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Temporal and Spatial Characteristics of Intracerebral Seizure Propagation: Predictive Value in Surgery for Temporal Lobe Epilepsy

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Summary: We examined the prognostic value of spatial and temporal characteristics of intracerebral propagation of seizures during temporal lobe epilepsy (TLE) surgery. Seven TLE patients resistant to standard anterotemporal lobectomy who had no known causes of resistance [e.g., extratemporal (ET), lesions, multifocal epilepsy] were matched with 7 seizure-free patients and 7 others who were almost seizure-free after operation. Intracerebral ictal propagation pathways were not different in the three groups. Propagation was multidirectional, most frequently to the frontal lobes and sometimes to the contralateral temporal lobe (CTL). ET propagation delays were significantly shorter in resistant patients than in markedly improved patients. The resistant group also had more frequent propagation delays <1.0 s, but propagation times >1.0 s were equally likely in all groups. The extent of ET propagation and frequency of focal onsets were not different among the groups. Results suggest that very short propagation times predict reduced efficacy of operation, and that long propagation times are not related to surgical success. **Key Words:** Temporal lobe epilepsy—Implant electrodes—Electroencephalography—Neurosurgery—Prognosis.

In medically refractory temporal lobe epilepsy (TLE), onset of intracerebral ictal activity is a very good indicator of the location of an epileptogenic region and generally leads to excellent postoperative outcome. About 80% of patients with intractable TLE are markedly improved by standard anterotemporal lobectomy (Engel's classes I and II) (Engel, 1987). However, some patients do not show as much improvement as expected from presumed removal of an epileptogenic focus (Engel's classes III and IV). One possible cause for these mitigated successes may be that the region of ictal onset is an insufficient characterization of the epileptogenic features of the patient's brain at the time of evaluation. Other characteristics of the intracerebral EEG (stereo-EEG, SEEG) may provide more clues to the location or extent of epileptogenic tissue or the pathophysiology of a patient's seizures and ultimately help predict resistance to neurosurgery.

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SEEG factors related to poor surgical prognosis in TLE have already been reported: short propagation times to contralateral temporal lobe (Lieb et al., 1986), nonfocal ictal onset or characteristics of multifocality (Lieb et al., 1981) and bitemporal ictal onset (So et al., 1989). However, in the only prognostic study of temporal seizure propagation times (Lieb et al., 1986) activity in the frontal lobes was not measured. The frontal lobes may contain additional foci which can affect postoperative outcome as well as the characteristics of seizure propagation. Our observations also suggested that frontal regions are more frequently invaded than is the contralateral temporal lobe (CTL) in ictal propagation.

We examined the prognostic value of temporal and spatial characteristics of SEEG seizure propagation in TLE while minimizing the confounding effects of other resistance factors. Variables examined included seizure propagation sequences, propagation times, frequency of focal ictal onsets, and extent of propagation outside the epileptogenic temporal lobe. We selected patients with only one epileptic focus who were resistant to neurosurgery and compared them with patients who were seizure-free or almost seizure-free.

PATIENTS AND METHODS

Patient selection

Among 23 patients with TLE resistant to neurosurgery (Engel's classes III and IV, seizure reduction <90%), operated between 1973 and 1990, 7 patients were selected because they had no known causes of resistance, such as potentially epileptogenic extratemporal (ET) lesions, bilateral temporal(or multifocal) SEEG ictal onsets, or nonstandard temporal lobectomy (resection limited to the lesion and surrounding cortex). Two of these patients became resistant several years after operation (patient 6 after 7 years of class IIA and patient 3 after 10 years of class IB).

The reasons for excluding 16 other patients were potentially epileptogenic ET lesion (n = 3), bitemporal or multifocal seizures in SEEG (n = 9), nonstandard operation (n = 2), and questionable temporal ictal onset during SEEG review (n = 2).

