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Does cortical mapping protect naming if surgery includes hippocampal resection?

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Abstract

Objective—Pre-resection electrical stimulation mapping is frequently used to identify cortical sites critical for visual object naming. These sites are typically spared from surgical resection with the goal of preserving postoperative language. Recent studies, however, suggest a potential role of the hippocampus in naming, although this is inconsistent with neurocognitive models of language and memory. We sought to determine whether preservation of visual naming sites identified via cortical stimulation mapping protects against naming decline when resection includes the hippocampal region.

Methods—We assessed postoperative changes in visual naming in 33 patients, 14 who underwent left temporal resection including hippocamal removal and 19 patients who had left temporal resection without hippocampal removal. All patients had preresection cortical language mapping. Visual object naming sites identified via electrical stimulation were always preserved.

Results—Patients without hippocampal resection showed no significant naming decline, suggesting a clinical benefit from cortical mapping. In contrast, patients who had hippocampal resection exhibited significant postoperative naming decline, despite pre-resection mapping and preservation of all visual naming sites ($P \leq .02$). These group effects were also evident in individual patients ($P = .02$). More detailed, post hoc examination of patients who had hippocampal resection revealed that overall, patients who declined were those with a preoperative, structurally intact hippocampus, whereas patients with preoperative hippocampal sclerosis did not exhibit significant decline.

Interpretation—Despite cortical language mapping with preservation of visual naming sites from resection, removal of an intact dominant hippocampus will likely result in visual naming decline postoperatively.

INTRODUCTION

Stimulation-based cortical mapping came into clinical use in the early 1900's in association with surgical resection of epileptogenic cortex in patients with pharmacologically refractory epilepsy 1. The procedure involves brief electrical stimulation directly to the cortical surface to identify areas critical for function. For motor and sensory cortex, stimulation produces

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positive responses such as movement or sensation. Language mapping, however, relies on negative responses in that stimulation disrupts performance of a language task. Language sites identified via cortical mapping are typically spared from resection, with the goal of preserving postoperative language function 2. Although a wide range of tasks have been utilized, the most widely used task is visual object naming 3, 4. Preservation of naming sites in particular is considered protective for postoperative language function 4, 5.

Temporal lobe resection, which offers a high likelihood of seizure freedom, also carries the risk of cognitive decline in episodic and semantic memory when surgery involves the language-dominant hemisphere. Naming decline, the most common form of postoperative semantic memory change 6, has traditionally been attributed to resection of lateral temporal cortex 7–9. Accordingly, clinical use of cortical language mapping prior to temporal lobe surgery reflects a widely held belief that naming, and likely most other semantic memory functions, are mediated by the temporal/temporoparietal region 7. The other main component of this neuro-functional model proposes that episodic memory (i.e., memory for personal events) is mediated primarily by the hippocampal region and that these two "memory" systems are functionally and anatomically distinct 10, 11. This model is based on numerous studies of patients with naturally occurring or surgically induced lesions 12–14.

Interestingly, and relevant to this discussion, recent findings suggest that the hippocampus, (i.e., presumably an episodic memory structure), is involved in visual object naming, (i.e., a semantic memory process). These findings include poorer visual naming in patients with left hippocampal sclerosis (HS) compared to those with structurally normal hippocampi 15, 16, and greater decline in visual naming following left anteromedial temporal lobe resection (AMTLR) without mapping in patients with structurally normal left hippocampi than in patients with compromised hippocampal integrity due to HS 16, 17. Additionally, significant correlations have been reported between naming performance and hippocampal metabolism, measured by ^{1}H- magnetic resonance spectroscopy (^{1}H-MRS) 18, 19 and hippocampal volume, measured by structural MRI 20. Furthermore, some studies have shown greater postoperative naming decline in patients with later age of seizure onset and shorter epilepsy duration, both of which are typically associated with the absence of HS (i.e., removal of a structurally intact hippocampus).

