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Distribution of Auditory and Visual Naming Sites in Nonlesional Temporal Lobe Epilepsy Patients and Patients with Space-Occupying Temporal Lobe Lesions

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Summary: *Purpose:* Current knowledge regarding the topography of essential language cortex is based primarily on stimulation mapping studies of nonlesional epilepsy patients. We sought to determine whether space-occupying temporal lobe lesions are associated with a similar topography of language sites, as this information would be useful in surgical planning.

Methods: We retrospectively compared the topography of auditory and visual naming sites in 25 nonlesional temporal lobe epilepsy patients ("nonlesional") and 18 patients with space-occupying lesions ("lesional") who underwent cortical language mapping before left temporal resection.

Results: Both groups exhibited a similar pattern of auditory naming sites anterior to visual and dual (auditory–visual) naming sites; no group differences were specific to auditory or visual naming sites. However, significantly fewer lesional (10 of 20) compared with nonlesional patients (21 of 25) exhibited any naming sites in the temporal region ($p = 0.04$). Although the proportion of naming sites on the superior temporal gyrus was similar between groups, naming sites were found on the middle temporal gyrus in 13 of 25 nonlesional patients, yet in only one of 18 lesional patients ($p = 0.002$). Across groups, patients with visual naming sites were older than patients without visual naming sites identified ($p = 0.02$).

Conclusions: The precise location of essential language cortex cannot be reliably inferred from anatomic landmarks or patient-related variables. As time constraints are a common quandary in stimulation mapping, the different patterns reported here for patients with and without space-occupying lesions can be used to guide mapping strategies, thereby increasing the efficiency by which positive naming sites are identified. **Key Words:** Cortical language mapping—Cortical stimulation—Epilepsy—Naming sites—Temporal lobe.

The temporal lobes are a common locus for neuropathology requiring surgical intervention, including two thirds of medically intractable partial seizures with demonstrable foci, many vascular malformations and nearly 40% of gliomas (Laws et al., 1986; Semah et al., 1998; Moran et al., 1999; Yeh et al., 2005; Zhao et al., 2005). One challenge of temporal lobe surgery is to remove a sufficient amount of pathologic tissue, without removing or damaging tissue that is critical for normal language function. Although the Wernicke area, loosely considered to represent the posterior portion of the superior temporal gyrus, serves as a general landmark for eloquent cortex supporting language, research in language localization has shown considerable interindividual variability in the location of essential language cortex (Penfield and Roberts, 1959; Ojemann, 1983a). Consequently, surgical resection involving the language-dominant temporal region often requires pre-resection, stimulation-based cortical language mapping (Black and Ronner, 1987; Ojemann et al., 1989). Mapping is performed either extraoperatively, by using permanently implanted electrode grids and strips, or intraoperatively, by using electrodes placed on the cortical surface after craniotomy (Luders et al., 1987; Ojemann, 1990). Cortical sites at which electrical stimulation impedes performance on one or more language tasks are considered essential for normal language function. These positive sites (and 1 cm of adjacent cortex) are typically then spared from resection, with the goal of preserving postoperative language abilities (Ojemann and Dodrill, 1981; Hermann et al., 1999).

The clinical population composing most stimulation-mapping studies has been nonlesional epilepsy patients, and the task most often used to identify essential language cortex has been visual object naming (Ojemann, 1983b; Hermann and Wyler, 1988). Within the parameters of this

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population and this particular task, naming sites have been considered to be located in the posterior portion of the temporal region, primarily on the superior temporal gyrus (STG) and middle temporal gyrus (MTG), with some suprasylvian representation. Recent work in stimulation mapping using auditory description naming (e.g., "a pet that purrs"), in addition to visual naming, has shown that auditory naming sites (i.e., sites at which stimulation impairs auditory but not visual naming) are generally located anterior to visual naming sites or "dual" sites (i.e., sites at which stimulation disrupts both auditory and visual naming) (Hamberger et al., 2001). Similar to that found for visual naming sites, removal or encroachment on auditory naming sites is associated with postoperative word-finding decline (Hamberger et al., 2005).

