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Speech Preservation during Language-dominant, Left Temporal Lobe Seizures: Report of a Rare, Potentially Misleading Finding

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Summary: *Purpose:* To evaluate the prevalence and mechanism of ictal speech in patients with language-dominant, left temporal lobe seizures.

Methods: We retrospectively reviewed the video-EEG telemetry records for the presence of ictal speech in 96 patients with surgically proven left temporal lobe epilepsy and studied the seizure-propagation patterns in three patients who required intracranial EEG recordings for seizure localization.

Results: Ictal speech preservation was observed in five patients. One patient's seizures demonstrated rapid propagation of the ictal discharges to the contralateral temporal area where the seizure evolved, resembling a nondominant temporal lobe seizure. The other two patients had ictal discharges that remained confined to the inferomesial temporal areas, sparing language cortex.

Conclusions: Preservation of speech in complex partial seizures of language-dominant, left temporal lobe origin is rare. Based on intracranial EEG recordings, the likely mechanism underlying this potentially misleading clinical finding is the preservation of language areas due to limited seizure-propagation patterns. **Key Words:** Ictal speech—Seizure semiology—Temporal lobe epilepsy—Language.

Speech and language manifestations during complex partial seizures of temporal lobe onset are reliable predictors of seizure lateralization. Previous investigators have demonstrated that ictal speech is a relatively common manifestation of nondominant temporal lobe seizures (1–3). Conversely, postictal language dysfunction is the hallmark of temporal lobe seizures originating from the dominant hemisphere (1,2,4). Preservation of speech during complex partial seizures of language-dominant, temporal lobe origin has not been reported or systematically studied. We evaluated all patients with language-dominant, left temporal lobe seizures for evidence of this potentially misleading finding. In addition, we assessed potential mechanisms of ictal speech in the subset of patients who underwent intracranial monitoring.

METHODS

We retrospectively reviewed records from our 1987–2002 database and selected all patients who met the following criteria: (a) left hemisphere language-dominant determined by intracarotid amobarbital test (Wada test); (b) ictal EEG onset from the left temporal lobe; and (c) resolution of seizures for $\geq$2 years after epilepsy surgery.

The Wada test was performed by using 125 mg sodium amobarbital injected into the internal carotid artery on the side of the seizure focus, followed by injection of the opposite hemisphere ~30 min later. Before sodium amobarbital injection, baseline testing was performed. A set of 11 stimuli (four objects, one picture, four single written words, one oral sentence for repetition, and a sentence to be constructed) was presented. The patients were asked to count, to sing, and to state their names. Language testing was performed within 2 min of the injection. Once evidence of hemispheric anesthesia appeared, 20 items (objects, toys, single written words, and sentences to repeat) were presented to the patients. The patients were then asked verbally to point to objects and to discriminate shapes and colors and were again asked to count, sing, and state their names. When grip strength and EEG returned to baseline, unscored free recall of items was elicited. The patients were next shown, randomly, all items presented at baseline and under drug along with 24 foils not shown before. A hemisphere was classified as language dominant if the patient failed language tasks with injection of amobarbital into that hemisphere but not the opposite hemisphere. Patients were classified as mixed dominant if both injections caused language errors.

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When intracranial electrodes were necessary for localization of the seizure focus, subdural strip and/or depth electrodes were implanted by using fluoroscopy and MRI stereotactic methods, respectively. Six-contact subdural strips were inserted bilaterally through frontal burr holes directed toward the frontal operculum and through temporal burr holes directed toward inferomedial temporal regions. As a result, the frontal strips contacted the middle and inferior frontal gyri, and the temporal strips contacted both lateral and inferior temporal regions. When necessary, bilateral depth electrodes were inserted into the amygdala and hippocampus via a posterior approach.

Video-EEG records of patients meeting inclusion criteria were studied. Ictal speech was defined as clearly understandable words or phrases that were well articulated and linguistically correct (2). For the purpose of this study, preservation of speech function had to occur while the patient exhibited automatisms and/or decreased level of consciousness. Our observations were based on interactions between the patients and their nurses. A standardized language-testing protocol was not used. Patients who spoke only during the aura or the postictal period were not included. Localization of onset, propagation, and termination of seizure activity was ascertained for each seizure.

For intraoperative stimulation mapping, the afterdischarge threshold was established during motor mapping. Language mapping was then performed with the largest current that did not evoke prolonged afterdischarges. Language function was assessed by object naming and counting. Stimulation sites were selected to cover classic language areas as well as the proposed resection site.

RESULTS

Ninety-six patients had complex partial seizures of language-dominant, left temporal onset and Engel class 1 outcome for $\geq$2 years. Preservation of at least some speech was observed in five (5%) of these patients (Table 1). None of them demonstrated paraphasic errors. During the seizures, some patients demonstrated only partial capacity for language function, and on rare occasions, they became aphasic toward the end of their seizures.