The 7 resistant patients were matched with 7 seizure-free patients (Engel's class IA) and with 7 patients with intermediate outcome who were almost seizure-free (Engel's classes IB,C,D, and II). The matching criteria were side of lobectomy, hemispheric dominance for language, age at onset of epilepsy, preoperative duration of epilepsy and, when appropriate, presence of a tumor in the lobectomy.

The resistant patients had only one region of SEEG ictal onset and even if some seizures with diffuse onset could be observed most seizures in all patients clearly had temporal onset. In 3 seizure-free patients (patients 9, 12, and 14), we observed one contralateral mesiotemporal seizure. Because they were the only patients we could find to match the corresponding resistant patients and were seizure-free, we decided to include them in the study.

All patients had medically refractory complex partial seizures (CPS) and underwent standard anterotemporal lobectomy involving resection of lateral tissue (average 6 cm from the temporal tip) and mesial tissue (amygdala and anterior hippocampus (average 5 cm from the temporal tip) and sparing of Heschl's gyri. To be selected, the patients had to have undergone a complete preoperative investigation, including intracerebral recording of seizures with temporal and frontal electrodes. The sample investigated comprised 15 men and 6 women.

Preoperative evaluation

Preoperative investigation was aimed at evaluating the appropriateness of neurosurgery and the location of the epileptogenic focus if possible. This investigation was based on clinical examination data, neuroimaging [computed tomography (CT) scan and, recently, magnetic resonance imaging (MRI)], neuropsychological data, amytal test, long-term video-EEG and video-SEEG recordings, and intracerebral electrical stimulation (Saint-Hilaire, 1987). SEEG was performed through electrodes implanted horizontally in a lateromesial axis with a stereotaxic frame (Bouvier et al., 1987). The electrodes were 6–10 stainless-steel contacts spaced every 5 mm and mounted on a flexible plastic probe.

The intracerebral investigation was comparable among the groups. The sites implanted were not identical for all patients, but always included symmetrical exploration of both right and left temporal and frontal lobes with 5–11 electrodes (except for patients 3 and 15 in whom only the frontal lobe on the epileptogenic side was explored). The temporal lobe was explored at three anteroposterior levels: amygdala and anterior and posterior hippocampus with corresponding lateral cortex. On the side of the focus, electrodes were generally placed at the three levels; on the opposite side, generally only the anterior hippocampus was implanted.

In the frontal lobes, electrodes were most frequently implanted in anterior cingulate gyrus and corresponding lateral cortex. The supplementary motor area and corresponding lateral cortex were also explored in several patients.

Orbitofrontal cortex was explored in only two markedly improved patients (patients 12 and 19), whereas the parietal lobe was explored at the level of posterior cingulate gyrus in 3 patients (patients 3, 9, and 19). Because patient 3 had two frontal electrodes but recordings of these electrodes were not retrieved for the review, this patient was not retained in analyses of propagation pathways and extent of propagation.

Seizure analysis

Individual ictal SEEG records were reviewed by 2 neurologists (J.-M. S-H. and C.A.) without knowledge of the patient's electrode positions and postoperative outcome. In case of disagreement, a consensus was reached by the 2 reviewers. We considered occurrence of rhythmic spikes or sharp waves that built progressively (often of low voltage at the beginning) or occurrence of high-amplitude spikes that evolved into sustained rhythmic activity clearly distinct from background EEG to be ictal onset activity. Of the 162 seizures initially analyzed in preoperative evaluation, we retrieved and reviewed 132 (SEEG and video), 60 for resistant patients, 72 for the 14 markedly improved patients (mean of six seizures per patient, range 2–18). All seizure types (clinical or subclinical) were included.

Propagation time to the first site outside of the epileptogenic temporal lobe (hereafter called ET

sites) was computed as the delay between time of seizure onset and time at which the first ET site was invaded by ictal activity. Seizure onset was considered focal when it was localized to only one pair of contiguous contacts. Purely temporal seizures were seizures without ET diffusion and were not taken into account in analyses of ictal propagation times. The extent of ictal ET propagation was computed as the percentage of implanted ET lobes invaded by ictal activity. Only frontal and temporal electrodes were considered in analyses of propagation extent because of the few parietal electrodes implanted. Statistical analyses were performed with chi-square test (Yates correction), Fisher's exact test, or nonparametric univariate analysis of variance (ANOVA).

