Epileptiform spikes in specific left temporal and mesial temporal structures disrupt verbal episodic memory encoding

Camarillo-Rodriguez L¹, Waldman ZJ¹, Orosz I⁵, Stein J⁶, Das S⁵, Gorniak R³, Sharan AD⁴, Gross R¹⁰, Lega BC¹¹, Zaghloul K¹², Jobst BC¹³, Davis KA⁹, Wanda PA⁸, Worrell G¹⁴, Sperling MR², Weiss SA¹

¹Depts. of Neurology and Neuroscience; ²Dept. of Neurology; ³Dept. of Radiology, ⁴Dept. of Neurosurgery, Thomas Jefferson University, Philadelphia, PA, U.S.A. 19107. ⁵Dept. of Neurology David Geffen School of Medicine at UCLA, Los Angeles, CA, U.S.A. 90095. ⁷Penn Image Computing and Science Laboratory, Department of Radiology, ⁸Department of Psychology, ⁹Department of Neurology, University of Pennsylvania, Philadelphia, PA 19104, USA. ¹⁰Department of Neurosurgery, Emory University, Atlanta GA, USA. ¹¹University of Texas Southwestern Medical Center, Department of Neurosurgery, Dallas TX, USA. 75390. ¹²Surgical Neurology Branch, NINDS, NIH, Bethesda, MD 20892, USA. ¹³Dartmouth-Hitchcock Medical Center, Department of Neurology, Lebanon NH, USA. 03756. ¹⁴Depts. of Neurology, Physiology & biomedical Engineering, Mayo Clinic, Rochester, MN 55905.
ABSTRACT (162 WORDS)

Patients diagnosed with epilepsy experience cognitive dysfunction that may be due to a transient cognitive/memory impairment (TCI/TMI) caused by spontaneous epileptiform spikes. We asked in a cohort of 166 adult patients with medically refractory focal epilepsy if spikes in specific neuroanatomical regions during verbal episodic memory encoding would significantly decrease the probability of recall. We found using a naïve Bayesian machine learning model that the probability of correct word recall decreased significantly by 11.9% when spikes occurred in left Brodmann area 21 (BA)21, (p<0.001), 49.7% in left BA38 (p=0.01), and 32.2% in right BA38 (p<0.001), and 21.4% in left BA36 (p<0.01). We also examined the influence of the seizure-onset zone and the language dominant hemisphere on this effect. Our results demonstrate that spontaneous epileptiform spikes produce a large effect TCI/TMI in brain regions known to be important in semantic processing and episodic memory. Thus memory impairment in patients with epilepsy may be attributable to cellular events associated with abnormal inter-ictal electrical events.
INTRODUCTION (208)

Patients diagnosed with epilepsy experience cognitive dysfunction due to a diverse set of risk factors\(^1\text{-}^3\), and epileptiform spike\(^4\text{-}^6\) and high-frequency oscillation (HFO)\(^7\) activity is one of several risk factors that are associated with memory deficits in patients with epilepsy\(^1\text{-}^3\). One possible mechanism is that epileptiform activity directly produces a transient cognitive/memory impairment (TCI/TMI)\(^4\text{-}^7\), although alternative mechanisms have been suggested\(^8\text{-}^10\). Several large recent studies have shown that when epileptiform activity occurs in the left temporal neocortex during the encoding and recall epoch of a verbal episodic memory task a significant decrease in the probability of recall results\(^4\text{-}^7\). In animal models of mesial-temporal lobe epilepsy, epileptiform spikes have also been associated with memory impairment\(^11,12\), although this effect is less clearly directly attributable to the spikes\(^10,13\).

The location wherein epileptiform spikes disrupt memory are contested\(^4\text{-}^7\): Theoretically, the neuroanatomical location where spikes occur should determine the type of transient cognitive impairment (i.e., semantic processing, memory, attention, visual, social deficit, etc). This theory assumes that epileptiform spikes causally produce TCI/TMI because of the changes in neural firing associated with these events\(^12,14\). In this study of considerable sample size, we used spikes in the intracranial EEG (iEEG) during encoding to statistically parametrically map the neuroanatomic structures critical in semantic processing and verbal episodic memory.