Three patients with preserved speech required intracranial recordings for seizure localization (patients 1 to 3). All seizures had onset in left mesial temporal structures. One patient's seizure pattern demonstrated rapid propagation of the ictal discharges to the contralateral temporal area where the seizure then evolved (Fig. 1A). The left temporal discharges abated quickly. The other two patients had ictal discharges that remained confined to the left inferior and medial temporal areas for $\sim$120 s after the seizure onset without spread to frontal lobe areas (Fig. 1B). Intraoperative speech mapping with electrical stimulation demonstrated only suprasylvian language representation in all three patients.

DISCUSSION

Our findings demonstrate that preserved speech during complex partial seizures arising from the language-dominant, left temporal lobe is rare and can be explained by at least two possible mechanisms. First, seizure activity may rapidly propagate from the dominant to the nondominant temporal lobe and then evolve, resembling a nondominant temporal lobe seizure. Second, the most prominent seizure activity may remain confined to the inferomesial temporal areas, sparing language cortex. In addition, the more restricted suprasylvian language representation found in three of the patients may have contributed to ictal speech preservation.

Previous studies using functional neuroimaging modalities demonstrated the relation between seizure semiology and anatomic localization in patients with temporal lobe epilepsy (TLE) (5,6). Recently it was shown that progression of seizure semiology in TLE depends on the propagation pattern of epileptic activity (7). Several investigators have noted that dominant temporal lobe seizures typically cause ictal and postictal language disturbances because of an involvement of cortical language areas (2,8). Moreover, the study in postictal language dysfunction suggests

TABLE 1. *Seizure semiology and the pattern of ictal speech in patients with language-dominant, left temporal lobe seizures*

Patient	Age at onset	Diagnosis	Intracranial monitoring	Seizure semiology before ictal speech	Pattern of ictal speech
1	7 mo	FCD	Yes	Behavioral arrest, hand automatisms	Sentence, "What about it."
2	2 mo	FCD	Yes	Behavioral arrest, hand automatisms	Simple words, "two" and "yes," then said "It's a secret."
3	10 yr	HS	Yes	Behavioral arrest, hand automatisms, and posturing of the right upper extremity	Sentence, "It was right." once, then "That's okay." repeatedly
4	22 yr	Astrocytoma	No	Unresponsive, looking around, and hand automatisms	Sentence, "I am okay." repeatedly
5	25 yr	PTE	No	Inert, confused, lip smacking, posturing of the right upper extremity	Simple word, "yes," and said, "No, I can't." when she was asked to name "watch."

FCD, Focal cortical dysplasia; HS, hippocampal sclerosis; PTE, posttraumatic epilepsy.

