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# Characterization of the basal temporal language area in patients with left temporal lobe epilepsy

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**Article abstract**—We evaluated 5 consecutive patients with subdural grid electrodes (including placement over the left basal temporal region) for focal resections for control of intractable epilepsy. All 5 had language dysfunction when we performed cortical stimulation over the basal temporal region (the inferior temporal gyrus, the fusiform gyrus, or the parahippocampal gyrus) using a systematic battery of language tests. The area in which language interference could be produced began from at least 11 to 35 mm posterior to the temporal tip and ended at least 39 to 74 mm posterior to the temporal tip. The most consistently impaired language tasks were spontaneous speech and passage reading, but there was impairment of all language functions tested in some patients. Language deficits after dominant temporal lobectomy may result from resection of this area.

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The temporal lobe is the most common target for surgical resection in patients with medically intractable epilepsy. Even though dominant temporal lobectomy is planned to spare primary language areas, it may result in a variety of impairments in verbal performance on assessments of IQ,[1-4] memory,[1,3-9] naming,[10] word finding,[5] and comprehension.[5]

These language deficits may correlate with the overall extent of dominant lateral temporal lobe resection.[7] However, such postoperative impairments may be due to damage of specific regions within the boundaries of the lobectomy. Previous clinicopathologic studies[11,12] found that the posterior inferior temporal gyrus was 1 such area involved with language. More recently, cortical stimulation of the basal temporal area temporarily altered language function.[13-16] Initial data suggested that this region was localized to a 2 × 2 cm area of the fusiform gyrus,[13] and detailed language testing found dysnomia to be the most consistent abnormality.[13,15] However, this area has been described in only a few patients. Therefore, the need exists to further define the site and function of the basal temporal language area (BTLA).

This study is based on a consecutive series of 5 patients undergoing cortical stimulation via the implanted subdural electrode grid technique whose grid placements included the left basal temporal region. We used a systematic battery of language tests to define the regions and functions involved. We performed neuroanatomic localization of electrode positioning through a combination of direct visualization and 3-dimensional computerized axial tomography (3-D CT).

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**Table 1. Baseline data before implantation of left basal temporal electrode grids**

| | Patient | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| Age | 34 | 34 | 26 | 28 | 35 |
| Sex | F | F | M | F | F |
| Handedness | R | R | R | R | R |
| Seizure type(s) | SP CP Sec gen | CP Sec gen | CP Sec gen | CP | CP Sec gen |
| MRI (head) | Possible L temp lesion | Infarct* | Normal | S/P previous L partial ant temp lobectomy | Normal |
| Neuropsych. results | | | | | |
| Dominance of language function (Wada) | L | L | Mixed | L | Mixed |
| WAIS-R | | | | | |
| VIQ | 89 | 102 | 78 | 92 | 103 |
| PIQ | 98 | 92 | 91 | 112 | 91 |
| FSIQ | 86 | 103 | 80 | 99 | 97 |
| Wechsler Memory Scale | 97 | 110 | 84 | 93 | 108 |
| Auditory Comp | + | + | + | + | + |
| Word Fluency | + | + | + | + | + |
| Repetition | + | + | + | + | + |
| Naming | — | + | — | — | + |
| Reading Comp | + | + | + | + | + |

SP Simple partial.
CP Complex partial.
Sec gen Secondary generalized.
Temp Temporal lobe.
* Left cerebral peduncle.
S/P Status post.
Wada Intracarotid sodium amobarbital test.
+ Intact.
— Impaired.

**Methods.** *Patients.* Thirteen patients had subdural electrode grids placed over portions of the left hemisphere as part of their evaluations before surgical resections for medically intractable epilepsy between June 1987 and December 1988. The placements included the basal temporal region in 6 patients, 1 of whom was not included in this study because she developed a small, thin, asymptomatic hematoma between the electrode grid and the cortex, which interfered with the stimulation testing. Baseline data of the 5 remaining patients before implantation of left basal temporal grids, including presurgical language laterality and language performance, are summarized in table 1.

