Adding or repositioning intracranial electrodes during presurgical assessment of neocortical epilepsy: electrographic seizure pattern and surgical outcome

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Object. The aim of this study was to investigate changes in electroencephalography (EEG) patterns obtained from added or repositioned electrodes after those initially implanted had failed to indicate the true local ictal onset zone. The authors focused on the following matters: rationale for adding or repositioning electrodes, topographic and frequency characteristics of ictal onset before and after adding or repositioning electrodes, the effect of the procedures, and the relationship between changes in intracranial EEG onset patterns and surgical outcomes.

Methods. Of 183 patients with intracranial recordings, 18 experienced repositioning of existing or implanting of additional electrodes 7 or 10 days later. All patients underwent resection and were followed up for more than 1 year. In particular, the relationship between surgical outcome and distribution/frequency of intracranial seizure onset was analyzed. Results of noninvasive presurgical evaluations in patients who had undergone single and double invasive studies were also evaluated.

By adding or repositioning electrodes, a new ictal onset zone was revealed in 13 patients. In another four, the second evaluation led to a change in defining the resection margin. Ictal onset in the partially sampled area, simultaneous or independent onset in two separate areas, and onset in the distal end of the electrode strip or grid were common reasons for failing to localize the ictal onset zone during the initial evaluation. Seven of 11 patients who were ultimately found to have a focal ictal onset zone on the second evaluation became seizure free after the operation. Only one of six patients with a regional ictal onset zone identified on the second evaluation became seizure free. There was no relationship between the frequency of the ictal rhythm and surgical outcome. Note, however, that surgical outcome was more favorable in patients who had undergone a single invasive study than in those who had undergone double invasive studies. The patients who needed a second evaluation had less localizing information and less consistent results on presurgical evaluations. When comparing nonlesional cases, surgical outcomes were not significantly different among patients with a single invasive study and those with double invasive studies. No additional morbidity or death occurred during the second study.

Conclusions. The addition or reposition of intracranial electrodes with a short-term interval should be considered in selected patients. Spatial restriction of the ictal onset rhythm identified on repeated evaluation is the most important predictor of a good surgical outcome.

Key Words • neocortical epilepsy • surgical outcome

Accurate localization of the ictal onset zone by intracranial EEG is the most sensitive and important method in the successful surgical treatment of epilepsy.1,4,19,22,24,29,30 During presurgical evaluation of nonlesional neocortical epilepsy, intracranial monitoring is indispensable but may provide insufficient sampling.7,19,20 To obtain appropriate information during intracranial monitoring, many intracranial electrodes are needed. Nevertheless, because these electrodes cover only a limited portion of the brain, the true ictal onset zone can sometimes be missed,20,21,29 and the repositioning of or adding electrodes may be required.

A variety of electrographic seizure onset patterns are known.1,7,10,17,26 Note, however, that the first electrographic change in a seizure does not always indicate a true ictal onset zone, and some patterns represent a propagated phenomenon.10,17 It is important to compare electrographic seizures recorded at the local onset site and the remote propagated electrode site.17 Identifying the change in the ictal onset pattern by implanting additional electrodes can also be a useful method to clarify this issue. In selected patients in whom invasive monitoring initially failed to demonstrate the site of seizure origin, repeated examination by using intracranial electrodes enabled its successful localization.16 Interestingly, the relationship between the effectiveness of implantation of additional intracranial electrodes and changes in the electrographic seizure onset patterns has not been described.

We studied patients in whom the repositioning of existing, or the insertion of additional, intracranial electrodes 7
Intracranial electrodes arranged in grids and strips were also performed using the same types of electrodes such as grid and strip. The rationale for repositioning or implanting intracranial electrodes were recorded for later analysis. All seizures were reviewed visually by a sole observer blinded to the study. An “intracranial ictal onset” was defined as “the first sustained rhythmic change in EEG classified as low-voltage fast beta activity, rhythmic spikes in the alpha or theta range, rhythmic sinusoidal waves in the theta range; SSMA = supplementary sensorimotor area; T = temporal; TP = temporoparietal; WNL = within normal limits; 2 GTCS = secondarily generalized tonic-clonic seizure; * = leading to.

† Indicates lesion location.
‡ Indicates location of hyperperfused area.
§ Indicates location of hypometabolic zone.
|| Indicates location of initial ictal rhythm.