RESULTS (1112)

The study cohort consisted of 166 adult patients with medically refractory focal epilepsy (Figure 1A) due to diverse etiologies implanted with depth intracranial electrodes (Figure 1B). Among the 166 patients 80 (48.2\%) of the subjects were male (ages 18-64, mean age=37, Table S1, S7). All the subjects participated in a verbal free recall task (Figure 1C). During each block of the task, subjects were instructed to memorize a list of 12 words that were presented sequentially on a computer screen, then solve arithmetic operations and finally recall as many words as possible during 30 sec. The proportion of words recalled was 27.5\% (95\% confidence interval, CI: 27.2-27.76\%) across all the subjects (Figure 1D). Word order influenced the probability of recall, and words presented first exhibited a primacy effect (Figure 1D1). Using a Generalized Linear
Mixed Model (GLMM) we determined whether subject’s verbal comprehension index (VCI), age or gender had an effect on word recall. Only the VCI and age had a significant effect (Figure 1D2, but not gender ($\beta_{VCI} = 0.02, p<0.0001$; $\beta_{Age} = -0.01, p<0.01$; $\beta_{gender} = -0.016, p>0.05$).

At the same time subjects solved the task, intracranial recordings were obtained from different neuroanatomical locations (Figure 1B, Table S2-S4). The left temporal neocortex was the most densely covered region and exhibited the most spikes across all the subjects, while the occipital lobes had the least coverage and exhibited the least spikes (Figure 1B, Table S2-S4). A previously validated algorithm was used to automatically identify inter-ictal epileptiform spikes (IED), during the word encoding epoch (Figure S3). Overall, we analyzed 459 unique iEEG recordings, 17,142 depth intracranial electrodes, 118,292 word encoding trials across 166 subjects. Prior to analysis, we visually validated every spike detection and eliminated all false positive spike detections.

Among the 166 subjects, only in 51.2% (n=85) of the patients did we detect epileptiform spikes during any of the word encoding trials (Figure 1A). After aggregating the identified epileptiform spikes that occurred in each word encoding trial by neuroanatomical region as defined by Brodmann area (BA), we calculated the mean rate of epileptiform spikes per minute per location across the entire cohort (Table S3, S4). We also derived the mean rate of epileptiform spikes by location in a subset of the cohort with detailed neuroanatomical segmentation of mesial-temporal lobe structures (Figure S1, Table S5). We found that the largest number of epileptiform spikes were detected in left BA21 (middle temporal gyrus, Table S3) and left amygdala (Table S5), but the highest rate of spikes were seen elsewhere in the few patients that had coverage of these regions (Table S3,S4).

We next asked if the detection of a epileptiform spike in a particular neuroanatomical region defined by BAs during encoding of a word reduced the probability of recalling that word. To assess this question, we implemented a naïve bayesian machine learning (NBML) model. The NBML model was used because its training utilized the joint prior probability distributions that account for the simultaneous occurrence of epileptiform spikes in multiple BAs. In order to improve the accuracy of the NBML model, we used forward sequential selection$^{15,16}$ to select the BAs (i.e. model features) wherein the occurrence of epileptiform spikes were most likely to influence the probability of recall. Using this criteria we excluded n=46 Brodmann areas due to
insufficient power (Table S3), and n=15 regions due to statistical independence (Figure 2A). To test the accuracy of the model we calculated the loss from 10-fold out of sample testing and found that it was 19.6% (i.e. successful recall could be predicted in 80.4% of trials using spikes alone, 50% chance).