The possibility that the hippocampus plays a critical role in visual naming raises questions regarding the clinical benefit of cortical mapping when surgery includes ashippocampal resection. Early work assessing the efficacy of language mapping involved relatively few patients and demonstrated greater decline on an aphasia screening test when resection boundaries were within 2 cm of a naming site compared to the decline observed with a wider resection margin 5. A subsequent, larger study that included both intractable temporal lobe epilepsy patients and patients with temporal lobe gliomas found significantly fewer postoperative naming deficits when the resection boundary was greater than 1 cm from the resection margin 21. However, in both of these studies, it was unclear how many, or which patients underwent hippocampal resection, and whether hippocampal resection had any influence on postoperative naming.

We sought to determine whether hippocampal removal compromises preresection cortical language mapping. We assessed postoperative visual naming changes in patients who underwent left temporal resection including hippocamal removal and patients who had left temporal resection without hippocampal removal, with both groups having had preresection cortical language mapping. We hypothesized that despite preresection language mapping, visual naming would decline following hippocampal removal, whereas visual naming would not decline following resections excluding the hippocampus.

METHODS

Subjects

Subjects were 33 consecutive patients who underwent cortical language mapping before left temporal resection and met inclusion criteria. Subjects were required to be left hemisphere language-dominant, native English speakers or to have learned English by age five, and to have been fully educated in English. Language-dominance was identified by Wada testing (n = 26), fMRI (n = 5), or intraoperative identification of language sites plus postictal speech disturbance,22 consistent with left hemisphere language-dominance (n = 2). Nineteen "Hippocampus Resected" patients had temporal lobe resection including medial temporal structures. Fourteen "Hippocampus Preserved" patients underwent left temporal/ temporoparietal resection without removal of medial temporal structures. Patient information is presented in Table 1. There were no significant group differences in age, education, gender, postoperative interval or epilepsy duration; differences in IQ approached, but did not reach statistical significance. The significant difference in onset age is addressed below (Results). This study was approved by the Institutional Review Board at Columbia University Medical Center (CUMC).

Pre and Postoperative Testing

Two visual naming instruments, the Boston Naming Test (BNT)23 and the Visual Naming Test (VNT) 24, were administered pre and postoperatively. Both tests require naming of line drawn objects (all VNT and BNT items are distinct) within 20 seconds. The VNT contains familiar, mid to high frequency items whereas the BNT contains a number of low frequency items (e.g., sphinx). VNT instructions emphasize rapid responding. Normative data are available for accuracy (i.e., number correct), response time (RT), and tip-of-the-tongue responses ("TOT," number correct within 2–20 seconds, or following phonemic cueing, e.g., "ha" for "hammer"). The BNT provides normative data only for number correct. We calculated an additional measure comprised of items not named within 20 seconds, yet subsequently named following phonemic cueing ("BNTcue").

Given the clinical significance of seizure freedom, seizure outcome was analyzed by dichotomizing patients as seizure free (i.e., Engel's classification 25, class I) versus not seizure free (i.e., classes II, III and IV). Results of chi square analysis indicted no significant group difference in the proportion of seizure free patients (P = .28). Seizure outcome rates in each group were as follows: Hipocampus Preserved: class I: 50%, class II: 21.5%, class III 21.5%, class IV: 7%; Hippocampus Removed: class I: 68.5%, class II: 26.5%, class III 5%, class IV: 0.

Surgical Procedure

Hippocampus Resected patients had resection of medial temporal structures; 17/19 underwent "standard" AMTLR: 3.0–3.5 cm of the anterior middle and inferior temporal gyri and fusiform gyrus 26. Two patients underwent selective amygdalohippocampectomy due to a temporary change in the standard of care for medial temporal lobe epilepsy patients at CUMC. In all Hippocampus Resected patients, the basolateral amygdala anterior to the choroidal point of the hippocampus and parahippocampal gyrus were resected.