RESULTS

Characteristics of the different groups

Table 1 summarizes some of the important characteristics of patients in the different groups. The groups did not differ with regard to age of seizure onset, preoperative duration of epilepsy, duration of postoperative follow-up, seizure frequency, and size of lobectomy. Mean IQs of surgically resistant patients were slightly lower than that of markedly improved patients (84 vs. 102, $p < 0.1$). No cerebral CT scan abnormalities were observed except in patients with tumors. Postoperative cerebral MRI in patients 1, 3, 6, and 8 showed no lesion outside the resected temporal lobe.

Interictal epileptiform EEG abnormalities of all resistant patients generally were unilateral, but could become synchronous bilateral generalized spikes (patients 1, 4, and 7). Three markedly improved patients had independent bilateral abnormalities, clearly predominant on the operated side (patients 14, 18 and 21). Pathologic examination showed hippocampal sclerosis or gliosis in 6 of 11 markedly improved and 1 of 7 resistant patients in whom hippocampal tissue was available for analysis ($p > 0.2$).

Ictal propagation pathways

Twenty-three percent of seizures showed no propagation outside the epileptogenic temporal lobe, and this percentage did not differ among the groups ($p > 0.2$). Seven of the 14 markedly improved patients had purely temporal seizures, as compared with 2 of the 7 resistant patients ($p > 0.6$). In seizures that did show ET propagation, we examined the propagation pathways of the ictal discharge outside the epileptogenic temporal lobe.

Markedly improved patients

In the markedly improved group, 56 of the 72 seizures analyzed propagated outside the epileptogenic temporal lobe. Table 2 summarizes the anal-

TABLE 1. *Patient characteristics*

Patient/sex/class	EEG	Main SEEG ictal onset	CT scan	Follow-up (yr)	Neurosurgery	Pathology
Resistant group						
1/F/IIIA	R FT	R T	N	8	R T	N
2/F/IIIA	R T	R T	N	6	R T	N
3/M/IIIA	R T	R Amyg/Hipp	MD	12	R T	HS
4/M/IVA	R T	R Hipp	N	14	R T	N
5/M/IIIA	R FT	R Hipp	N	6	R T	N
6/M/IIIA	L T	L Hipp	N	8	L T	N
7/F/IIIA	R T	R Amyg	MD	6	R T	Tu
Seizure-free group						
8/F/IA	R T	R Amyg	N	5	R T	N
9/M/IA	R T	R Hipp	N	5	R T	N[a]
10/M/IA	R FT	R Hipp	N	7	R T	N
11/F/IA	R FT	R Hipp	N	6	R T	HG
12/M/IA	R T	R Amyg/Hipp	N	9	R T	HS
13/M/IA	L FT	L Hipp	N	10	L T	N[a]
14/M/IA	R > L FT	R Hipp	MD	6	R T	Tu
Intermediate group						
15/M/IIC	R FT	R Hipp	N	5	R T	N
16/M/IIB	R FT	R Hipp	N	5	R T	HS
17/M/IC	R T	R Hipp	N	5	R T	HS
18/M/IIB	R > L T	R T	N	7	R T	HS
19/M/IC	R T	R Amyg	N	12	R T	N[a]
20/F/IIB	L T	L Amyg	N	7	L T	HS
21/M/IIA	R > L FT	R Amyg/Hipp	Tu	5	R T	Tu

Amyg, amygdala; SEEG, stereo EEG; FT, frontotemporal; Hipp, hippocampus; HS, hippocampal sclerosis; HG, hippocampal gliosis; T, temporal; Tu, tumor; N, normal; MD, missing data.