The NBML model demonstrated that only when epileptiform spikes occur in the left middle temporal gyrus (BA21), left temporal pole (BA38), left perirhinal cortex (BA36), and right temporal pole (BA38) the probability of correct word recall decreased significantly by 11.9% (left BA21, p<0.001, n=1,693 events), 49.7% (left BA38, p=0.01, n=618), 32.2% (right BA38, p<0.001, n=304), and 21.4% (left BA36, p<0.01, n=191) respectively (Figure 2A, Table S5).

One shortcoming of the NBML model was that electrodes were localized using segmentation of the T1 volumetric MRI images resulting in imprecise localization of electrodes implanted in mesial-temporal structures. Fortunately, in a subset of 117 (70.5%) of the 166 subjects, T2 oblique sequences through the mesial-temporal lobes were available and used for a more accurate segmentation protocol. To examine the effect of epileptiform spikes on recall probability in each subregion of the medial temporal lobe, we used logistic regression models (LRMs). We found that epileptiform spikes did not decrease the odds of word recall in area CA1 (p>0.05, adaptive Hochberg corrected, n=1,376 events, Figure 3). However, IED in the left entorhinal (OR of word recall: 0.7, 95% confidence interval (CI): 0.56–0.88, n = 224, adaptive Hochberg, p=0.016, Figure 3, Table S7) perirhinal (OR: 0.8, 95% CI: 0.73–0.87, n = 1,708, adaptive Hochberg p<0.001, Figure 3) and parahippocampal cortex (OR: 0.73, 95% CI: 0.67–0.79, n= 371, adaptive Hochberg, p<0.001, Figure 2B) did disrupt memory encoding. In the right mesial-temporal regions we did not find a significant effect (p>0.05, Figure 2B, Table S6).

To assess if the IED that are within the seizure onset zone (SOZ) are most disruptive in comparison with the IED that are outside, we used LRMs to investigate the interaction between the occurrence of these events with the SOZ for those neuroanatomical areas that showed a statistically significant effect irrespective of the location of the SOZ. Epileptiform spikes within the SOZ disrupted encoding in the left entorhinal cortex (OR: 0.63, CI: 0.56–0.71, p<0.001, adaptive Hochberg corrected, Figure 4), left perirhinal cortex (OR: 0.78, 95% CI: 0.73–0.83, p<0.001), left Brodmann area (BA) 36 (OR: 0.4, 95% CI: 0.27–0.61, p<0.001), and right BA 38 (OR: 0.68, 95% CI: 0.6–0.76, p<0.001). Only in the left BA21 and BA38, did epileptiform spikes not disrupt
memory encoding more when they occurred inside the SOZ (Figure 4B, OR: 0.66, 95% CI: 0.49–0.89, p<0.001).

Finally, we utilized LRM s to determine if epileptiform spikes occurrence in the subject's hemisphere that is dominant for language would have a larger effect on disrupting word encoding. Of 166 patients, 65% had left hemisphere dominance (n=108), 5.4% right dominance (n=9), 1.8% bilateral hemisphere dominance (n=3), and in 27.7% this information was not available (n=46). In the left BA21, BA36, BA38 and left PHC if an epileptiform spike occurred ipsilateral to the hemisphere dominance the odds of word recall were significantly decreased (p<0.01, adaptive Hochberg, Table 1). Only in the left Entorhinal Cortex the odds of recall significantly decreased independent of the subject’s language dominant hemisphere (OR: 0.43, CI: 0.33–0.55, p<0.001; and OR: 0.34, CI: 0.19–0.6, p<0.001 respectively).