Given this history of stimulation language mapping, relatively little is known regarding the topographic representation of visual naming sites in patients with space-occupying lesions, and no published studies address the topography of auditory naming sites in this population. Such information would be useful in guiding surgical planning and mapping strategies, particularly given the time constraints inherent in intraoperative mapping. In this retrospective study, we sought to examine and compare the cortical distribution of auditory and visual naming sites in nonlesional temporal lobe epilepsy patients and patients with space-occupying temporal lobe lesions. We hypothesized that space-occupying lesions might displace both auditory and visual naming sites outside the lateral temporal region, where language sites have been most frequently identified in nonlesional epilepsy patients. Specifically, we reasoned that lesional patients would have fewer positive naming sites in lateral temporal cortex compared with those demonstrated in nonlesional patients. To address this hypothesis, we retrospectively analyzed the topography of auditory and visual naming sites in 25 nonlesional and 18 lesional patients who underwent cortical language mapping before left temporal surgical resection.

METHODS

Subjects

A series of 43 consecutive patients who underwent cortical language mapping before left temporal surgical resection and met inclusion criteria was included in this study. Subjects were required to be left hemisphere language dominant, and either native English speakers or to have learned English before age 5 years. Left hemisphere language dominance was identified by intracarotid amobarbital testing, functional imaging, and/or intraoperative identification of language sites. Twenty-five of these patients had temporal lobe epilepsy and were categorized as "nonlesional," in that they had no space-occupying lesions (13 with medial temporal sclerosis as defined by MRI, 12 with no abnormality on MRI), and 18 patients, categorized as "lesional," had space-occupying temporal lobe lesions. Lesions consisted of 11 temporal tumors (two oligodendrogliomas, three gangliogliomas, one astrocytoma, one glioblastoma multiforme, two dysembryoplastic neuroepithelial tumors, one mixed oligoastrocytoma, and one low-grade glioma), five cavernous malformations, and two arteriovenous malformations. All patients with nonlesional temporal lobe pathology had medically intractable seizures, as did eight of 18 patients with temporal lobe lesions. Twenty-six patients underwent intraoperative language mapping before resection, all at Columbia University Medical Center (CUMC). The remaining 17 patients (16 nonlesional) underwent extraoperative language mapping via subdural electrodes, nine at CUMC and seven at New York University Medical Center (NYU). Demographic and clinical information for the two groups was as follows: age at surgery (nonlesional, 35.6, SD, 11.5; lesional, 30.9, SD, 12.6; $p = 0.21$), years of education (nonlesional, 14.6; SD, 3.0; lesional, 15.0; SD, 3.1; $p = 0.69$), and age at onset (onset of recurrent seizures or detection of lesion; nonlesional, 16.8 years; SD, 11.3; lesional, 27.8 years; SD, 13.9; $p = 0.007$). The group difference in onset age is addressed later. Preoperative scores for Verbal IQ (VIQ) (Wechsler, 1997), Visual naming (Boston Naming Test, Kaplan et al., 1983), and Auditory naming (Hamberger and Seidel, 2003) were available for 23 nonlesional and 13 lesional patients (see Results).

Electrodes

For patients evaluated intraoperatively (17 lesional, nine nonlesional), perisylvian sites were stimulated by using a bipolar stimulator with 2-mm-diameter ball contacts separated by 5 mm (Ojemann Cortical Stimulator; Radionics, Burlington, MA, U.S.A.). The sites were chosen based on gyral/vascular anatomy and spaced <10 mm apart. Electrode positions were documented by using digital photography and schematic diagrams. For patients who underwent extraoperative mapping (one lesional, 16 nonlesional), a 64-contact (i.e., eight-by-eight) grid array, with 5-mm-diameter electrodes embedded in Silastic with center-to-center interelectrode distances of 1 cm (Ad-Tech, Racine, WI, U.S.A.), was positioned over the temporal/perisylvian region (trimmed as needed to conform to the covered area). The exposed cortical surface and grid position were documented by digital photography and schematic diagrams. Initial schematics were drawn by the surgeon intraoperatively, while looking directly at the brain surface. Digital photographs were then used postoperatively to refine the diagrams. Additionally, subdural electrode positions were verified by skull radiographs postoperatively. The STG and MTG, >3.5 cm to ~ 8.5 cm from the temporal pole, were reliably mapped in all 43 patients. This was determined empirically by segmenting the STG, MTG, inferior temporal gyrus (ITG), and suprasylvian region into centimeter-wide sections from

the temporal pole and comparing the number of sites tested within each section between groups. Although many patients also had anterior temporal (i.e., ≤3.5 cm from the temporal pole) and suprasylvian mapping, only the areas that were comparably mapped in all patients were included in the analyses described later.