[a] Hippocampus not available for examination.

yses of propagation sequences. We first analyzed the frequency of ictal invasion of a given lobe as a percentage of the total number of propagating seizures. The ipsilateral frontal lobe (IFL) was most frequently involved, followed by the contralateral frontal lobe (CFL) and the CTL.

We examined the first site of ictal propagation to ET lobes as a percentage of total number of propagating seizures. IFL was the most frequent first site of propagation (Fig. 1), followed by CTL and CFL. We also compared the ET lobes in terms of their position in the propagation sequence as a percentage of the total number of propagating seizures. IFL was invaded before CFL much more often than the opposite; CTL was also involved before CFL more often than the opposite. However, CTL was invaded before IFL in many seizures.

Finally, we examined whether mesial or lateral cortex in each ET lobe was first invaded by the seizure, expressed as a percentage of seizures invading a given lobe. Mesial structures were invaded earlier than lateral structures more often than the opposite in the three ET lobes. CTL mesial invasion without lateral invasion was observed in 4 patients (six seizures); mesial CTL involvement sometimes occurred without frontal invasion (patient 13, three seizures) (Fig. 2).

TABLE 2. *Percentage of propagating seizures showing specific characteristics in markedly improved and resistant groups*

Characteristics	Markedly improved patients	Resistant patients
Frequency of invasion		
IFL	95	100
CFL	61	56
CTL	48	51
First propagation site		
IFL (simultaneous)	66 (11)	62 (36)
CFL (simultaneous)	4 (9)	0 (33)
CTL (simultaneous)	18 (5)	3 (21)
Propagation sequence		
IFL → CFL (opposite)	46 (7)	21 (0)
IFL → CTL (opposite)	21 (16)	28 (3)
CTL → CFL (opposite)	16 (5)	15 (5)
Cortex first invaded		
IFL		
Mesial	49	77
Lateral	32	8
CFL		
Mesial	69	58
Lateral	12	23
CTL		
Mesial	47	40
Lateral	16	20

IFL, ipsilateral frontal lobe; CFL, contralateral frontal lobe; CTL, contralateral temporal lobe; simultaneous, invaded simultaneously with another lobe.

Data are percentages of total number of propagating seizures except in "cortex first invaded," in which percentages of total number of seizures invading a given lobe are shown.

Resistant patients

In the resistant group, 46 propagating seizures were similarly analyzed and compared with those of markedly improved patients (Table 2). The propagation pattern of resistant patients was very similar to that of markedly improved patients. The only differences were that in resistant patients CTL was almost never invaded before IFL and the first site of propagation frequently involved more than one lobe, reflecting occurrence of rapid and widespread diffusions in that group. In resistant patients, the presence of seizures with diffuse onset sometimes made precise estimation of the propagation sequence impossible.

Ictal propagation times

Table 3 summarizes the main measures of seizure propagation times to the first ET site for each patient, including median propagation time, range, and percentage of seizures with ET propagation delay <1.0 s. Median propagation times were different among the three groups, averaging 7.2 s for resistant patients, 11 s for the seizure-free group, and 17.9 s for the markedly improved group ($p < 0.06$) and also between the resistant group and the markedly improved group ($p < 0.05$). Average minimum propagation times of the three groups (3.6 s for resistant patients, 7.1 s for seizure-free patients, and 12.7 s for almost seizure-free patients) were also significantly different ($p < 0.05$).

To establish which propagation times produced the median time difference, we compared the groups by frequency of propagations that occurred at delays of 0, 0–1, 1–2, 2–5, 5–10, 10–20, 20–30, 30–40, 40–50, 50–60, and >60 s. A propagation time of 0 s was observed in 11% of propagating seizures in resistant patients (3 of 7 patients showing at least one) and in 0% of seizures in markedly improved patients ($p < 0.06$). Propagation times of 0–1 s were observed in 30% of propagated seizures in resistant patients (5 of 7 patients showed at least one) versus 7% of seizures in markedly improved patients (2 of 14 patients showed at lease one) ($p < 0.05$). No difference was noted in >1-s intervals and, in particular, long delays were not more frequent in markedly improved patients. We also examined propagations to CTL independent of the other propagation sites: >1-s intervals were equally frequent in the markedly improved and resistant groups of patients. Comparisons using longer intervals did not show more significant results for >1-s delays.