Hippocampus Preserved patients underwent resection of epileptogenic cortex identified via subdural electrode grid recording (n = 5) or resection of a space-occupying lesion plus surrounding epileptogenic cortex, determined via intraoperative ECoG. The amount and location of the neocortical resections differed between the two groups, but overlapped substantially. Resections among Hippocampus Preserved patients were individualized, but often included more posterior portions of the middle temporal gyus and inferior temporal

gyrus and some included the superior temporal gyrus. Cortical resection sizes within Hippocampus Preserved group ranged from 1.5–4.25 cm for the superior temporal gyus, 1.5–4.25 cm for the middle temporal gyus, and 2.0–4.0 cm for the inferior temporal gyrus. In all patients, visual naming sites were spared, with a 1–2 cm margin from the resection boundary.

Electrodes

In the Hippocamus Resected group, 15 patients had extra-operative mapping and four patients had intra-operative mapping, whereas in the Hippocampus Preserved group, five patients had extra-operative mapping and nine patients had intra-operative mapping (Fisher's Exact, P = .03). However, most relevant, there were no group differences in the number of sites tested per patient (Hippocapmpus Resected mean = 24.68 sites (SD = 12.10) Hippocamus Preserved: mean = 18.36 (SD = 11.14; P = .13).

Intra-operatively, cortical sites were stimulated using a carbon-tipped bipolar stimulating electrode with 2 mm diameter ball contacts separated by 5 mm (Ojemann Cortical Stimulator, Radionics Inc.). Sites were spaced less than 10 mm apart. For extra-operative mapping, an eight by eight (i.e. 64 contact) grid array, with 5 mm diameter electrodes embedded in silastic with center to center inter-electrode distances of 1 cm (Ad-Tech, Racine, Wisconsin), was positioned over the frontal-parietal-temporal region (trimmed to conform to covered area). Grid position was documented by digital photography and schematic diagrams. Subdural electrode positions were verified by skull X-rays, postoperatively.

Mapping Procedures

Mapping was conducted while antiepileptic drug levels were in the therapeutic range to minimize afterdischarges and seizure activity. Extra-operatively, stimulation was applied to adjacent electrodes. When positive, each electrode was studied individually, referenced to a different adjacent "silent" electrode, if possible, or to a remote "silent" electrode. Intra-operative patients were initially anesthetized with propofol. Practice trials ensured adequate patient responsiveness. Stimulation sites were primarily in the vicinity of the resection, determined by lesion location or intracranial EEG evidence of seizure onset. If no visual naming cortex was identified, additional perisylvian sites were tested with the goal of identifying visual naming cortex (rather than relying on the absence of naming sites).

Bipolar stimulation mapping parameters followed well established methods 4, 27. For both intra- and extraoperative mapping, a constant current stimulator (Ojemann Cortical Stimulator, Radionics Inc.) delivered a biphasic square waveform at a frequency of 60 Hz with a 1 msec pulse duration and amperage ranging from 3–15 mA during extra-operative mapping and 2–12 mA during intra-operative mapping. Results were considered valid if no afterdischarges were elicited. Both visual naming and auditory description naming were tested at each site; however, surgical boundaries were determined using only visual naming results. A minimum of two trials per task were conducted at each site. If results were ambiguous additional trials were administered. Patients were shown line drawings of common items and instructed to say, "This is a …" Stimulation began immediately before item presentation and lasted a maximum of 10 seconds, terminating immediately upon the production of a correct response. Trials were considered positive if the patient could not name the item during stimulation, but responded correctly upon stimulation cessation. Sites were considered critical for task performance when a minimum of 75% of responses were inaccurate.

Statistical Analyses

Within-group differences between pre and postoperative naming scores were assessed via paired-sample T-tests. Independent sample T-tests and Fishers Exact test assessed group differences. To define "clinically significant" change between pre and postoperative naming scores in individual patients, we used reliable change indices ("RCIs") 28› 29, which provide change scores that exceed normal variability due to measurement error and potential practice effects. RCIs for the VNT 24 and BNT 29 were calculated using test-retest data from nonsurgical epilepsy patients.

RESULTS

Group comparisons of preoperative naming scores revealed no significant baseline differences for any of the five naming scores (all P >.20). Additionally, there were no group differences in the number of naming sites identified via cortical mapping (mean per patient Hippocampus Resected: 1.32, SD 1.56; Hippocampus Preserved: 1.29, SD 1.97; P = .96). However, groups showed clear differences in naming changes postoperatively (Table 2A–B).