Clinical Material and Methods

Patient Selection

We retrospectively reviewed all consecutive chronic intracranial EEG recordings performed at Seoul National University Hospital between January 1994 and March 2000. Of 183 patients, 18 underwent a second invasive study involving the repositioning of existing, or the implantation of additional, intracranial electrodes. This second study was performed 7 or 10 days after the initial invasive study. The remaining 165 patients underwent only a single invasive study. All patients experienced resection and were followed up for more than 1 year. Results of the initial presurgical evaluation, including MR imaging, FDG-PET studies, interictal and ictal SPECT scanning if available, and long-term scalp video-EEG monitoring, were analyzed. Intracranial electrodes arranged in grids and strips were used in various combinations. The second evaluation was also performed using the same types of electrodes such as grid and strip. The rationales for repositioning or implanting intracranial electrodes were recorded for later analysis.

Seizure Analysis

All seizures were reviewed visually by a sole observer blinded to the study. An “intracranial ictal onset” was defined as “the first sustained rhythmic change in EEG clearly differentiated from background EEG and interictal waves.” The “ictal onset zone” was defined as “the area covered by intracranial electrodes with intracranial ictal onset.” The onset frequency was characterized in traditional EEG bands such as beta, alpha, theta, and delta. Considering seizure onset frequency and waveform, seizure onsets were classified as low-voltage fast beta activity, rhythmic spikes in the alpha or theta range, rhythmic sinusoidal waves in the theta or delta range, or semirhythmic delta activity.

The distribution of seizure onset was categorized as focal, regional, or widespread. A “focal onset” was defined as one “involving fewer than five adjacent electrode contacts,” and “regional onset” as “involving five or more adjacent contacts.” A “widespread ictal onset zone” was defined as “an ictal rhythm at onset involving more than 20 adjacent contacts.” Ictal symptoms were described using semiological classification.

Invasive Studies

The location of electrode grids and strips was determined based on clinical, neurophysiological, imaging, and EEG.
data obtained during noninvasive evaluation. In patients with neocortical epilepsy and a lesion demonstrated on MR imaging, subdural electrodes were placed over a lesion and the surrounding area including eloquent cortex. In patients with extratemporal epilepsy and no lesion revealed on MR imaging, the locations of subdural arrays were guided by the results of ictal scalp EEG, FDG-PET, and ictal SPECT studies and symptomatology. A more widespread coverage of the neocortex was conducted in these nonlesional cases. In patients with TLE, ictal EEG was performed in both lateral and mesial temporal areas by using grids and multiple strips to determine from which site seizure onset originated. Furthermore, in patients with mesial TLE in whom results of presurgical evaluations did not reveal which temporal lobe was the epileptogenic zone, electrodes were implanted, either bilateral depth electrodes to the mesial structures or multiple strips reaching to bilateral parahippocampal gyri. If a discrepancy existed between various data or if the localization of the focus was not sufficient to proceed with resection (for example, in a patient who had a structural lesion on MR imaging or a focal hypometabolic zone on FDG-PET scanning, but whose scalp ictal EEG or ictal SPECT studies demonstrated a seizure onset elsewhere), the electrode strips and grids would cover both of these areas. Lateral neocortex was usually covered by grids with additional strips, depending on the situation. Mesial temporal, orbitofrontal, and interhemispheric areas were examined using multiple strips. Commonly used grids included 8 × 8, 8 × 4, and 5 × 4 arrays. The 4 × 1, 6 × 1, and 8 × 1 strips were selected, depending on the situation.

Electrode repositioning was based on the results of the first invasive study. When ictal onset occurred in a strip situated in an area without enough coverage, additional strips were inserted or the strip was replaced with a grid. When ictal onset occurred in the distal end of a strip or grid, additional grids and strips were placed or the original electrodes were moved to an area outside the distal end. For example, if grids/strips were placed in the temporal lobe and ictal onset occurred in the posterior temporal area, additional grids and strips were inserted or original electrodes were moved to cover occipital and parietal areas based on the patterns of the propagating ictal rhythm. When encountering two separate, widespread ictal onsets or clinical onset preceding ictal onset, it is reasonable to think that only propagated rhythm was captured. In these patients we inserted additional grids and strips in the surrounding area outside the region covered during the initial evaluation. For example, when we sampled the frontal area and found two ictal onset areas, additional grids and strips were inserted to sample centroparietal and temporal areas.