DISCUSSION (306 WORDS)

Our study confirms that epileptiform spikes in brain regions known to be involved in semantic processing in the left temporal neocortex\textsuperscript{17,18} and verbal episodic memory in the left mesial-temporal lobe\textsuperscript{19,20} result in a TCI/TMI. We found that discharges in the left entorhinal cortex, left perirhinal cortex, and left parahippocampal cortex during encoding significantly disrupted the odds of word recall. However, we did not observe a similar statistically significant effect of discharges in hippocampal regions including area CA1, or right mesial-temporal structures. The hippocampus is thought to be important in memory encoding\textsuperscript{19,20}, and epileptiform spikes in the human hippocampus disrupt verbal episodic recall\textsuperscript{6}. From a structural anatomical perspective, the entorhinal cortex is the entry point of information into the hippocampal formation\textsuperscript{20}, and from a functional perspective the entorhinal cortex is the “pacemaker” essential for forming temporal associations and in temporal coding\textsuperscript{21–24}. Thus, when discharges occur there, it may temporarily disable the “pacemaker” and render temporal associations and temporal coding ineffective. This observation may help to resolve the paradoxical and often debated effects of electrical stimulation of hippocampal and entorhinal cortex stimulation on memory encoding\textsuperscript{25–28}.

We found that in the left temporal pole (BA38), and the left perirhinal cortex (BA35) and entorhinal cortex, epileptiform spikes within the SOZ disrupted encoding with a larger effect size. Our study and prior studies\textsuperscript{5} have demonstrated that in the left lateral temporal neocortex, epileptiform spikes outside the SOZ may result in a
relatively larger disruption of verbal episodic memory encoding. This result is important because it suggests that resecting mesial-temporal, but not lateral neocortical temporal, epileptogenic regions may help to reduce transient memory impairment from spontaneous epileptiform discharges. In addition, our results suggest that epileptiform discharges in mesial-temporal regions in patients with Alzheimer’s disease\textsuperscript{29}, and also in the temporal poles in patients with semantic dementia\textsuperscript{18}, contribute to verbal episodic memory impairment.

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Conflicts of Interest

S.A.W. and Z.J.W. both hold more than 5% equity interest in Fastwave L.L.C., an EEG software manufacturer.

METHODS

Subjects: Patients undergoing iEEG monitoring as part of the presurgical treatment for drug-resistant epilepsy were recruited in this multicenter study\textsuperscript{4,4,30,31}. Data were collected from the following: Mayo Clinic (Rochester, MN), Thomas Jefferson University Hospital (Philadelphia, PA), Dartmouth-Hitchcock Medical Center (Lebanon, NH), Hospital of the University of Pennsylvania (Philadelphia, PA), Emory University Hospital (Atlanta, GA), University of Texas Southwestern Medical Center (Dallas, TX), Columbia University Medical Center (New York, NY), and National Institute of Health Clinical Center (Bethesda, MD). Each respective Institutional Review Board (IRB) approved the research protocol, and informed consent was obtained from each subject. Electrophysiological recordings were collected from clinical subdural and depth electrodes (AdTech Inc., PMT Inc.).
Electrode localization and defining the location of the SOZ: Coordinates of depth electrode contacts were obtained from post-implantation computed tomography (CT) scans. Preimplantation volumetric T1-weighted magnetic resonance imaging (MRI) scans were coregistered to the CT scans as well as to the normalized Montreal Neurological Institute 152 (MNI152) standard brain to enable comparison of recording sites in a common space across subjects. The clinically defined SOZ was identified using visual inspection of ictal iEEG by clinicians at each of the research sites4,7.

Memory task: Subjects engaged in a list learning free recall memory tasks. During each testing block, 12 words chosen at random were displayed on a computer screen. Each word was displayed for 1600 ms, and the interword interval was jittered between 750 and 1000 ms. Lists were chosen from (http://memory.psych.upenn.edu/WordPools) a pool of high-frequency nouns. Following the word display block, an arithmetic task was used as a distractor for 20 s. Participants then verbally recalled the words in any order for 30 s. Patients performed up to 25 blocks per session, and some of the patients performed more than one session4,7.

Intracranial EEG data: Intracranial data were recorded using either a Nihon Kohden EEG-1200, Natus XLTek EMU 128, or Natus Quantum, or Grass Aura-LTM64. The iEEG signals were sampled at either 500, 1000, or 1600 Hz and were referenced to a common contact placed either intracranially, on the scalp, or the mastoid process. Next, a bipolar montage was derived. To examine the effects of spikes on encoding, we analyzed iEEG between the 750 ms of interword interval and 1600 ms during word display.

