




Removing Basal Temporal Language Cortex in Epilepsy Surgery: Short-Term Disruption or Long-Lasting Problem?

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Temporal Lobe Regions Essential for Preserved Picture Naming After Left Temporal Epilepsy Surgery

Binder et al. *Epilepsia*. 2020;61(9): 1939-1948. doi: 10.1111/epi.16643

Objective: To define left temporal lobe regions where surgical resection produces a persistent postoperative decline in naming visual objects. **Methods:** Pre- and postoperative brain magnetic resonance imaging data and picture naming (Boston Naming Test) scores were obtained prospectively from 59 people with drug-resistant left temporal lobe epilepsy. All patients had left hemisphere language dominance at baseline and underwent surgical resection or ablation in the left temporal lobe. Postoperative naming assessment occurred approximately 7 months after surgery. Surgical lesions were mapped to a standard template, and the relationship between presence or absence of a lesion and the degree of naming decline was tested at each template voxel while controlling for effects of overall lesion size. **Results:** Patients declined by an average of 15% in their naming score, with wide variation across individuals. Decline was significantly related to damage in a cluster of voxels in the ventral temporal lobe, located mainly in the fusiform gyrus approximately 4-6 cm posterior to the temporal tip. Extent of damage to this region explained roughly 50% of the variance in outcome. Picture naming decline was not related to hippocampal or temporal pole damage. **Significance:** The results provide the first statistical map relating lesion location in left temporal lobe epilepsy surgery to picture naming decline, and they support previous observations of transient naming deficits from electrical stimulation in the basal temporal cortex. The critical lesion is relatively posterior and could be avoided in many patients undergoing left temporal lobe surgery for intractable epilepsy.

Commentary

Naming decline is among the most common and problematic consequences of epilepsy surgery, affecting up to 60% of patients undergoing dominant hemisphere anterior temporal lobectomy (ATL).¹ Decades of research have defined a broad neuro-anatomical circuit *involved* in naming including anterolateral temporal,² lateral frontal, basal temporal, and hippocampal regions³; but the regions *essential* for successful naming performance are not fully known and vary among individuals. In the context of epilepsy surgery, defining the full extent of essential naming areas has been a challenge due to the complexity of naming, the broad tissue damage induced by most open surgeries, and the limited variability in surgical approaches included in most studies. Current practice has prioritized testing and sparing regions in the lateral temporal cortex. However, given the high rate of naming decline it is reasonable to assume that additional regions need equal consideration when tailoring surgeries. To identify eloquent regions, clinicians rely on either indirect sources of information, including fMRI activation patterns, or direct but spatially limited information including cortical stimulation of exposed cortex.

A recent study by Binder et al.⁴ employed an advanced MRI approach, *voxel-based symptom lesion mapping (VSLM)*, to

address regions essential to naming performance in epilepsy. VSLM leverages the heterogeneity in lesions across patients to study structure–function relationships with spatial precision (ie, voxel-wise). In this study, pre- and post-operative structural MRI and visual object naming data were acquired for 59 patients with drug-resistant temporal lobe epilepsy (TLE) who were part of a multi-site, prospective study. Patients underwent a variety of temporal lobe resective or ablative surgeries, including ablation of the temporal pole, ventral temporal region, and medial temporal lobe, as well as selective amygdalo-hippocampotomies (SLAH) and neocortical temporal resections. Although the sample was dominated by ATLS (N = 22), the posterior and superior extent of the ATLS varied within the group, as did the degree to which resections were tailored based on language mapping. This heterogeneity in surgical locations and approaches provided ample opportunity to characterize the association between lesion location/extent and naming decline. Using VSLM, the authors evaluated the relationship between the presence or absence of a surgical lesion at each voxel and the degree of pre- to postsurgical naming decline. Binder et al. observed that a greater decline in naming was associated with larger resections, older age at surgery, and older age of seizure onset. Interestingly, visual object naming decline was not associated with damage to many traditional regions implicated in






naming, including the temporal pole, hippocampus or core perisylvian cortex. Rather, decline was associated with a cluster of voxels in the left mid-fusiform gyrus, and this cluster explained over 50% of the variance in naming decline. The authors concluded that this basal temporal language area (BTLA) is critical to visual object naming and could be avoided in many patients undergoing epilepsy surgery given that it is located at the posterior end of the standard ATL resection zone.