Surgery and Follow Up

The margin of resection was determined based on the ictal onset zone and the results of electrical mapping. Surgical outcome was classified according to the classification of Engel and colleagues, and all resected tissue was subjected to pathological diagnosis.

Results

Clinical data in the patients who underwent double invasive studies are summarized in Table 1. Twelve patients were male and six were female. Results of MR imaging demonstrated no lesion in 16 patients. One patient had bilateral hippocampal sclerosis so that ictal rhythms did not start. Another patient had focal frontal atrophy, but his intracranial ictal rhythm originated from the parietal lobe. Based on the resected lobe, four patients had frontal lobe epilepsy, 11 had parietal lobe epilepsy, and three had lateral TLE. A second intracranial invasive study was undertaken 7 days after the initial evaluation in 16 patients, and 10 days after in two patients. The mean period of electrode implantation, including the first and the second studies, was 14 days (range 9–18 days).

The mean number of intracranial electrodes in the first and the second evaluation was 64 (range 36–114 electrodes) and 77 (36–122 electrodes), respectively (Table 2). The addition or reposition of electrodes revealed a new ictal onset zone in 13 patients. In another four patients, results of the second evaluation led to a change in the resection margin. We made no change in one case.

The initial evaluation failed to localize the ictal onset zone for the following reasons, which were the rationale for the second evaluation (Table 2). 1) Ictal onset occurred in the partially sampled area (five patients). 2) Ictal onset occurred in the distal end of the electrode strip or grid (four patients). 3) Simultaneous or independent ictal onset occurred in two separate areas (five patients). 4) There was a widespread ictal onset zone (two patients). 5) Clinical onset of the seizure occurred before any EEG change (two patients). The ictal onset zone in the partially sampled area was defined when the initial ictal rhythm occurred in the sparsely covered area next to a strip.

Of eight patients with regional ictal onset in the initial evaluation, six had focal ictal onset in the second study (Table 2 and Fig. 1). Four of them were seizure free after the operation. In four of five patients with two separate ictal onset zones in the first study, the second study revealed regional ictal onset zones and only one of the patients was seizure free after resection. Three patients with focal ictal onset zones during the first and second invasive studies were seizure free after resection. Four patients with only a change in the resection margin at the time of surgery—a decision based on results of the second study—were also seizure free. Spatial restriction of ictal rhythm was significantly correlated with a good surgical outcome (p < 0.05). Seven of 11 patients who were ultimately found to have a focal ictal onset zone during the second evaluation became seizure free after the operation. Another patient who had an unchanged focal ictal onset zone during the first and second studies was also seizure free after resection. Note, however, that only one of six patients with a regional ictal onset zone identified on the second evaluation became seizure free. The focal ictal onset zone was significantly correlated with a good surgical outcome (Fisher exact test, p = 0.0068). The frequency of ictal rhythm changed in the second study compared with the frequency in the first study in four patients. No ictal rhythms at the newly found ictal onset zone during the second study became slower compared with its frequency in the first. There was no clear relationship between the frequency of ictal rhythm and surgical outcome (Fisher exact test, p = 0.307; Table 3). There was no additional morbidity or death associated with the second invasive study. No patient demonstrated parietal lobe syndrome after resection. We tested variable parietal lobe functions by using
electrical cortical stimulation to avoid Gerstmann syndrome or constructional apraxia. Surgical outcome was more favorable in the patients who had undergone a single invasive study (p = 0.034; Table 4). Parietal lobe epilepsy occurred in only 16 patients (9.7%) with a single invasive study. Ten of these 16 were seizure free after surgery. Patients who needed a second evaluation had less localizing information and fewer concordant results on presurgical evaluation (Table 5), compared with patients in whom only one invasive study had been conducted. The results of presurgical evaluations, except ictal SPECT scanning, were localizing more often in patients who had undergone a single invasive study (p < 0.001).