Intracranial EEG analysis: To examine the effects of epileptiform spikes on encoding, we analyzed iEEG during both the 750 ms of interword interval and 1600 ms during word display. Prior to data preprocessing, we first determined which depth electrode iEEG recordings exhibited a signal-to-noise ratio sufficient for accurate ripple detection. iEEG recordings were bandpass filtered (80-240 Hz) using a finite impulse response filter. The filter order was set to be 1/3 of the number of data points in the trial, to maximize the filter order for the calculation. For each electrode contact recording we calculated the root mean square (RMS) value during each encoding word trial. We took the mean of this RMS value and excluded electrode contacts exceeding between 1.5-1.8 µV depending on the amplifier manufacturer used to acquire the data7.

We next generated a time–frequency (TF) plot of each trial using wavelet convolution in the time domain. Complex Morlet wavelets were created with constant frequency domain width $f_o = \frac{\sigma_f}{\sigma_f}$, where $f_o$ is the wavelet central frequency and $\sigma_f$ is the standard deviation of its Gaussian envelope in the frequency domain$^{7,32,33}$. The central frequency, and its standard
deviation of the Gaussian envelope values were frequency dependent and varied between the lower and upper limits (50-240 Hz) of the time-frequency (TF) analysis. Prior to performing the wavelet convolution, the digital recordings of the iEEG were padded with zeros until the sample count was equal to the closest power of two greater than the initial number of samples. The time frequency plot was not normalized. Due to boundary effects caused by continuous wavelet convolution of finite length signals, a range of 50 – 240 Hz was selected for the TF plot in order to buffer the frequency range of interest, then frequencies below 80 Hz were discarded. To further reduce boundary effects, we also discarded the initial and final 70 msec of the TF plot. To identify the candidate epileptiform spike events in the TF plot, we used a power magnitude threshold of $1 \times 10^7$ arbitrary units or detected the maximum power. A second stage algorithm identified the presence of an interictal discharge, within 200 ms of the maximum or threshold power, on the basis of an analysis of TF plots resulting from wavelet convolution.

Sharply contoured epileptiform spikes produce a motif on TF spectrograms related to the waveform morphology and not the amplitude of the event. The novel epileptiform spike detector sought to identify this motif by using a topographical analysis of the TF plot that identified and characterized distinct elevations in both the power (Figure S1b), and the gradient of the power (Figure S1c), in TF space. These elevations represent objects and we hypothesized that objects that met certain criteria would always correspond with interictal epileptiform spike events.

We identified these candidate events by first creating objects by thresholding the TF plot and its gradient to values >20% of the maximum. After applying this threshold it was possible to define the borders of the objects in the resulting TF plot, and its gradient, by using this same threshold to derive a binary map. We subsequently calculated the volume of each object, within its defined boundaries, using a trapezoidal surface integration for each of the objects. To determine if any of the identified and characterized objects were representative of interictal epileptiform spikes we applied separate thresholds to the derived volumes for the objects in the TF plot and its gradient.

Specifically, the iEEG trials were processed using a real Morlet-based wavelet convolution to compute the TF map. The wavelets were created with constant frequency domain width $\frac{f_0}{\sigma_f}$=6, to better distinguish shapes associated with spikes in the TF plot from background activity. We analyzed the portion of the resulting TF plot centered around the detected ripple event ±200 ms. We derived a gradient plot of the TF plot by calculating both the horizontal gradient of the TF plot $\nabla P_t = \frac{\delta (\text{power magnitude})}{\delta (\text{time})}$ and the vertical gradient of the TF plot $\nabla P_f = \frac{\delta (\text{power magnitude})}{\delta (\text{frequency})}$ and combining these two gradients as $\nabla TF_{map} = \sqrt{(\nabla P_f)^2 + (\nabla P_t)^2}$. 
To define the thresholds for both the TF plot and its gradient used to define the object boundaries we used 20% of the respective maximum values. Following binarization of the maps using this threshold we identified the boundary coordinates of each object using a Moore-neighbor tracing algorithm modified by Jacob’s stopping criteria. We then determined the volume of each object within its boundaries by approximating the surface integral using trapezoidal numerical integration.