*General testing procedures.* The patients were evaluated as part of a protocol for surgical resection for treatment of medically intractable epilepsy, which included an extensive preoperative evaluation consisting of prolonged video/scalp EEG monitoring; neuropsychological testing, including the same tasks to be used during cortical stimulation; MRI of the head; intracarotid sodium amobarbital (Wada) testing; and cerebral angiography in the context of the Wada testing. When scalp-recorded data alone were deemed insufficient to make a final decision regarding the extent of surgical resection, patients had operative placement of multicable subdural grid electrodes (Wyler Electrode Grid, Ad Tech Medical Instrument Corp., Racine, WI). The grid arrays were tailored, in terms of size and location, according to the needs and anatomic constraints of each patient. The general method and rationale for subdural electrode grid placement for localization of epileptic foci and functional mapping of language, motor and sensory cortical areas have been reviewed elsewhere[17,18] and will be summarized only briefly here. The grids consisted of medical grade stainless steel or platinum-iridium electrodes of 3 mm diameter embedded in Silastic with center-to-center interelectrode distances of 10 mm. The grids were positioned according to the needs of the preoperative evaluations. Arrays of 5 × 4 (patient 1) or 8 × 2 (patients 2 through 5) electrodes were placed over the basal temporal region.

*Stimulation testing procedures.* The stimulation procedures were done primarily for clinical purposes. The patients gave informed consent for any additional testing done in accordance with a protocol approved by the Joint Committee on Clinical Investigation of The Johns Hopkins Medical Institutions. Functional localization of language, motor and sensory regions, using the cortical stimulation procedure, was done for an average of 2 to 3 hours per day over several days. Grass S-88 or S-12 cortical stimulators (Grass Instrument Co., Quincy, MA) were used to produce 1- to 7-second trains of 0.3-msec duration pulses. Fifty alternating polarity pulses/sec were given to individually selected electrode pairs beginning at 1.0 mA and increasing by 0.5- to 1.0-mA increments to a maximum of 15 mA. The maximum stimulation intensity used depended on the occurrence of motor or sensory phenomena or an impairment in the level of consciousness as observed by testing personnel or reported by the patient; the occurrence of afterdischarges on EEG, which could possibly interfere with cognitive function,[17] or the attainment of a maximum current level of 15 mA.

Stimulation testing began once the maximal stimulus intensity possible for a given electrode pair was attained. All tests were carried out with and without stimulation. The patients were evaluated for evidence of motor impairment of their eyes, tongue, mouth, and extremities by observing them for either contractions at rest or inability to perform voluntary movements on command. All patients were then given the following tasks, which were chosen so as to widely survey language functions in a systematic manner using standard clinical tests[19]: *spontaneous speech* was assessed using a topic of known interest to the patient; *auditory comprehension* was tested using a modified 1-step version of the Token Test[20]; *repetition* was tested using single words, both regular and irregular[21]; *visual confrontation naming* was tested with the Boston Naming Test, Experimental Edition[22]; *naming to definition* was tested using definitions that took no longer than 3 seconds to present; *single word reading* was tested with the same list as used for auditory repetition; *reading of passages* was assessed with short stories.

All tests were given in advance of the stimulation sessions in order to acquaint the patients with the tasks, assess baseline performance, and exclude items unfamiliar to the patient. Visual materials were presented in front of the patient. In the case of reading material, the stimuli were printed in large letters. Individual items were given on a trial-by-trial basis when the subject was ready. For spontaneous speech and reading of passages, stimulation began after the patient was speaking fluently. For the other tasks, electrical stimulation was begun about 1 second before the start of stimulus presentation and continued until presentation was completed; stimulation in no case lasted more than 7 seconds. Each task was typically given for at least 3 trials with stimulation. All trials were videotaped and transcribed.

*Analysis of language data.* A function was considered intact if it was performed at the level and latency of baseline



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testing. Any other response was counted as an error, provided that there was no evidence of motor impairment, seizure activity (either behaviorally or electrographically, including prolonged afterdischarges), fatigue, painful sensations due to stimulation of the dura or of trigeminal fibers accompanying blood vessels in the pia-arachnoid,[23] or other alterations of level of consciousness, sensation, or ability to respond apparent to the patient or the observer. If any of these occurred, testing was suspended until baseline patient behavior and EEG activity resumed. Almost all errors were of complete failure of response.

Performance during stimulation at a given site, with each individual task, was compared to nonstimulated performance (summing over all nonstimulated trials with the subject) using the binomial test. The significance level for reporting impairment was set at $p < 0.05$ (1-tailed).

