intracranial recording had hippocampal onset seizures, nor were they included in statistical analyses concerning seizure origin. Indeed, our thesis requires us to predict that surgical failures are more likely to occur in patients in this group who had type 2 and 3 seizures, because their seizures were not, or at least were not solely, of hippocampal origin.

Before our study was conducted, the EEG character of partial seizures had been described by various groups of investigators (5-9). Blume et al. (9) divided the EEG morphology of partial seizures in 66 patients into two basic patterns: sinusoidal waves and repetitive epileptiform potentials. They reported that both patterns occurred with almost equal frequency at seizure onset. Most seizures evolved in pattern or frequency, and the changes could occur in either direction. Three fourths of the partial seizures they studied had <8-Hz frequency and, in general, slower frequencies were more pronounced in the later stages of a seizure. Although these results laid a foundation for later investigations of the ictal EEG, few clinical correlations were provided.

More recently, Risinger et al. (10) tested the reliability and accuracy of scalp ictal EEG, which included sphenoidal electrodes, as a predictor of seizure localization in 110 patients with suspected temporal lobe epilepsy who subsequently underwent intracerebral EEG monitoring. They defined three ictal patterns: “initial focal,” in which a unilateral >5-Hz temporal or sphenoidal rhythm was the first discernible ictal activity or was evident <30 s after seizure onset; “delayed focal,” in which a similar >5-Hz rhythm evolved with a >30-s latency; and “nonlocalizing,” in which only slower, more diffuse, or no clearly defined rhythms were recorded. Half the patients had focal and half had nonlocalizing seizure patterns. Intracerebral recordings from depth electrodes confirmed seizure onset from the mesial temporal lobe in 86, 79, and 64% for initial focal, delayed focal, and nonlocalizing groups, respectively. What was not well defined by their seizure categorization was an ictal pattern that was poorly associated with mesial temporal lobe seizures or was highly associated with neocortical seizures.

Our results are in accord with those of Risinger et al. (10) on two major points: an early >5-Hz lateralized ictal rhythm is highly predictive of mesial temporal lobe epilepsy, and <5-Hz lateralized seizure frequency is less likely to be associated with a mesial temporal seizure onset. We depart from their conclusion that the delayed focal pattern is almost as accurate as the initial focal pattern in predicting a mesial temporal epileptogenic focus. Delayed development of a lateralized theta rhythm in our pa-
tients was most commonly observed later in the course of type 2B seizure patterns, which were associated predominantly with neocortical onsets. In general, our results support the concept that it is the initial ictal rhythm that is more predictive of seizure origin than any later developments in the ictal discharge. Other researchers have reported that scalp-recorded seizure patterns exhibiting high-frequency rhythms are a sign of temporal neocortical origin (3,21) because high-frequency discharges often indicate the ictal onset region in intracranial recordings (22). Our data clearly do not support this. Indeed, we noted that low frequencies characterized the scalp-recorded onset of most temporal lobe neocortical seizures. When we have observed beta-frequency seizure discharges from scalp electrodes on wide bandpass reviews, their origin has been from extratemporal neocortex (J. S. Ebersole and S. V. Pacia, unpublished observations).

More detailed comparisons with previous studies are difficult to make for a variety of reasons. Most investigators have used traditional EEG printouts in bipolar montages with standard filter settings. We believe that seizure pattern classification is more accurate and easier when supplementary inferotemporal electrodes are used and off-line editing is performed to allow appropriate manipulations of montages, gains, and filters. Although the frequency and evolution of seizure rhythms were usually portrayed adequately in standard bipolar montages, we noted that changes in discharge morphology and distribution were better perceived in a referential recording. Certain ictal phenomena that are key to our classification scheme were not recognized in previous attempts to categorize partial seizures. These include the positive vertex theta rhythm of type 1B seizures; the irregular, slow, nonstationary rhythm of type 2A seizures; and the ictal onset potentials of type 2C seizures. Validation of the origin of seizures by intracranial EEG was obtained in previous studies from depth electrodes only and not from a combination of depth and subdural recordings such as we used. Finally, and perhaps most important, the patient populations among the various studies were probably different in important ways. We believe that our present series (1989–1993) contains a much higher percentage of patients with neocortical temporal lobe epilepsy than do most earlier patient series, including our own. Our ability to define better the scalp EEG patterns associated with neocortical temporal seizures was actually dependent on our having a large number of such cases.

The cerebral EEG substrates of these various seizure patterns will be the subject of a separate report (23). Simultaneous intracranial and scalp recordings were obtained from 24 of the 58 patients in this study who had invasive monitoring. Clearly, the appearance of an ictal rhythm at the scalp is dependent on a variety of factors, such as the amplitude and synchrony of cerebral seizure discharge and the geometry of the involved cortex, including its area, location, and orientation. Scalp ictal patterns that superficially appear very different, such as types 1A and 1B, can result from seizures that originate in the same cerebral region, if subsequent propagation activates different cortical areas. In the example cited, different relative contributions to the scalp field from lateral and basal temporal cortex explains the difference in seizure patterns, as has been demonstrated in temporal lobe spikes (24–26). The knowledge we gained from simultaneous intracranial and scalp EEG recordings gave us more confidence in the associations between ictal patterns and seizure origins that were observed in patients without simultaneous data.

We specifically decided not to relate EEG seizure patterns to ictal behavior. Analysis of behavior during seizures has already been demonstrated to be useful in localization (27). What the present results show is that analysis of the ictal EEG, without reference to patient behavior, can also localize seizure onsets. Likewise, we chose not to use surgical outcome to measure the accuracy of seizure categorization. Instead we asked a more direct question: Can classification of scalp ictal EEG patterns predict seizure origin as determined by intracranial EEG? That the answer is yes is clinically significant, because many epileptologists consider seizure localization by intracranial EEG the gold standard for planning surgical resections and predicting probable outcomes. We believe that the major clinical utility of our EEG seizure pattern classification is that it is capable of differentiating temporal neocortical from hippocampal onsets. We encourage other investigators to confirm our results, preferably in a blinded study. We are confident that analysis of scalp EEG ictal patterns will prove to be an aid in the presurgical evaluation of patients with temporal lobe epilepsy.

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