Finally, in the seizure-free group, 3 patients showed a second less active mesiotemporal contra-

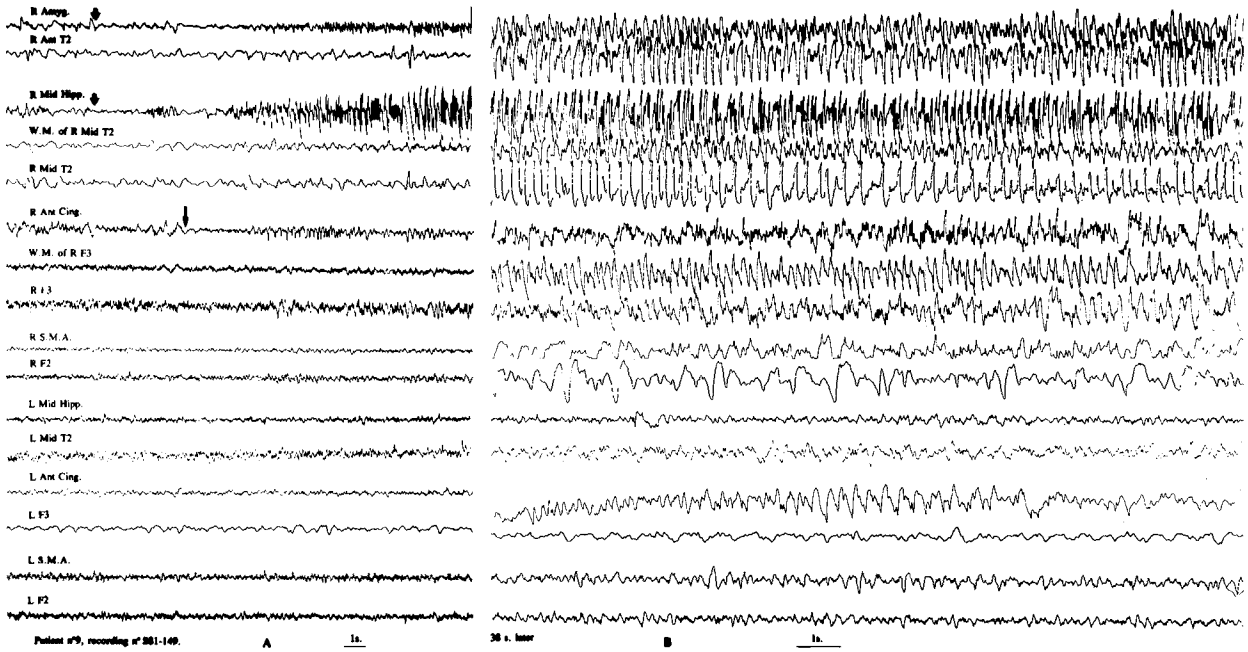

FIG. 1. Example of the most common stereo EEG (SEEG) propagation pattern of temporal lobe seizures. **A:** Right mesiotemporal ictal onset [amygdala (Amyg) and hippocampus (Hipp), short arrows], with diffusion to ipsilateral anterior cingulate (Cing) gyrus 5 s later (long arrow). **B:** Thirty-eight seconds after SEEG interruption. Ictal activity has invaded right laterotemporal cortex and right F2 gyrus and reaches left anterior Cing. The right supplementary motor area (SMA) and its corresponding lateral cortex and all the left structures (except anterior Cing.) are spared by seizure propagation. F2, middle frontal gyrus; F3, inferior frontal gyrus; T2, middle temporal gyrus; WM, white matter.

lateral focus. Their propagation times were very variable and almost always in the time range of those of other markedly improved patients; therefore, these 3 patients were not different from markedly improved patients in ET propagation speed.