When comparing nonlesional cases, surgical outcome was not significantly different between patients with a single invasive study and those with double invasive studies (p = 0.37; Table 6). Three of six patients with nonlesional PLE who had undergone only a single invasive study were seizure free after the operation. This result was comparable to the surgical outcome in patients with PLE who had un-
Repositioning of intracranial electrodes

**Case Number 18**

This 17-year-old female patient was admitted for presurgical evaluation of intractable epilepsy. Her seizures had started at 6 years of age. She had no perinatal problem or family history of epilepsy. Her habitual seizure was characterized by a brief loss of consciousness for 10 to 15 seconds. She experienced secondarily generalized tonic-clonic seizures 5 or 6 times a year. She also occasionally lost control of her standing ability and fell to the ground because of a seizure. She had an uncertain feeling immediately before losing consciousness. Her seizures were medically intractable and a brief loss of consciousness occurred 5 or 6 times a day despite combination therapy of lamotrigine, valproic acid, topiramate, and carbamazepine. Her interictal EEG demonstrated frequent independent spikes on T5 and F3 electrodes as well as occasional generalized spike-and-waves (secondary bilateral synchrony). Results of brain MR imaging were normal. An FDG-PET scan revealed left anterior temporal hypometabolism (Fig. 2). Her visual field was normal. Long-term scalp video-EEG monitoring was conducted for 3 days. A total of 16 seizures were recorded. Her typical seizure involved a brief loss of consciousness with mild bimanual automatism. The mean duration of each seizure was 13 seconds. She experienced one secondarily generalized tonic-clonic seizure during monitoring. Ictal onset was identified on the T5 electrode as periodic spikes followed by rhythmic alpha activity (Fig. 3). Based on results of the noninvasive presurgical evaluation, she was diagnosed as having posterolateral TLE. One 8 × 4 grid and multiple strips were placed on the left posterolateral and inferior temporal areas. An additional 5 × 4 grid was inserted on the left centroparietal area, and two strips on the left frontal region (Fig. 4). Low-voltage fast beta activity was identified as an ictal onset on the distal end of the upper grid. The ictal rhythm rapidly spread to the upper part of the inferior grid. We considered the possibility of the presence of a true ictal onset zone outside the area covered by the 5 × 4 grid. We replaced the upper grid with a new 8 × 4 grid to cover the posterior parietal cortex (with two strips

**Discussion**

The second invasive study was performed 7 or 10 days after the initial examination. The issue of when the second evaluation with intracranial electrodes should be conducted has not been clearly addressed. A repeated evaluation by using grid and depth electrodes is usually performed after at least a few months.18 We used only grids and strips for the evaluation of neocortical epilepsy, and no additional morbidity occurred following this short-term repeated evaluation. The reasons for the second invasive study have been described in Results and Table 2. There was a high incidence of PLE in the patients who needed a second evaluation. Excluding four patients with poor outcomes, at least seven patients had PLE. Four patients with frontalictal rhythms and two with temporal ictal rhythms on scalp EEG were ultimately found to have parietal ictal onset zones. Pat-

**TABLE 3**

<table>
<thead>
<tr>
<th>Frequency of Ictal Rhythm</th>
<th>Surgical Outcome (no. of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
</tr>
<tr>
<td>β</td>
<td>6</td>
</tr>
<tr>
<td>α</td>
<td>1</td>
</tr>
<tr>
<td>θ</td>
<td>0</td>
</tr>
<tr>
<td>δ</td>
<td>2</td>
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**TABLE 4**

<table>
<thead>
<tr>
<th>Epileptic Syndrome</th>
<th>Surgical Outcome (no. of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
</tr>
<tr>
<td>PLE</td>
<td>31</td>
</tr>
<tr>
<td>lat TLE</td>
<td>43</td>
</tr>
<tr>
<td>PLE</td>
<td>10</td>
</tr>
<tr>
<td>OLE</td>
<td>14</td>
</tr>
<tr>
<td>med TLE</td>
<td>17</td>
</tr>
<tr>
<td>no surgery†</td>
<td>3</td>
</tr>
<tr>
<td>total</td>
<td>115</td>
</tr>
</tbody>
</table>

* med = medial; OLE = occipital lobe epilepsy. † Invasive study was performed, but resection was not because of the presence of eloquent cortex at the ictal focus.

on the medial parietal area; not shown in the figure). This showed a new focal ictal onset zone in the posterior parietal cortex. The patient underwent a focal neocortical resection of the posterior parietal cortex. She has been seizure free following the operation for 2.5 years.
rietal lobe epilepsy occurred in only 16 patients (9.7%) who had undergone a single invasive study. This unexpected location of an ictal onset zone in the parietal lobe in patients who had undergone double invasive studies was the impetus for a second evaluation. The role of electrocorticography has not yet been established in guiding the placement of grids and strips. Nevertheless, the use of electrocorticography to determine the position of electrode grids and strips during the first invasive study may obviate the need for a second evaluation. Further study is required to clarify this issue, however.