Despite mapping and sparing of visual naming sites, the Hippocamapus Resected group exhibited significant decline in all five visual naming scores, reflecting a reduction in number of items correct, increased naming latencies and greater reliance on phonemic cueing. In contrast, the Hippocampus Preserved group exhibited no significant decline in visual naming.

Individual change

Given these group differences, we sought to determine whether these effects would be evident in individual patients. We classified patients as having declined if they met or exceeded RCI values for any visual naming scores (except BNTcue, as RCIs are not available), bearing in mind that RCIs represent stringent criteria, as naming changes that fail to meet RCI values might also be clinically meaningful. Nevertheless, results of Fisher's Exact test indicated that the group pattern results were evident in most individuals (P = .02; Table 3).

Hippocampal integrity

As noted, there is evidence that naming decline is greater in patients without HS relative to those with HS following left AMTLR. To determine whether pre-resection mapping with preservation of visual naming sites might protect against naming decline in patients without HS, we further examined naming changes among patients who had hippocampal resection, comparing patients with HS (n = 9) with those without HS (n = 10) (see Table 4A–B).

Similar to previous reports on patients without cortical mapping, and despite a smaller sample, nonHS patients exhibited significant decline on all but one naming score, whereas the HS group exhibited no significant decline, approaching significance on only one of five naming scores. Further examination of individual patients using RCIs revealed naming decline in 9/10 nonHS patients and 5/9 HS patients. Although Fisher's Exact test was not significant (P=.14), likely related to the small sample size, results suggest that despite mapping, removal of an intact hippocampus is predictive of visual naming decline.

As noted, previous studies report greater naming decline with later seizure onset age and shorter epilepsy duration. Interestingly, onset age was significantly later and duration tended to be shorter in the Hippocampus Preserved group (Table 1), which showed no significant naming decline.

Location of visual naming sites

Given the relatively common practice of performing AMTLR in medial temporal lobe epilepsy patients without cortical language mapping, it would be important to note the frequency in which visual naming sites fell within these resection boundaries. In our sample, none of the patients in the “Hippocampus Removed” group and only one patient in the “Hippocampus Preserved” had a visual naming site within the region of a “standard” resection (2 cm from temporal pole on middle temporal gyrus).

DISCUSSION

Visual object naming as well as other language and semantic memory processes, has generally been believed to be mediated by lateral temporal/temporoparietal cortex. Given recent findings implicating the hippocampus in the mediation of visual naming, we investigated whether pre-resection cortical language mapping protects against visual naming decline when temporal lobe resection includes hippocampal removal. Specifically, we assessed naming changes following temporal lobe resection preceded by cortical language mapping in patients with hippocampal removal and patients whose surgery was limited to neocortical resection.

As expected, patients without hippocampal resection showed no significant naming decline, suggesting a clinical benefit from cortical mapping. In contrast, and consistent with our hypothesis, patients who had hippocampal resection exhibited significant naming decline, despite pre-resection mapping and preservation of visual naming sites. Closer examination of this group revealed that overall, patients who declined were those with a preoperative, structurally intact hippocampus, whereas as a group, patients with preoperative HS did not exhibit significant decline. These results are consistent with studies of non-mapped patients, demonstrating greater naming decline in patients without HS 6.

Although previous reports of naming decline following AMTLR were suggestive of hippocampal involvement in naming, the absence of pre-resection language mapping left open the possibility that the true source of naming decline was the removal of unidentified cortical naming sites in the absence of cortical mapping. The current results suggest that even with sparing of stimulation-identified naming sites, hippocampal removal results in naming decline. Also relevant are results from a previous study comparing the location of naming sites in patients with HS and without HS, showing that patients with a structurally normal dominant hippocampus were more likely to have naming sites within the anterior portion of the temporal lobe 30, i.e., the cortical region removed with AMTLR, a procedure typically performed without preresection language mapping 6. Those results suggested that nonHS patients were more likely to have cortical naming sites removed with AMTLR, which could potentially explain why patients without HS were more likely to exhibit postoperative naming decline. However, the current results, in which visual naming sites were preserved, yet, naming nevertheless declined following removal of a structurally intact hippocampus, strengthens the argument that the hippocampus is not merely *involved* in visual naming, but represents a critical component of the neural system that mediates visual object naming.