*Anatomic localization.* The exact location of the subdural electrodes was noted visually in relation to anatomic landmarks (such as the anterior temporal tip, the sylvian fissure, etc) upon placement and removal, as well as on 3D-CT.[24-26] The 3D-CT was given greater weight due to the greater reliability of postoperative imaging when compared with intraoperative estimates.[27]

The 3D-CT examinations were performed on a Siemens Somatom DR3 scanner (Siemens Medical Systems Inc., Islin, NJ). The scanning parameters included 4-mm slice thickness, 3-mm table incrementation, 0 degrees gantry angulation, 450 mA, 125 kVp, and a high-resolution convolutional filter. The image data were transferred to a CEMAX 1500 multidimensional imager (CEMAX, Medical Systems, Santa Clara, CA) capable of displaying the original data and performing 2-dimensional multiplanar reconstruction as well as creating 3-dimensional images. The 3D-CT data may be displayed from several angles, including the basal view used in this study. Specific measurements were performed from the anterior temporal tip to the appropriate electrode contact. The accuracy of the surface measurements was evaluated using a linear phantom, which demonstrated that the linear measurements were without error.

**Results.** As expected from baseline testing, error rates in the absence of electrical stimulation were low (mean proportion correct for each task, by subject, was 0.98 ± 0.03).

In all 5 patients stimulation of part of the basal temporal region, including portions of the inferior temporal, fusiform or parahippocampal gyri, caused temporary language dysfunction (table 2). These deficits usually resolved immediately when stimulation ceased and never persisted beyond the first few post-stimulation seconds.

There were deficits in all facets of language tested, but the pattern of dysfunction was not identical for each patient. Spontaneous speech and passage reading were impaired in all 5 patients. All language tasks were impaired in patients 2, 3, and 5. In addition to spontaneous speech and passage reading, patient 1 had impaired repetition and reading single words and patient 4 had impaired visual confrontational naming and auditory comprehension.

The rostral-caudal length of the BTLA was variable. It began at least 11 to 35 mm posterior to the anterior tip of the temporal lobe and ended at least 39 to 74 mm posterior to the anterior tip of the temporal lobe. However, this area extended to the anterior margin of the grid in 3 patients and to the posterior margin of the grid in 4 patients (table 2).

**Table 2. Anterior and posterior margins of the basal temporal language area (BTLA), as measured back from the anterior tip of the temporal lobe, and gyri involved**

| | Patient | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| Anterior margin (mm) | 35 | 30 | 16 | 24 | 11 |
| Posterior margin (mm) | 66 | 58 | 72 | 39 | 74 |
| Gyri | | | | | |
| Inf temp | * | | | | |
| Fusiform | * | * | * | * | * |
| Parahippo | | | * | * | * |
| Speech area extends to anterior margin of grid | Yes | No | No | Yes | Yes |
| Speech area extends to posterior margin of grid | Yes | Yes | Yes | No | Yes |

Inf temp Inferior temporal.
Parahippo Parahippocampal.
* BTLA found to include portions of these gyri.

This same region was a source of significant interictal epileptiform activity in 3 patients, minimal interictal epileptiform activity in 1 patient, and a site of spontaneous seizure onset in 1 patient.

All 5 patients underwent subsequent partial left temporal lobectomies for intractable epilepsy (table 3). Only patient 1 had a resection that included basal temporal areas where stimulation had caused temporary language deficits, and this patient had a postoperative dyslexia that totally resolved over a 1-year period. No resection of the posterior aspects of the superior or middle temporal neocortex had occurred, and no language deficits had been produced by stimulation of the anterior (subsequently resected) aspects of the superior and middle temporal gyri in this patient. The posterior border of the lateral resection was 1 cm anterior to the anterior border of the superior temporal speech area. Because of the deficit noted in patient 1, it seemed imprudent to resect this region in the other 4 patients without further data. None of the 5 patients had any language deficits noted as of their most recent neuropsychological testing (table 4).

**Discussion.** This study demonstrates that electrical stimulation of the left basal temporal region, including not only the fusiform gyrus (as previously noted),[13] but also portions of the lower inferior temporal and parahippocampal gyri, can interfere with at least some aspects of language processing. Three different laborato-



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**Table 3. Extent of partial left temporal surgical resections**

| Pts | Distance from anterior temporal tip resected | Limbic structures resected |
|---|---|---|
| 1 | 8 cm along basal lobe, 6 cm along lateral lobe | Amygdala, hippocampus |
| 2 | 3.5 cm over the middle gyrus, 3.0 cm elsewhere | Amygdala, 0.5 cm anterior hippocampus |
| 3 | 4 cm over entire lobe | Amygdala, pes hippocampus |
| 4 | 4 cm over middle and superior gyri | Amygdala pes hippocampus |
| 5 | 2 cm middle gyrus | Amygdala, 0.8 cm anterior hippocampus |