Mapping procedures

All patients performed both auditory and visual naming tasks. All auditory and visual naming stimuli were administered to patients within 1 to 4 months before surgery. Auditory and visual items, selected from previously published stimuli, were equated for word frequency and were similar in difficulty level (Hamberger and Seidel, 2003). Only items that patients successfully completed at baseline were administered during cortical mapping (items associated with word-retrieval errors at baseline could not be used to identify stimulation-related errors during mapping). For epilepsy patients, mapping was performed while antiepileptic drug (AED) levels were in the therapeutic range to minimize afterdischarges and seizure activity.

Extraoperative language mapping was conducted after video-EEG monitoring to identify the seizure-onset zone. Testing was conducted during electrical stimulation applied to adjacent electrodes. When results were positive, each electrode was studied individually and referenced to a remote electrode in "silent cortex." All available sites along lateral temporal cortex, as well as parietal sites in the perisylvian area, were stimulated.

Patients who underwent intraoperative mapping were initially anesthetized with propofol. Language mapping began after craniotomy/dural opening, electrocorticography, and stimulation to determine the threshold for afterdischarges. Several practice trials were conducted to ensure an adequate level of patient responsiveness, and that naming ability was at the level the patient demonstrated at baseline, preoperatively. Stimulation sites were primarily in the vicinity of the anticipated resection, as determined by the presence of a lesion or intracranial EEG evidence of seizure onset. If no visual naming cortex was identified, additional perisylvian sites were tested in an attempt to identify positively the visual naming cortex (rather than relying on negative responses alone). Sites were tested with a bipolar stimulator, as described earlier.

Stimulation mapping followed well-established methods (Ojemann, 1983a, 1991). For both intra- and extraoperative mapping at CUMC, a constant-current stimulator (Ojemann Cortical Stimulator, Radionics, Inc., Burlington, MA, U.S.A.) delivered a biphasic square waveform at a frequency of 60 Hz with a 2-ms pulse duration and delivered amperage ranging from 3 to 15 mA during intraoperative mapping. Mapping at NYU was conducted by using a Grass Instruments S-12 cortical stimulator (Quincy, MA, U.S.A.) with a biphasic square waveform at frequency 50 Hz with a 0.3-ms pulse duration, with amperage ranging from 3 to 15 mA. Afterdischarge levels were determined by increasing amperage until an afterdischarge was elicited, with an upper limit of 15 mA. Amperage for stimulation was set at 0.5 to 1 mA below that which elicited an afterdischarge (or 15 mA), which was determined for each site individually. Results reported here are from trials during which no afterdischarges were elicited.

At least two trials of both visual and auditory naming were conducted at each site. If results were ambiguous or the patient was temporarily inattentive, additional trials were administered. For visual naming, patients were shown line drawings of common items (e.g., bench, helicopter), and for auditory naming, patients heard oral descriptions of concrete items (e.g., "What a king wears on his head"). For visual naming, patients began with the phrase, "This is a" to enable differentiation between speech arrest and anomia, whereas, for auditory naming, patients were instructed to name the target item. To reduce differences in duration of cortical stimulation across tasks, the auditory stimuli were limited to those that contained a maximum of eight words and could be presented clearly within 4 s. Additionally, the requirement for patients during visual naming to articulate the carrier phase (i.e., This is a ———) before naming the pictured object further balanced the stimulus-processing and stimulation-duration times among tasks. For each task, electrical stimulation began immediately before presentation of pictures or auditory descriptions and lasted for a maximum of 10 s, but terminated immediately on the patient's production of a correct response. For both tasks, patients were instructed to respond as quickly as possible. Sites were considered critical for task performance if the patient could not name target items during stimulation, but provided correct responses on cessation of stimulation. When one of two trials was performed inaccurately, another two trials were administered. Sites were considered critical for task performance only when responses to both of these two trials were incorrect. Sites at which this further testing resulted in 50% accuracy were not considered critical for task performance. Additionally, if it became apparent during the procedure that naming abilities deteriorated to a level below that demonstrated at baseline, mapping was discontinued.