These findings are somewhat at odds with traditional thinking, thereby raising both clinical and theoretical questions. The most relevant clinical question is whether cortical language mapping bears any value in patients who require hippocampal resection. It remains unknown whether naming decline would have been more severe had cortically based visual naming sites been removed along with medial structures. This question was also raised from a large, retrospective multicenter study that found no difference in naming decline following AMTLR between patients who did and did not receive pre-resection mapping 31. A

definitive answer to this question would require a randomized study involving sparing versus removal of stimulation-identified visual naming sites in patients without HS.

It is also reasonable to question why the hippocampus, which is indisputably the primary brain structure in the episodic memory system, would play an essential role in visual object naming. Unlike the traditional memory models, which propose a clear distinction between episodic and semantic memory, more recently developed models such as multiple trace 32 and connectionist 33 theories allow for functional and anatomical overlap between semantic and episodic memory. Nevertheless, these models would also fail to place a semantic memory process such as object naming under the functional domain of the hippocampal region. That said, perhaps the critical processing component provided by the hippocampus is not linguistic. In a non-memory, intracranial event-related potential study requiring patients to distinguish between line drawings of meaningful and meaningless objects, hippocampal responses differentiated between the two 34. Further removing any potential linguistic contribution, ablation and neurophysiological studies of both nonhuman primates and rodents implicate the hippocampal region as primarily responsible for object recognition and identification, possibly by serving as the final stage in the ventral visual cortical pathway that represents stimulus features 35. Thus, the critical component provided by the hippocampus might be pre-linguistic, yet nevertheless, inseparable from visual object naming.

Potential Limitations

A common difficulty in clinical research is non-randomization. Although groups appeared comparable regarding relevant demographic and clinical factors, other unknown factors might have contributed to group differences. As noted, the amount and location of neocortical resection overlapped, yet differed between the two groups. Nevertheless, despite superior temporal gyrus resection in some patients and the more posterior resections in the Hippocampus Preserved group, naming decline was greater among patients who underwent hippocampal resection. Another factor to consider is that it was not possible to disentangle potential contributions to visual naming from the hippocampus itself and potentially critical connections between the hippocampus and essential neocortical naming areas. There might also be contribution from other structures affected by surgery, such as the amygdala and paraphippocampal gyrus, although the different outcomes in patients with and without HS implicate the hippocampus as playing a critical role. Assessment of visual naming in patients who undergo targeted hippocampectomy, perhaps via gamma knife surgery 36, might better answer questions regarding unique hippocampal contribution to visual naming. Similarly, stimulation to discrete areas within the hippocampus and adjacent structures might better define non-cortical areas critical for naming. Finally, given reports of both behavioral and cortical dissociations between visual naming and auditory-based naming 24, 37, a more comprehensive study using auditory description naming during stimulation, and assessing a wider array of language functions pre and postoperatively would more thoroughly assess the clinical value of pre-resection mapping in patients who undergo hippocampal resection.

In answer to the question, “Does mapping protect naming when surgery includes hippocampal resection,” it is important to consider that cognitive systems are complex, and the neural substrates essential for a given function are often distributed anatomically 38. Accordingly, it is not always possible to map and/or spare all essential areas, particularly within the clinical context. What can be gleaned from this study is that patients with an intact dominant hippocampus who require hippocampal resection will likely exhibit visual naming decline postoperatively, despite mapping. Nevertheless, hippocampal resection in particular is associated with good long term seizure outcome 39, and so the risk of naming decline must be weighed against the potential benefit of improved seizure outcome. The

functional significance of this decline remains to be determined, and is currently under study in our laboratory. In the interval, we will continue to operate under the assumption that to some extent, cortical language mapping is beneficial, in the hope that we are maximizing the likelihood that patients will achieve the best possible functional outcome.