To identify the objects that corresponded to interictal epileptiform spikes, we first identified the object with the maximum power coordinate. We then determined if the volume of this object met a predetermined threshold and if in the gradient plot corresponding objects also met a predetermined threshold. The correspondence of the object in the TF plot and the objects in the gradient plot was confirmed by measuring the distance between the centroids of these objects. Due to edge effects we excluded objects in the TF plot that had a power maximum value near the TF plot borders. We also excluded objects that had a height-width ratio less than 0.7, because these objects more often represented bursts of gamma oscillations.

The custom software written in Matlab™ (Mathworks, Natick, MA) generated an XML file that was imported into Micromed Brain Quick™ (Micromed S.p.A, Veneto TV, Italy). The XML file specified the iEEG electrode recording channels and trials that exhibited epileptiform spikes. LCR subsequently visually validated these spike detections and deleted false positive epileptiform discharge detections specified by the custom software. The specified trials and electrode recording channels that exhibited the visually validated epileptiform discharges were imported from XML into Matlab™ and used for statistical analysis. In a small subset of the records from 10 patients SAW also edited the XML files in Brain Quick and the intraclass correlation coefficient was similar to previously reported values measured in the laboratory using a prior version of the algorithm.

**Naive Bayesian Machine Learning Model:** Naïve Bayesian machine-learning models (NBML) are advantageous in that when these models are trained the resulting prior probabilities reflect discharges in a variety of neuroanatomical regions occurring within the same word encoding trial. However, the trained model still assumes that the discharges in each region are conditionally independent. The epileptiform discharges recorded from individual electrodes were aggregated within the corresponding neuroanatomical regions of interest (Brodmann areas). A two-dimensional matrix that indicates for each trial the locations wherein a spike occurred (Brodmann areas x trials) and an outcome vector (i.e. recalled vs. forgotten trials) were constructed by concatenating the word encoding trials across multiple sessions and patients. When a patient did not have electrode coverage in a given Brodmann area, it was coded in the matrix as missing values. Brodmann areas that exhibited fewer than 180 discharges across the concatenated word encoding trials were excluded due to simulations indicating that these regions would exhibit a Bayes factor less than 3 or higher than 1/3 which are associated with insufficient
evidence assuming a large effect size80,85. Prior to constructing the NBM, regions in which epileptiform discharges produced no significant effect (X² test, p>0.05) on the probability of recall, were excluded. A categorical Naïve Bayesian machine learning model (NBM) was trained using the remaining matrix and outcome array in Matlab™ (fitcnb.m). This model could calculate the posterior probability of correct recall given the occurrence of an epileptiform discharge in a specified Brodmann area (effect size) on the basis of the maximum likelihood estimation using the prior probabilities derived from the two-dimensional matrix and outcome array. The significance of the posterior probability estimation was determined by implementing a bagging algorithm63 by first performing a label shuffling permutation test (n=2000 permutations) using the success of word recall for each subject as the label (1 or 0), but maintaining the overall recall probability for each subject. Then, a categorical NBM was trained using each permuted outcome i.e. (recalled words) array. The posterior probabilities (Δrecall probability) corresponding to a discharge in any one of the Brodmann areas calculated from the permuted NBMs (n=2000) were used to derive a p-value for the corresponding posterior probability from the unpermuted NBM under the assumption of a normal distribution35 (multiple comparisons corrected, p=0.01)36–38. The non-permuted trained NBM was cross-validated by calculating the loss from 10-fold out of sample testing (crossval.m).