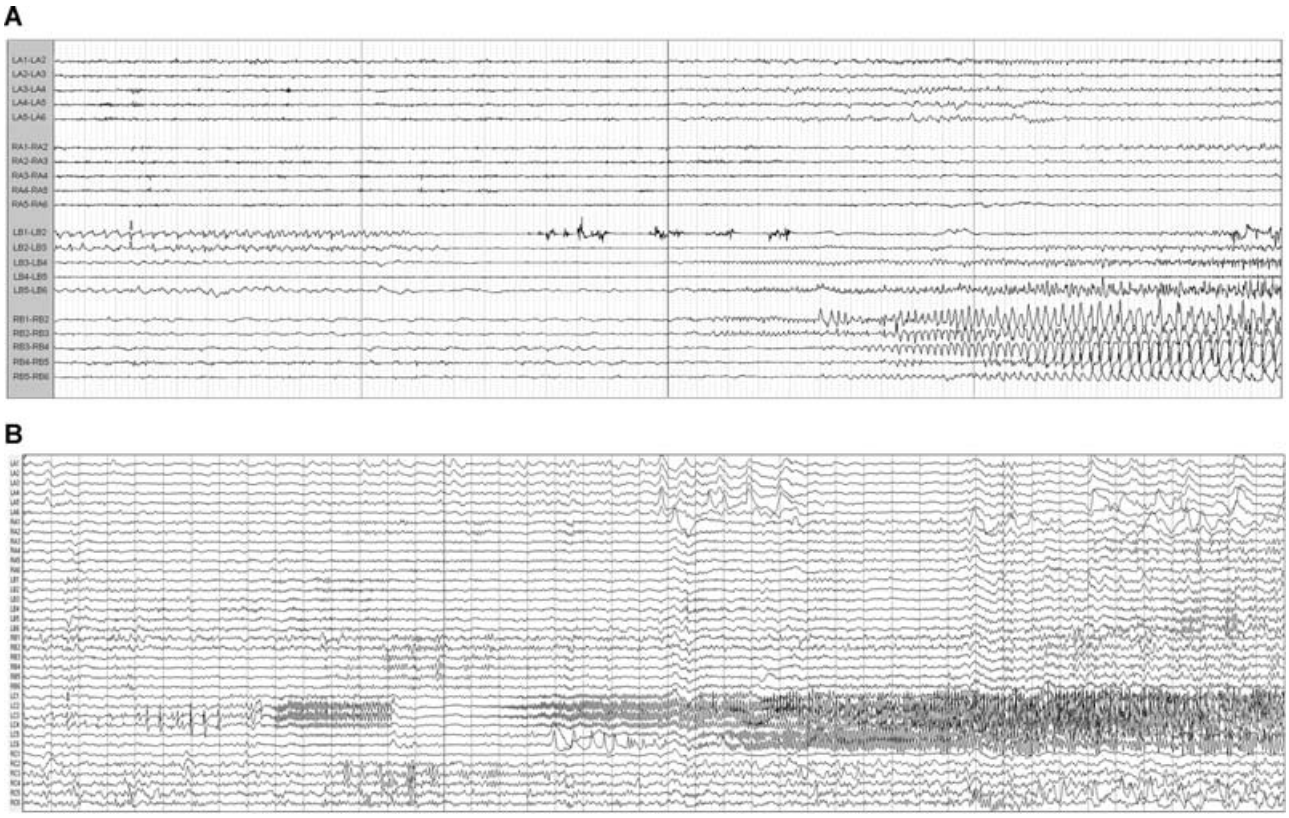

FIG. 1. Intracranial EEG recordings demonstrate two mechanisms of ictal speech preservation. **A:** Rapid propagation to the nondominant temporal lobe. *Arrow,* Electrographic onset. **B:** Relatively confined ictal EEG patterns with limited propagation to language areas. *Arrow,* Electrographic onset at the left mesial temporal electrodes. LA, left frontal; RA, right frontal; LB, left subtemporal; RB, right subtemporal; LC, left hippocampal depth; RC, right hippocampal depth.

that propagation of the electrographic discharges from the nondominant to dominant temporal lobe can result in postictal language disturbances (8).

Several ictal propagation patterns have been described in patients with mesial temporal lobe epilepsy (MTLE) (9–11). Data from depth electrode recordings in this particular group of patients demonstrate that most seizures spread to the ipsilateral neocortex (9). In some seizures, however, rapid ictal propagation occurs from one mesial temporal structure to the contralateral mesial temporal structure via the dorsal hippocampal commissure without involvement of the neocortex (9,10). This atypical spreading pattern, as documented in our patient (Fig. 1A), may lead to false lateralization by seizure semiology, noninvasive EEG recordings (12), and rarely subdural electrode recordings (13). It is therefore possible that rapid ictal propagation from the dominant to nondominant temporal lobe, as seen in our patient (patient 1), can result in speech preservation during complex partial seizures of language-dominant, left temporal lobe onset.

Although the study of cerebral anatomy underlying language function was first described more than a century ago, our knowledge about cortical language function still remains rudimentary. Several studies using electrocortical stimulation mapping (14–16) and functional neuroimaging techniques (17) have implicated multiple regions of the dominant hemisphere, including perisylvian inferior frontal, temporoparietal, and basal temporal cortex, in language function. However, the studies of precise location of language-specific areas within the dominant hemisphere in patients with focal epilepsy often show extensive variability (15,16). This variability is observed particularly in temporoparietal cortex and may represent an effect of language reorganization. More recently, functional magnetic resonance imaging studies of language reorganization in patients with left TLE suggest a possibility of inter- and intrahemispheric functional reorganization outside the left temporal lobe (18,19). Consistent with these findings, intraoperative electrocortical stimulation in our patients showed reproducible sites causing naming dysfunction only in perisylvian inferior frontal cortex, a region that did not become involved in our patients' seizures.

Recently, Privitera et al. (4) reported preserved speech in two patients with subclinical electrographic seizures involving only mesial temporal structures in the language-dominant hemisphere. In both patients, however, typical language disturbances developed when the seizures progressed to become complex partial seizures. The authors (4) hypothesized that mesial temporal involvement alone is unlikely to cause language dysfunction in dominant

temporal lobe seizures. Two of our patients also demonstrated speech preservation with isolated left inferomesial temporal seizures (Fig. 1B). However, with further spread of seizure activity, both of our patients clearly demonstrated an alteration of consciousness while they retained the ability to speak. Given a combination of suprasylvian language representation and limited infrasylvian involvement during their seizures, our patients provide an example of how, on rare occasions, complex partial seizures with isolated inferomesial temporal involvement need not result in complete speech impairment.

The main limitation of our study is that subdural strip and/or depth electrodes provide only a limited sample of cerebral activity. It is likely that all language cortex was not covered by our subdural electrode recordings. In addition, all potential language sites are not tested during intraoperative stimulation mapping, which is simply performed to ensure that the proposed resection can be accomplished safely. We suspect that some potential language cortex that we do not test routinely in the operating room, such as basal temporal regions, became involved during our patients' complex partial seizures. It is therefore possible that involvement of "noncritical" language areas may allow at least some preservation of speech. Alternatively, the ictal discharge may sometimes be inadequate to produce complete functional impairment of the tissue.

In summary, our findings suggest that ictal speech is rare in patients with language-dominant, left temporal lobe seizures. When this potentially misleading clinical feature occurs, it can usually be explained by seizure propagation that largely spares critical language areas. Because the number of patients in our study is small, we cannot exclude the possibility that other mechanisms might produce similar clinical effects.

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