**Table 4. Postoperative language testing data**

| | Patient | | | | |
|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 |
| Postoperative period when tested (mos) | 12 | 6 | 1 | 4 | 1 |
| WAIS-R | | | | | |
| VIQ | 85 | 104 | 80 | 99 | 95 |
| PIQ | 99 | 105 | 81 | 119 | 111 |
| FSIQ | 90 | 104 | 79 | 107 | 100 |
| Wechsler Memory Scale | 86 | 103 | 86 | 97 | 94 |
| Auditory Comp | + | + | + | + | + |
| Word Fluency | + | + | + | + | + |
| Repetition | + | + | + | + | + |
| Naming | — | + | — | — | + |
| Reading Comp | + | + | + | + | + |

— Impaired.
+ Intact.

ries have now identified the BTLA using subdural electrodes,[13-16] including reproduction of findings by 1 investigator (R.P.L.) in 2 settings. The BTLA encompassed 2 previously described regions thought to be important for language functions: the inferior temporal gyrus, on the basis of clinicopathologic correlations,[11,12] and the fusiform gyrus, on the basis of cortical stimulation.[13,15] Perhaps because of the larger arrays used, we identified the BTLA in each patient studied. This was not the case in previous reports.[13-16] Spontaneous speech and passage reading were the most consistently impaired tasks in the patients that we studied, whereas dysnomia was most prominent in previously studied patients.[13,15] Interpatient variation is the most likely explanation for this discrepancy, although differences in testing procedures could also contribute.

The rostral-caudal dimension of this area was up to 63 mm in this study, which would include the language region defined by Lüders et al.[13] Our electrode grids covered up to 73 mm of the rostral-caudal length of the basal temporal region as opposed to the 33 mm evaluated by others.[13-15] However, the BTLA extended to the anterior margin of the electrode grids in 3 patients and the posterior margin of the electrode grids in 4 patients in this report and in another patient previously described.[13] Therefore, the true rostral-caudal length of the region may be underestimated in both studies. The length may also be underestimated due to burying of part of this region within sulci in some patients, which would interfere with stimulation from the cortical surface.

How much the results of cortical stimulation can be generalized to normal subjects is not known. Patients with epilepsy may be more susceptible to language disruption by cortical stimulation.[28] Also, the BTLA might not normally be present, but could develop due to a cerebral lesion which also results in epilepsy. However, language areas should migrate away from, not into, damaged cortex such as regions of epileptic tissue. In contrast, the BTLA was a common site of epileptiform activity in our patients. Nonetheless, we cannot state with certainty that this region exists in the normal population or even in other patients with epilepsy.

We cannot infer the exact role of the BTLA from these data. It is possible that this region is not a primary language area, but is instead connected by oligosynaptic chains to areas that are more directly involved with language. The occurrence of transynaptic effects in response to cortical stimulation has been reported.[29] Alternatively, spread of stimulation current could produce a disconnection syndrome by disrupting subcortical fiber tracts involved with language. Finally, rather than being involved in language processing per se, the BTLA could be involved with memory retrieval necessary for language processing[30,31] with the apparent disruption of language-related functions being a secondary phenomenon. The homologous region in primates has been shown to have connections to limbic structures involved with memory.[32]

There is evidence that severity of language deficits may be a function of lesion size in other language areas.[33,34] Since dominant temporal lobectomies are often limited to the anterior 4 to 5 cm of cortex (due to the location of Wernicke's area) standard resections might cause minimal damage to the BTLA and minimal functional deficits due to the variable boundaries of the region. However, in some cases, such resections might also remove a larger portion of the BTLA. In other cases, larger resections might spare this region due to a more posterior location. Since this region is seldom lesioned in isolation, perhaps previous examples of deficits due to injury of this area were presumed to reflect damage elsewhere as part of more widespread processes. It is possible that resections of this area could be a contributing factor to the previously outlined postoperative verbal (including verbal memory) language deficits following dominant temporal lobectomy (see the introduction and references 1 through 10) with greater deficits occurring with larger resections.[7] Unfortunately, limiting the extent of resection of epileptogenic cortex may reduce the likelihood of ultimate



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seizure control.[7] When epileptogenic cortex is coexistent (at least in part) with the BTLA, a cautious decision may have to be made regarding the most prudent margins of resection.

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