Repeated measures logistic regression models (LRMs) for word encoding trials: In contrast to the NBML model, logistic regression models (LRMs) cannot easily account for the effects of discharges that occur in multiple brain regions within a single trial. However, LRMs can include covariates like whether a neuroanatomic region is in the SOZ, or hemispheric dominance. The epileptiform spike events recorded from individual electrodes were aggregated across the corresponding regions of interest. If a given subject did not have coverage of a neuroanatomical region, then, the data from that subject were missing for the region. The events of each word recall and proportions of word recall events in 12-word blocks were analyzed using the generalized linear mixed model (GLMM) with the assumption of binomial distribution. The generalized estimating equation (GEE) estimation approach for population-average GLMM was used with the exchangeable working correlation matrix to account for correlations between rates of recall in repeated words within blocks, repeated blocks within experiment, and repeated experiments within the same subject. The GLMM allows for variable number of observations per subject representing variable number of experiments and word blocks per patient. Separate models were fitted for each neuroanatomical region of interest. For each region, predictors of individual word recall included the type of event and location of the corresponding electrode inside or outside the SOZ or whether the patient’s language dominant hemisphere was (0) not determined (1) left hemisphere dominant (2) right hemisphere dominant. Predictors of proportions of word recall events in 12-word blocks included the The p-values from each set of models (including models for multiple regions of interest) were corrected for multiple comparisons using the adaptive Hochberg algorithm39. The data were analyzed in R (Vienna, Austria) and SAS 9.4 (Cary, NC). A
GLMM in SAS 9.4 was also implemented to correlate recall performance with age, gender, and the verbal comprehension index.

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Figure 1. Patient Recruitment, task overview and performance. (A) Patient inclusion and exclusion criteria. Over a four-year period from different clinical sites, a total of 344 neurosurgical patients to engage in cognitive tasks at the same they were under intracranial monitoring. Of the 344, we focus exclusively on a subset of depth electrode patients who participated in a verbal free recall task (FR1) or its categorical version (CatFR1) recruited between Nov 2013 and April 2017. Of the 169 patients included, we discarded three subjects because their iEEG recordings exceeded a noise threshold. (B) Lateral views of a rendered brain and neuroanatomical locations wherein the electrophysiological signals were recorded. The color bar indicates the number of depth electrodes recorded in each region across all subjects. (C) Subjects participated in a verbal free recall task, that has three main components (encoding, distractor, and recall). First, the subject is instructed to memorize a list of 12 words that are presented sequentially on a computer screen separated by an inter-word interval (encoding). Follows the distractor period during which the participants are required to solve arithmetic tasks for 20 sec. Finally, the subject has 30 seconds to recall as many words as possible. (D1) Percentage of words recalled across all subjects as a function of their presentation during a list. Note that the performance in the categorical verbal free recall (Catfr1) was higher in comparison to the non-categorical version of the task (Fr1). (D2) Percentage of words recalled plotted as a function of subject’s verbal comprehension index. As the verbal comprehension index increased also the odds of word recall (LRM, OR:1.02, 95% CI:1.016-1.028, p < 0.0001). Amy, Amygdala; EC, Entorhinal Cortex; PHC, Parahippocampal Cortex; PRC, Perirhinal Cortex. MTL, Medial Temporal Lobe; IED, interictal epileptiform spikes.
Figure 2. The occurrence of epileptiform spikes in left temporal and mesial temporal structures during the word encoding period was associated with a decrease in the probability of recall. (A) Neuroanatomical sites wherein the occurrence of an epileptiform discharge during the word encoding period change significantly the probability of recalling a word in the naïve Bayesian model (NBM) (lighter shade red corresponds to effect size, \(p<0.01\)). We estimated the probability values with a naïve Bayesian model (Methods). Dark blue colored regions did not show a significant effect with \(\chi^2\) testing. Light blue areas showed no significant effect in the model (see Methods). (B1) Odds ratio values were obtained from logistic regression models of mesial-temporal subregions in a distinct subset of patients (lighter shade blue corresponds to effect size, \(p<0.05\), adaptive Hochberg corrected) NS, non significant regions shown in red. (B2) A comparison of the odds ratio computed from logression regression models (LRMs) utilizing the complete and mesial-temporal patient cohorts, epileptiform spikes within the seizure onset zones (SOZ) disrupted encoding in the left entorhinal cortex, perirhinal cortex, Brodmann area (BA) 36, and right BA 38. We did not fit a SOZ model either for the Left PHC or Left BA 38, because there were no electrodes in SOZ regions in these areas. (Asterisks correspond to \(p<0.05\), adaptive Hochberg corrected).
Tables