Focal temporal ictal onset

For all patients, seizures of exclusively temporal origin usually had onset at the level of temporomesial structures (amygdala and/or hippocampus), except in patients 1, 2, and 18, in whom seizures had both mesial and lateral onset. The focal or nonfocal aspect of ictal onset varied in the same patient. We considered a patient focal when most seizures were of focal onset. By this definition, 71% of markedly improved patients were focal as compared with 43% of resistant patients ($p > 0.4$). The number of patients in each group with systematic focal onsets was not significantly different (29% for markedly improved vs. 14% for resistant patients, $p > 0.2$).

Extent of ictal propagation

Finally, we compared the extent of ET ictal propagation in the different groups. The proportion of different partial seizure types (subclinical/simple partial, CPS, secondarily generalized) were equal in the different groups (resistant group 39, 40, and 21%, respectively; markedly improved group 36, 42, and 22%, respectively). Analysis of propagation extent showed that 77% of ET lobes of resistant patients were reached by ictal activity versus 59% in markedly improved patients ($p > 0.3$). When we considered only the subtype of subclinical/simple partial seizures, this difference tended toward significance (ET propagation extent of 50% in resistant patients versus 18% in markedly improved patients, $p < 0.17$).

DISCUSSION

Intracerebral recording of seizures (SEEG) is a very useful measure to localize the site of ictal onset (Spencer et al., 1982; Saint-Hilaire et al., 1991). The present data suggest that SEEG data may be used for more than locating the site of ictal onset and that some temporal characteristics of propagation of ictal activity are associated with resistance to TLE surgery. In our data, the key temporal characteristics appear to be very rapid propagation to extratemporal sites (mostly ipsilateral frontal sites but sometimes contralateral) or diffuse onsets. This evidence agrees with our previous observations suggesting that fast ictal propagation is also associated with resistance to frontal epilepsy surgery (Turmel et al., 1992).

The prognostic value of ictal propagation was previously recognized for the propagation time to