Data analysis

For each patient, the location of electrode sites was determined by intraoperative digitized photographs and schematic drawings, and supplemented by postoperative skull radiographs. Naming sites from each patient were plotted on a schematic of the temporal lobe region and coded to indicate whether auditory, visual, or both auditory and visual naming were disrupted by stimulation. The topographic distribution and proportion of auditory and visual naming sites in the STG versus the MTG were

compared in lesional and nonlesional patients by using χ^2 analysis or Fisher's exact test, depending on cell sizes. Although some patients had more extensive mappings that included anterior and inferior temporal cortex, group comparisons of the topography of naming sites included only the STG and MTG regions that were reliably mapped in all patients. T tests were used for comparisons of demographic and patient-related data. Pearson correlations were performed to assess the relation between the number of naming sites identified per patient and patient-related variables. Data from auditory and visual naming were analyzed separately, and subsequently grouped together when no differences were found in the modality-specific results.

RESULTS

Overall, patients in the nonlesional group were more likely to have naming sites identified relative to patients in the lesional group (Fig. 1). Of the 25 nonlesional patients, at least one positive naming site was found in 21 patients, whereas, of the 18 lesional patients, only 10 patients exhibited any positive naming sites ($p = 0.04$). No group differences were noted with respect to the number of patients who exhibited auditory ($p = 0.63$) or visual ($p = 0.10$) naming sites.

Although fewer sites were identified in the lesional compared with the nonlesional group, the spatial patterns of auditory naming sites anterior to visual naming sites was similar and significant in both groups (nonlesional $p = 0.001$; lesional $p = 0.013$). However, the superior/inferior distribution of positive naming sites (both auditory and visual), differed. Specifically, 17 of 25 nonlesional patients (25 sites) and 10 of 18 lesional patients (18 sites) had sites identified on the STG ($p = 0.40$), whereas 13 of 25 nonlesional patients (23 sites) yet only one of 18 lesional patients (one site) had sites identified on the MTG ($p = 0.002$).

Although most lesional patients were tested intraoperatively and most nonlesional patients were tested extraoperatively, no group differences were found in the number of sites tested in lesional (mean, 12.4; SD, 6.5) versus nonlesional patients (mean, 14.2; SD, 5.7; $p = 0.35$). As noted, analyses of mapping results were limited to the temporal areas that were reliably mapped in both groups. Thus the number of naming sites identified is not merely a function of the number of sites or the size of the region tested. Additionally, no significant differences were seen in number of naming sites identified in patients with (mean, 2.2; SD, 1.9) versus without epilepsy (mean, 1.7; SD, 3.1; $p = 0.53$). To determine whether the presence of naming sites in the lesional group was related to lesion location, lesion locations were classified as "anterior" if the posterior margin of the lesion was <5 cm from the temporal pole, and as "posterior" if the anterior margin of the lesion was >5 cm from the temporal pole. Of the six patients with anterior lesions, three had positive naming sites, and of the 12 patients with posterior lesions, seven had positive naming sites ($p = 1.0$). Thus to the extent we were able to quantify lesion location, this did not account for the presence or absence of positive naming sites. Analysis of lesion type also showed no consistent relation with presence or absence of naming sites ($p = 0.67$).

As noted earlier, age at onset was significantly earlier in the nonlesional group. Although onset age showed no systematic correlation with number of naming sites identified ($r = -0.04$; $p = 0.82$), it should be noted that correlations involving number of naming sites are likely limited by the restricted range. The number of naming sites identified per patient ranged from none to 10, yet with a mean of 2.1 and a median of 2. T tests comparing patients with and without any naming sites identified (likely a more valid means of analyzing these data) showed no significant differences in onset age ($p = 0.79$), VIQ ($p = 0.23$), or education level ($p = 0.26$). However, patients with sites identified were older at the time of surgery (mean age, 36.8 years; SD, 12.1) than were patients with no sites identified (mean age, 25.8; SD, 7.8; $p = 0.006$). This difference was specific to the presence of visual (not auditory) naming sites (visual naming sites found: mean age, 37.7; SD, 10.7; visual naming sites not found: mean age, 29.2; SD, 12.2; $p = 0.02$). As noted earlier, age at the time of surgery was comparable between lesional and nonlesional groups ($p = 0.23$).