The existence of the BTLA is well-established in the literature⁵ and is supported by functional neuroimaging, lesion-based, and stimulation studies describing language sites distributed along the ventral temporal lobe spanning ~1 to 9 cm from the temporal tip. However, *what role* the BTLA, or rather the mid-fusiform region, plays in naming and whether this region is specific to *visual object* naming is less clear. One theory is that lesions in the fusiform disrupt visual object naming by interfering with the formation of an abstract visual object representation as information proceeds along the ventral (ie, object) processing stream. However, compelling evidence from electrocorticography and fMRI has demonstrated increases in broad-based gamma activity and BOLD activations within this region to both visual and auditory descriptive naming, as well as disruption to naming by direct cortical stimulation that is not modality-specific.⁶ Early PET data have also demonstrated activations in the mid-fusiform to both auditory and visual stimuli alike.⁷ These data favor theories implicating this region as a lexical semantic “hub” for accessing heteromodal semantic information.⁶ Thus, whether the mid-fusiform is essential for visual object naming specifically or plays a broader role that is modality-independent is not answered in Binder’s study, which did not include auditory naming. A deeper understanding of the specificity of this region will require multi-modality testing of naming pre- and post-operatively, precise knowledge of BTLA naming sites and surgical borders, and additional studies with heterogeneous surgical samples.

From a clinical perspective, the importance of sparing the BTLA may depend on whether BTLA-associated naming decline is transient or long-lasting. Binder et al. observed a significant naming decline in almost half of patients at a relatively early (~7 month) time interval post-ATL. However, a recent study by Abdallah et al.⁸ has provided some evidence that removal of BTLA sites (BTLA+) may produce a transient naming decline that resolves in many patients over a 1–4 year period. Naming recovery, however, did not reach the level of patients in whom the BTLA was spared (BTLA–) and was not achieved by all patients. In fact, none of the patients in the BTLA+ group showed naming improvements relative to their baseline performance, whereas 33% of those in the BTLA– group showed gains over time that exceeded their pre-surgical naming scores. These gains are similar to those reported following many SLAH surgeries,⁹ which leave the collateral white matter and temporal neocortex, including the BTLA intact. Consolidating findings across different research methodologies will be key to delineating how to identify critical BTLA sites and defining which BTLA sites may cause permanent deficits if removed.

In the highlighted study by Binder et al., we are reminded of the importance of the BTLA to object naming and how its

removal increases risk for post-surgical naming decline. The VSLM approach employed and the heterogeneity in surgeries are key strengths of this study, enabling the detection of structure-function effects across a range of resected areas. However, the study leaves us wondering not only *what exact role* the mid-fusiform plays in naming, but for *which* patients might naming not improve over time if critical BTLA regions are removed? Even if the removal of critical BTLA areas does not cause permanent language deficits in many patients, concerns remain that removing these regions could still limit post-operative gains in the long term. Further, as an older age at surgery is a risk factor for 1-year naming decline,¹⁰ the possibility arises that older individuals or those with low functional reserve may be less prone to functional re-organization or post-operative compensation by adjacent or homologous brain regions. Thus, older individuals could be at higher risk of permanent naming deficits if BTLA regions are removed. This will need to be considered as it is becoming common to perform ATLs on patients in their 60s and 70s—an age group whose brains may be less capable of reorganization. Finally, it is not clear whether the naming deficits that ensue, whether transient or long-lasting, are the result of removing BTLA cortex or a network of regions that includes white matter pathways (eg, inferior longitudinal fasciculus) that connect the fusiform to other critical language structures. Future studies combining structural MRI and stimulation mapping with diffusion tensor imaging may help to address this question and advance our understanding of how essential the BTLA is to the broader language network.

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