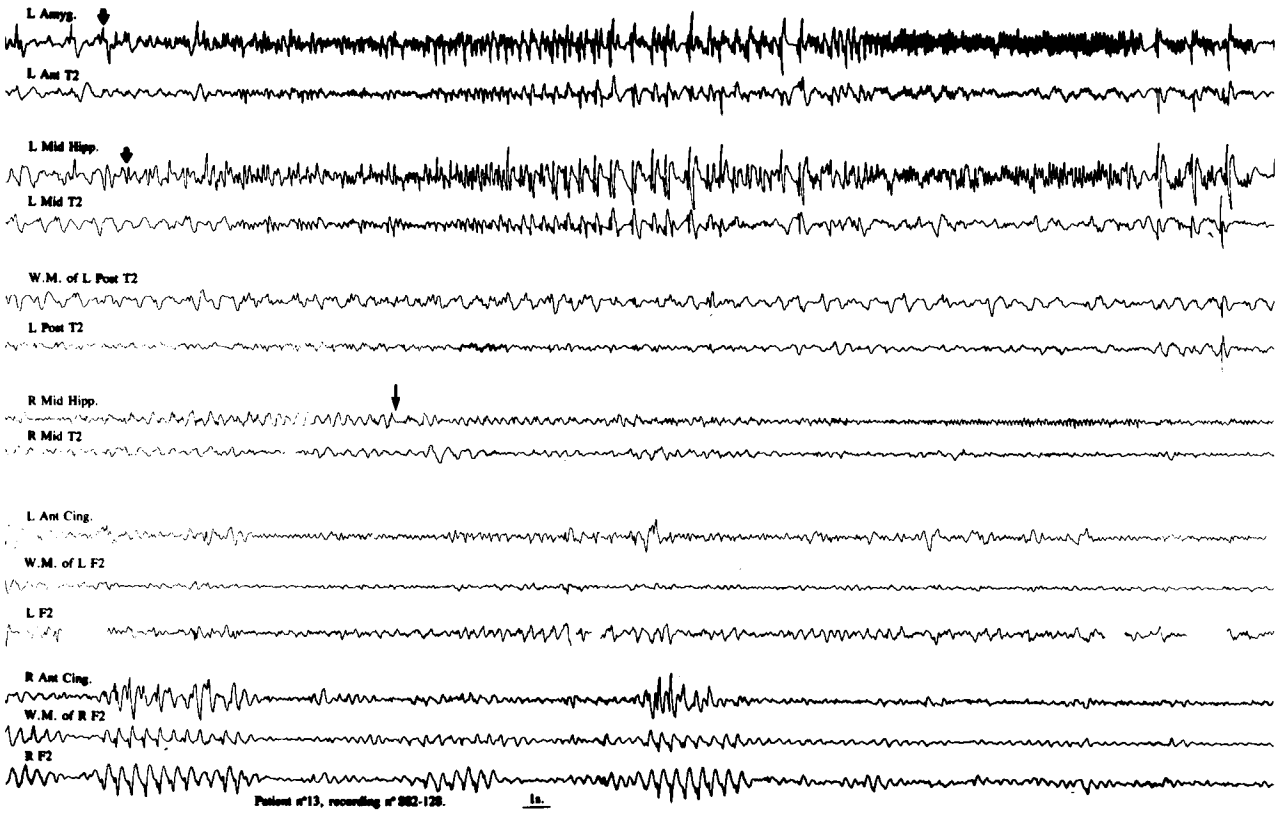

FIG. 2. Example of a stereo EEG seizure originating from mesiotemporal structures (amyg and middle Hipp, thick arrows), with clear contralateral propagation to right Hipp, without involvement of right laterotemporal cortex and the frontal structures. Frontal lobes exhibit their interictal discharge pattern. Abbreviations as in legend to Fig. 1.

CTL (Lieb et al., 1986) and for diffused ictal onsets of temporal origin studied with only bitemporal electrodes (Lieb et al., 1981). Our data extend these earlier findings to what appears to be the most frequent first site of propagation: IFL. Our data also suggest that late ictal propagations are not good markers of surgical success. Indeed, no propagation time >1.0 s was more frequent in markedly improved patients: resistant patients and markedly improved patients had comparable frequencies of long propagation delays. This finding differs from that of Lieb et al. (1986), who reported good prognostic value for late propagations to CTL. This difference in results could be due to several factors, including number of lobes examined, electrode positions, and patient selection criteria.

In contrast to temporal characteristics, spatial factors such as focal onset of seizures, propagation extent, or presence of purely temporal seizures did not appear to differ among the groups, possibly owing to the small number of patients included, but may indicate that spatial characteristics are less important than temporal characteristics in surgical prognosis.

The frequent fast propagations in patients who

TABLE 3. *Median and range of propagation times to first extratemporal site invaded by seizure and percentage (n) of propagating seizures with propagation time <1 s*

Case	Median time (s)	Range	Seizures with fast propagation
Resistant group			
1	0.5	0–1.5	83% (5/6)
2	17	8–26	0% (0/2)
3	10	0–87	14% (1/7)
4	0.5	0–1	100% (3/3)
5	16	15–27	0% (0/3)
6	1.7	1–14	50% (4/8)
7	5	1–14	6% (1/17)
Seizure-free group			
8	2	1–4	17% (2/12)
9	8	5–9	0% (0/5)
10	18	18–20	0% (0/4)
11	17	11–23	0% (0/2)
12	23	9–35	0% (0/7)
13	8.5	5–18	0% (0/4)
14	0.75	0.5–1	100% (2/2)
Intermediate group			
15	25	13–37	0% (0/2)
16	11	10–11	0% (0/3)
17	29	24–33	0% (0/3)
18	4.75	1.5–11	0% (0/4)
19	19.5	19–20	0% (0/2)
20	—	—	—
21	18	9–51	0% (0/6)