Preoperative scores on verbal measures were available for 23 nonlesional patients and 13 lesional patients (Table 1). As shown, VIQ and visual naming were significantly stronger in the lesional group; auditory naming was similar in direction, but the group difference was not significant. Neither VIQ ($r = 0.04$; $p = 0.80$) nor Visual naming ($r = 0.17$; $p = 0.33$) correlated with the number of auditory or visual naming sites identified. Additionally, no differences in verbal scores were noted between patients who did and did not have naming sites identified (VIQ: $p = 0.23$; Visual naming: $p = 0.66$; Auditory naming: $p = 0.24$). Of note, VIQ scores for both lesional and nonlesional patients were well within the average range, whereas visual and auditory naming scores were below the average range in both groups with impaired naming performance in the nonlesional group.

TABLE 1. *Verbal test scores*

	Lesional	Nonlesional	p Value
VIQ	106.2 (10.0)	94.7 (14.1)	0.021
Visual Naming (BNT)	52.9 (8.6)	46.2 (8.8)	0.040
Auditory Naming	47.6 (3.9)	45.2 (4.4)	0.133

Values expressed as mean (SD).

VIQ, verbal IQ; Visual Naming maximum score, 60; Auditory Naming maximum score, 50.

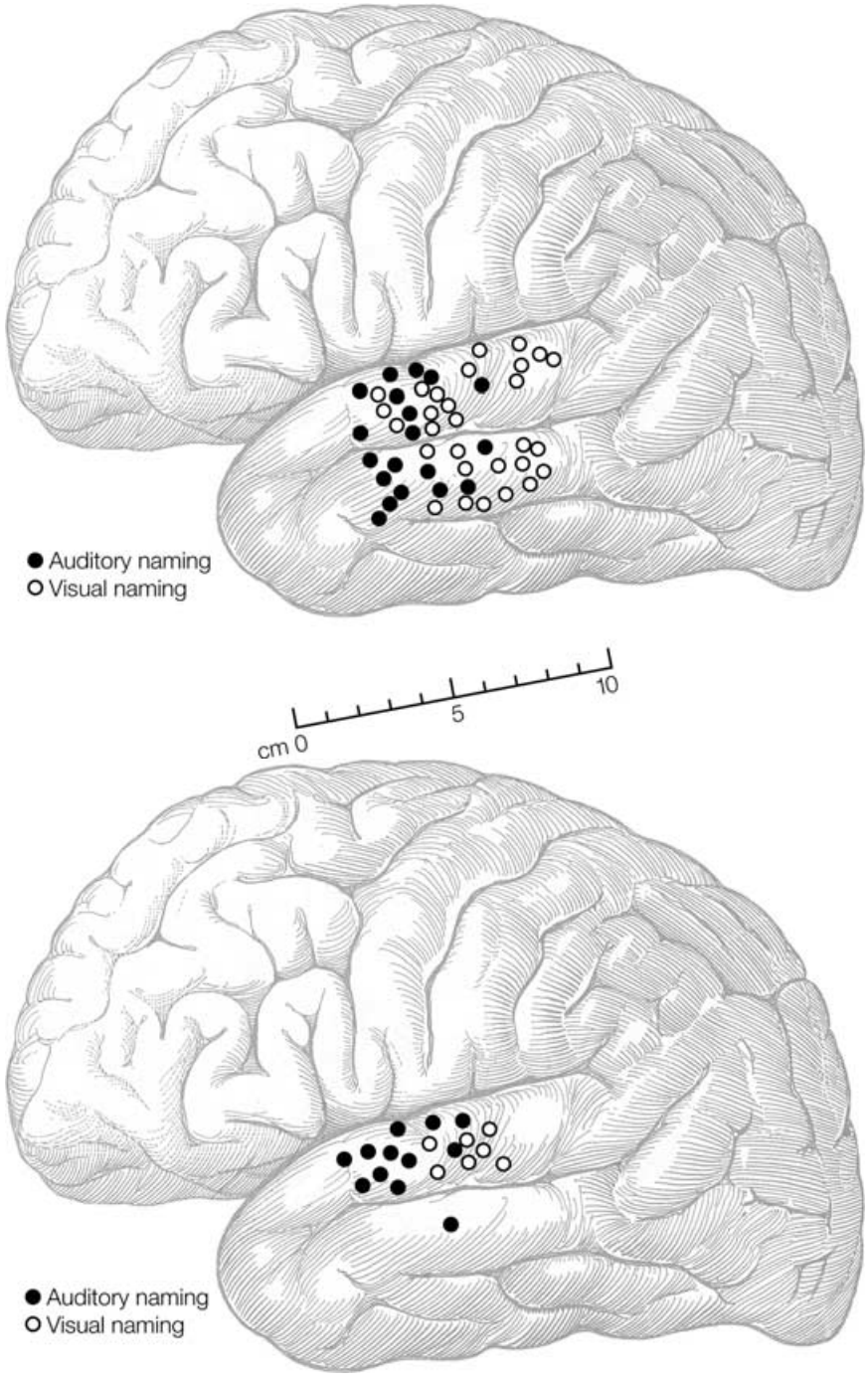

FIG. 1. Topographic distribution of auditory (*solid circles*) and visual (*open circles*) naming sites in nonlesional epilepsy patients **(A)** and patients with space-occupying temporal lobe lesions **(B)**.

DISCUSSION

Previous work in stimulation language mapping describes, primarily, the topography of visual naming sites in nonlesional epilepsy patients. In this study, we sought to elucidate the cortical distribution of both auditory and visual naming sites in both nonlesional epilepsy patients and in patients with space-occupying lesions in the temporal lobe. Consistent with our hypothesis, we found that lesional patients were less likely to have any naming sites identified in lateral temporal cortex. Additionally, whereas naming sites in nonlesional patients were scattered equally across the STG and MTG, naming sites were rarely found on the MTG in lesional patients, with most naming sites clustered in the posterior portion of the STG. In both groups, however, auditory naming sites were generally anterior to visual naming sites, concordant with previous findings (Hamberger et al., 2001).