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Table 1

Demographic and Clinical Information

	Hippocampus Resected (n = 19)	**Hippocampus Preserved (n = 14)**	**P value**
Age (years)	34.58 (11.58)	36.57 (14.88)	.67
Education (years)	14.73 (2.94)	14.64 (3.62)	.94
Male/Female	6/13	8/6	.14
IQ	96.53 (13.74)	106.50 (14.19)	.08
Age of seizure onset (years)	15.89 (7.82)	26.35 (15.11)	.03
Epilepsy Duration (years)	18.6 (15.7)	10.8 (13.5)	.15
Neuropathology	HS: 9 No structural pathology: 10	Temporal tumor: 5 (1 ganglioglioma, 1 glioblastoma multiforme, 1 dysembryoplastic neuroepithelial tumor, 1 ependymoma, 1 low-grade glioma) Cavernous malformation: 4 No structural pathology: 4	
Months from surgery to postoperative testing	15.11 (SD = 7.4)	14.75 (SD = 6.5)	.89

Mean (SD); IQ based on WAIS-R or WAIS-III 40 Full Scale IQ.

Table 2

Pre and postoperative visual naming scores in patients with resected versus preserved hippocampus.

Table 2A. Hippocampus Resected			
	Preoperative Score	**Postoperative Score**	**P value**
VNT Number Correct	48.84 (1.07)	47.42 (2.79)	*.02
VNT RT	1.19 (0.41)	1.50 (0.51)	*.02
VNT TOT	4.95 (4.28)	7.74 (4.85)	*.02
BNT Number Correct	47.11 (7.92)	40.79 (9.12)	*<.01
BNTcue	5.83 (3.18)	8.56 (4.17)	*.02

Table 2B. Hippocampus Preserved			
	Preoperative Score	**Postoperative Score**	**P value**
VNT Number Correct	48.43 (3.08)	49.36 (1.08)	.14
VNT RT	1.25 (0.69)	1.39 (0.70)	.31
VNT TOT	5.93 (8.93)	5.57 (5.51)	.81
BNT Number Correct	51.46 (11.06)	54.00 (7.05)	.24
BNTcue	5.50 (7.55)	3.08 (2.90)	.21

Mean (SD),

* P < .05 VNT = Visual Naming Test, BNT = Boston Naming Test (VNT maximum Number Correct = 50, BNT maximum Number Correct = 60). For number correct, higher scores indicate better performance; for RT, TOT and BNTcue, lower scores indicate better performance.

Table 3

Number of patients showing RCI decline in each group

	RCI Decline	No RCI Decline
Hippocampus Removed	14	5
Hippocampus Preserved	4	10

Table 4

Pre and postoperative visual naming scores in patients who had hippocampus resected: Patients with HS (A) and without HS (B).

Table 4A. Patients with Hippocampal Sclerosis			
	Preoperative Score	**Postoperative Score**	**P value**
VNT Number Correct	49.33 (.86)	48.67 (1.50)	.26
VNT RT	1.25 (0.32)	1.37 (0.49)	.39
VNT TOT	4.78 (3.70)	6.11 (3.95)	.33
BNT Number Correct	47.44 (7.50)	44.33 (7.89)	.10
BNTcue	5.22 (2.58)	9.00 (4.17)	.06

Table 4B. Patients without Hippocampal Sclerosis			
	Preoperative Score	**Postoperative Score**	**P value**
VNT Number Correct	48.40 (2.17)	46.30 (3.26)	*.04
VNT RT	1.14 (0.49)	1.62 (0.52)	*.03
VNT TOT	5.10 (4.95	9.20 (5.30)	*.03
BNT Number Correct	46.80 (8.67)	37.60 (9.34)	*<.01
BNTcue	6.30 (3.56)	7.60 (4.40)	.31

Mean (SD),

* P < .05 VNT = Visual Naming Test, BNT = Boston Naming Test (VNT maximum Number Correct = 50, BNT maximum Number Correct = 60). For number correct, higher scores indicate better performance; for RT, TOT and BNTcue, lower scores indicate better performance.