were resistant to neurosurgery may have had several possible causes. The epileptic focus may not have been temporal and SEEG data may have been misleading. Intracerebral investigation can provide only limited sampling of brain regions; theoretically, a seizure of ET origin could appear to have temporal onset if activity always propagated first to the anterotemporal lobe. All resistant patients in our study had frontal electrodes, but none had orbitofrontal, frontopolar, or insular electrodes. An orbitofrontal onset could propagate to amygdala through fronto-amygdalian projections and mimic temporal onset followed by rapid propagation to anterior cingulate gyrus. However, evidence supports the existence of a temporal focus in our resistant patients. That >50% of ictal onsets were temporal, that seizures were restricted to the temporal lobe in some patients, and that postoperative disappearance of the EEG interictal focus in 4 patients (patients 2, 3, 5, 6; missing data for patient 7) suggests that our patients had at least a temporal focus.

If the resistant patients had a temporal focus, the rapid diffusion we observed may be a sign of a qualitatively different seizure disorder. The epileptogenic region may be said to be wider (e.g., temporofrontal) in such patients. However, none of their seizures showed distinctly frontal onset, and most seizures showed clear temporal onset. Thus, our patients were clearly different from patients with two or more independent epileptogenic regions that can also be detected, and also from patients with diffuse-onset epilepsy. The situation observed in the resistant group appears to be intermediate between the typical clear-cut temporal focus and that of two or more independent foci.

Such intermediate epilepsy may appear alone or may have developed from a temporal focus. If this seizure disorder is the product of development, short propagation delays may reflect facilitated connections between a temporal focus and a nonresected epileptogenic or potentially epileptogenic region, which could result from a process described as "secondary epileptogenesis" (Morrell, 1989); e.g., ictal events originating in temporal lobe frequently invade an ipsilateral mesiofrontal region may acquire some potentially epileptogenic properties, even if many ictal onsets indicate a main temporal epileptogenic region. This appears to be an important avenue for further research.

In our study, seizure propagation pathways did not vary as a function of postoperative outcome. This finding is in agreement with the results of Lieb et al. (1991). However, the propagation pathways observed in our patients were variable and could deviate from the pattern reported by Lieb et al., (1991) involving IFL, CFL, and CTL successively. Our data indicate that CTL can be invaded independent of frontal lobes, implying that interhemispheric propagation is not unidirectional but can proceed through multiple pathways, at least one to IFL and one to CTL.

As did Spencer et al. (1987), we observed that CTL is most often initially invaded mesially, without necessary frontal or lateral CTL involvement. These observations may indicate that ictal propagation can sometimes proceed through a more direct pathway than the frontal one, such as the hippocampal commissure. Studies of EEG coherence (Gotman, 1987; Lieb et al., 1987) and stimulation of human mesiotemporal structures (Wilson et al., 1991) have not shown a functional role of the commissures in interhippocampal propagation. However, variation in stimulation parameters made responses appear in contralateral mesiotemporal structures in the study of Wilson et al. (1991). In physiopathologic conditions during epileptic seizures, the hippocampal commissure may be implicated in interhemispheric propagation.

Our data on ictal propagation to frontal lobes show that their activation is sequential, with IFL invaded before CFL. Mesial structures of these lobes are most often invaded first. Propagation to ipsilateral anterior cingulate gyrus can follow amygdalocingulate projections (Amaral et al., 1984) or parahippocampocingulate projections (Amaral, 1987). Propagation to contralateral cingulate gyrus can proceed through callosal projections, but many possible alternate routes exist.

Temporal characteristics of intracerebral ictal activity can be useful in interpretation of SEEG data in TLE. Very short propagation times and diffuse ictal onsets indicate that anterotemporal lobectomy will have reduced efficacy and suggests that the epileptic focus is either difficult to localize or subject to secondary epileptogenesis. On the other hand, our data suggest that late propagation has no prognostic value.

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