Surgical outcome was more favorable in patients who had undergone a single invasive study. Note, however, that one half of the patients who had undergone double invasive studies were also seizure free following surgery. Furthermore, the difference in surgical outcome was probably due to the difference in results of the presurgical evaluation. The most striking difference was the evidence of a structural lesion on MR images. One half of the patients (50.3%) who had undergone a single invasive study demonstrated a focal structural lesion on MR imaging, whereas no one who had undergone double invasive studies revealed a concordant structural lesion. The difference in localizing results on presurgical evaluations was also evident when a number of concordant results were compared. At least 77.6% of patients who had undergone a single invasive study had at least two concordant results on presurgical evaluations regarding the localization of seizure foci. In contrast, only five (27.8%) of 18 patients who had undergone double invasive studies had two concordant results (p < 0.001).

None of the patients with double invasive studies had more than two concordant results on presurgical evaluation. In patients whose MR imaging studies demonstrate no lesion, the spreading ictal rhythm can cause the mislocalization of a seizure focus and lead to inadequate placement of invasive electrodes. The lack of characteristic symptomatology and the poor localization of ictal EEG in patients with PLE can be the main reasons for the high incidence of PLE in patients who undergo double invasive studies. The surgical outcome in patients with PLE whose MR imaging results demonstrated no lesion was not significantly different between patients who had undergone a single invasive study and those who had undergone double invasive studies. Although the lack of definitive results on other presurgical evaluations indicating the location of the ictal focus may lead to the need for a second evaluation, careful repositioning or insertion of additional electrodes can make for a comparable surgical outcome.

Some of the localization-related epilepsy, particularly seizures originating from the posterior cerebral cortex, might have a multiple seizure-spread pattern.15,16,27,28 Five patients in our series seemed to have two separate ictal onset zones in the first evaluation; four of them had PLE. Three patients had two independent presumed ictal onset zones, and two patients showed simultaneous onset of ictal rhythms in two separate areas. We could locate the new foci

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TABLE 6
Surgical outcome in patients with no lesion demonstrated on MR imaging studies

<table>
<thead>
<tr>
<th>Surgical Outcome</th>
<th>Single Invasive Study</th>
<th>Double Invasive Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>seizure free</td>
<td>47</td>
<td>9</td>
</tr>
<tr>
<td>rare seizure</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td>worthwhile improvement</td>
<td>14</td>
<td>4</td>
</tr>
<tr>
<td>no change</td>
<td>14</td>
<td>5</td>
</tr>
<tr>
<td>no surgery</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>82</td>
<td>18</td>
</tr>
</tbody>
</table>

TABLE 8
Frequency of ictal rhythm in patients who underwent a single invasive study and surgical outcome

<table>
<thead>
<tr>
<th>Frequency (no. of patients)</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
<th>Surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>β</td>
<td>68</td>
<td>3</td>
<td>10</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>α</td>
<td>9</td>
<td>1</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>θ</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>δ</td>
<td>6</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>periodic spikes (1–2 Hz)</td>
<td>12</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>medial TLE*</td>
<td>16</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>total</td>
<td>115</td>
<td>7</td>
<td>20</td>
<td>20</td>
<td>3</td>
</tr>
</tbody>
</table>

* Frequency of the ictal rhythm of medial TLE was not analyzed because only the lateralization of the ictal focus was the main concern.