The consistency in the anterior/posterior distribution of auditory and visual naming sites across groups is not surprising, as this maintains the spatial relation between auditory and visual association areas. The lower proportion of lesional patients with positive naming sites is consistent with results of a previously reported series that included 40 patients with temporal gliomas and 83 nonlesional epilepsy patients (Haglund et al., 1994). However, unlike our findings, this previous series found a lower percentage of STG naming sites in the lesional group and a lower proportion of MTG relative to STG naming sites in both groups. Although our use of auditory naming provided more positive sites overall, the use of auditory naming in our study does not appear to account for these differences, as no group differences in topography were specific to auditory versus visual naming.

One possible explanation for the overall fewer positive sites in the lesional group is that as a space-occupying lesion expands, existing sites might be altered or eliminated (Haglund et al., 1994). It should be noted, however, that stimulation mapping is limited spatially by clinical and patient-related factors (see later) or by the extent of subdural grid coverage. Thus it is possible that in some patients, naming sites may have been located in unexpected areas that were not tested. Additionally, it is infrequent that patients who undergo stimulation mapping require remapping at a subsequent time; however, repeated mapping would potentially address the question of changes in language site location over time. Although simulation mapping is the standard of care for clinical purposes, functional imaging carries the advantage of repeatable testing and has been used to demonstrate changes in both intra- and interhemispheric language representation after a stroke (Thulborn et al., 1999).

The literature on language reorganization after left hemisphere insult is notable for controversy regarding the nature and location of pathology or injury associated with cortical reorganization. As we limited our study to left hemisphere–dominant patients, we were interested specifically in intrahemispheric language organization in these two groups. Whereas some investigators have concluded that epileptogenic tissue does not typically displace language cortex (De Vos et al., 1995; Duchowny et al., 1996), others report otherwise (Rausch et al., 1991; Devinsky et al., 1993; Schwartz et al., 1998). In addition to nature and location of insult, patient-related factors such as IQ, age at onset, and educational attainment have been shown to correlate with location and quantity of positive naming sites (Ojemann, 1983a; Devinsky et al., 1993). Although both VIQ and age at onset differed significantly between our lesional and nonlesional groups (i.e., higher VIQ and later onset age in lesional group), we found no systematic relation between VIQ or onset age and the presence/absence of positive naming sites, consistent with other reports (Duchowny et al., 1996; Liegeois et al., 2004). The more-superior/posterior representation of naming sites in the lesional group is concordant with theories that the maintenance of left hemisphere dominance in patients with left temporal lesions is accomplished by reorganization of speech to adjacent posterior structures in the left hemisphere (Rasmussen and Milner, 1977) that reach functional maturation and commitment later in childhood (Fennell et al., 1977; Satz et al., 1988, 1990). Our findings are also consistent with results of a relatively recent functional imaging study, suggesting that either physical displacement or intrahemispheric reorganization of language occurs in patients with space-occupying lesions (Stowe et al., 2000). Although it is possible that the development of a space-occupying lesion merely pushes aside normal brain tissue, resulting in fewer sites identified within the region studied, several investigators have reported the presence of functional tissue (including language cortex) within clearly tumor-infiltrated brain regions (Ojemann et al., 1996; Skirboll et al., 1996; Duffau et al., 2004). Thus the relation between functional and abnormal tissue does not appear to be straightforward (Haglund et al., 1994).

An alternative explanation to consider, particularly given the lower baseline verbal scores and earlier onset age in the nonlesional group, is that the pattern of positive sites in the lesional group is actually a closer approximation of normal language organization than that observed in the nonlesional group. Accordingly, the location of naming sites in the posterior portion of the STG is consistent with the purported location of the Wernicke area inferred from studies of stroke patients (Brust et al., 1976; Mazzocchi and Vignolo, 1979), which, although lacking in resolution, likely reflects normal language organization. In keeping with this line of thought, the broad distribution of naming sites in nonlesional epilepsy patients might reflect the development of additional naming sites, perhaps in an attempt to compensate for damage to the original language area caused by chronic epileptiform activity.

It is also reasonable to take a more theoretic stance and consider what positive naming sites might actually represent. In the current data set, we found that individuals with positive (visual) naming sites were older than individuals without any naming sites identified. Although we tend to infer structure/function relations from positive naming sites, we can state with certainty only that stimulation at a particular location disrupts the task at hand. Thus it is possible that both chronic epilepsy and aging induce changes in cortical function such that normal function is more readily disturbed by stimulation (i.e., increasing the likelihood of finding positive naming sites). This perspective is most consistent with the position that the lesional group provides a closer representation of "normal" organization than does the nonlesional group.

A limitation of the current study is the relatively restricted region of cortex that was compared between groups. Although analysis of a broader region of cortex would have been preferable, practical limitations, such as time constraints, patient fatigue and cooperation, clinical concerns, and IRB regulations, render such data difficult to obtain.

The main limitation of stimulation mapping, in general, is that it is invasive; only pathologic populations can be studied. Thus the patterns identified in the two populations studied here might both represent abnormal variants: one due to chronic, irritative electrophysiologic activity, the other due to structural displacement. In accordance with this, the finding of weak baseline performance on both visual and auditory naming in both groups suggests that neither group is entirely "normal," at least from a functional perspective. Perhaps discerning both the consistencies and discrepancies between stimulation mapping data from patient populations and functional imaging data from normal subjects by using similar tasks might help elucidate the normal cortical organization of language.

Given the inconsistencies in the literature on language localization after left temporal insult, it is reasonable to conclude that the precise localization of essential language cortex cannot be reliably inferred from anatomic landmarks or from demographic or patient characteristics. The findings reported here demonstrate two general patterns that can be used to guide the search for language cortex in patients with and without space-occupying lesions. Stimulation mapping, which remains the gold standard for identifying essential language cortex, is a time-intensive, yet also a time-constrained, procedure. It is hoped that the current results will increase the efficiency by which positive sites are identified.

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