TABLE 7
Intracranial ictal onset patterns and surgical outcome in patients who underwent single or double invasive studies

<table>
<thead>
<tr>
<th>Surgical Outcome</th>
<th>Single Invasive Study</th>
<th>Double Invasive Study</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Focal Onset</td>
<td>Regional Onset</td>
</tr>
<tr>
<td>seizure free</td>
<td>57</td>
<td>43</td>
</tr>
<tr>
<td>rare seizure</td>
<td>1</td>
<td>5</td>
</tr>
<tr>
<td>worthwhile improvement</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td>no change</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td>no surgery</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>total</td>
<td>71</td>
<td>76</td>
</tr>
</tbody>
</table>

* Ictal onset patterns of medial TLE were not analyzed because only the lateralization of the ictal focus was the main concern in patients with medial TLE.
with the initial ictal rhythm spreading to these two separate areas. Regardless, surgical outcome in these patients was unsatisfactory for unknown reasons. An inability to find the true focal ictal onset zone might be the cause of this failure.

Low-voltage fast activity at seizure onset is reportedly associated with good surgical outcome.1,2,7,26 A slower-frequency ictal onset might mean a propagated electrographic pattern or a larger epileptogenic zone.7,10,17 Electrodes that record faster-frequency ictal discharges at seizure onset are believed to be closer to the seizure onset zone.8,9 Some researchers have argued that low-voltage localized beta activity is a marker of the site of seizure onset,20,22 but others have disputed this hypothesis.10,17,25 We found that the most common early ictal discharge in patients with neocortical epilepsy was low-amplitude beta activity. No ictal rhythm in the newly found ictal onset zone in the second study became slower compared with that in the first study. We could not find a clear relationship between the frequency of ictal rhythm and surgical outcome, however. Furthermore, some of the propagation rhythms confirmed on the first and second invasive studies also had low-amplitude beta activity. Such activity alone would not allow us to locate a true ictal onset zone. These findings can be explained by the hypothesis that a subset of remote propagated electrographic seizures represent independent initiation of secondary electrographic seizures indistinguishable from those at the local onset site.17

Spatial restriction has been said to be a characteristic of an intracranial ictal onset close to a true ictal onset zone. In contrast, regional onset may imply volume conduction or propagation from a distant generator.5,13,19 Some authors have also argued that focal ictal onset predicts good surgical outcome in patients with TLE.11,14 Our results support this concept. Spatial restriction of intracranial ictal rhythm was significantly correlated with good surgical outcome in patients who had undergone double invasive studies as well

**Fig. 2.** Two FDG-PET scans revealing the left anterior temporal hypometabolism. R = right.

**Fig. 3.** Tracings from ictal scalp EEG showing the initial periodic spikes maximally on T5 (A), followed by the rhythmic spikes within the alpha range (B). Arrows indicate the initiation of ictal rhythm.
as in those who had undergone a single study. Seven of 11 patients who were ultimately found to have a new focal ictal onset zone during the second evaluation were seizure free after the operation. Only one of six patients with regional ictal onset became seizure free, however. Furthermore, of eight patients with regional ictal onset in the initial evaluation, six had focal ictal onset in the second invasive study, four of whom were seizure free after the operation.

Although the spatial restriction of ictal rhythm was significantly correlated with good surgical outcome in the patients who had undergone a single invasive study as well as in those who had undergone double invasive studies, and surgical outcome was more favorable in patients who had undergone a single invasive study, the proportion of the focal onset pattern was not significantly different between these two groups. The presence of a structural lesion or the concordant results on presurgical evaluation seems more important in predicting surgical outcome than the ictal onset pattern.

Increasing the number of electrodes in cases of partial epilepsy might result in an increased chance of recording a focal onset. Because increasing the number of electrodes...
Repositioning of intracranial electrodes

can be related to increasing morbidity, however, it is not possible to increase the number without limitation. Careful placement of electrodes based on presurgical evaluation and additional coverage of electrodes on adjacent areas should be considered important.

Conclusions

Neocortical epilepsy in patients whose MR imaging results demonstrate no lesion or with fewer concordant results on presurgical evaluation can cause insufficient coverage of the true ictal focus and lead to the need for a second evaluation. Adding or repositioning intracranial electrodes with a short-term interval in patients who had unsuccessful initial intracranial evaluations allowed for a good surgical outcome in one half of them. Spatial restriction of the ictal rhythm identified on repeated evaluation is the most important predictor of a good surgical outcome. There was no additional morbidity associated with the second evaluation. Such results support the consideration of adding or repositioning of intracranial electrodes with a short-term interval in selected patients.

References


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