Table 1. Effect of spike occurrence on word recall performance by language hemisphere lateralization across subjects. Odds of successful recall given the interaction between the hemisphere dominance for language and the occurrence of an IED in selected mesial temporal regions and BA during the word encoding period. Best fit parameters were obtained with a logistic regression model per each area. CI, Confidence Interval; ND, lateralization not determined.

| Hemisphere dominance | OR     | CI 95%     | p corrected |
|----------------------|--------|------------|-------------|
| **Left Entorhinal Cortex** |        |            |             |
| ND                   | 0.84   | [0.69-1.01]| 0.059       |
| Left                 | 0.43   | [0.33-0.55]| <0.0001*    |
| Right                | 0.34   | [0.19-0.6] | 0.0002*     |
| **Left Parahippocampal** |        |            |             |
| Left                 | 0.7    | [0.66-0.74]| <0.0001*    |
| **Left Perirhinal Cortex** |        |            |             |
| ND                   | 0.51   | [0.29-0.88]| 0.015*      |
| **Left BA 21**       |        |            |             |
| ND                   | 0.78   | [0.46-1.31]| 0.34        |
| Left                 | 0.56   | [0.4-0.796]| 0.008*      |
| Right                | 0.72   | [0.71-0.73]| ND          |
| **Left Brodmann area 36** |        |            |             |
| ND                   | 0.93   | [0.29-2.9] | 0.89        |
| Left                 | 0.93   | [0.71-1.22]| 0.59        |
| **Left Brodmann area 38** |        |            |             |
| ND                   | 0.9    | [0.61-1.33]| 0.61        |
| Left                 | 0.83   | [0.67-1.02]| 0.07        |
| Right                | 0.2    | ND         | ND          |
| **Right Brodmann area 38** |        |            |             |
| ND                   | 1.24   | [0.88-1.73]| 0.21        |
| Left                 | 0.61   | [0.53-0.69]| <0.0001*    |
Supporting Materials

Epileptiform spikes in specific left temporal and mesial temporal structures disrupt verbal episodic memory encoding

Camarillo-Rodriguez L¹, Waldman ZJ¹, Orosz I⁵, Stein J⁶, Das S⁵, Gorniak R³, Sharan AD⁴, Gross R¹⁰, Lega BC¹¹, Zaghloul K¹², Jobst BC¹³, Davis KA⁹, Wanda PA⁸, Worrell G¹⁴, Sperling MR², Weiss SA¹

¹Depts. of Neurology and Neuroscience, ²Dept. of Neurology, ³Dept. of Radiology, ⁴Dept. of Neurosurgery, Thomas Jefferson University, Philadelphia, PA, U.S.A. 19107. ⁵Dept. of Neurology David Geffen School of Medicine at UCLA, Los Angeles, CA, U.S.A. 90095. ⁶Penn Image Computing and Science Laboratory, Department of Radiology, ⁷Department of Psychology, ⁸Department of Neurology, University of Pennsylvania, Philadelphia, PA 19104, USA. ⁹Department of Neurosurgery, Emory University, Atlanta GA, USA. ¹¹University of Texas Southwestern Medical Center, Department of Neurosurgery, Dallas TX, USA. 75390. ¹²Surgical Neurology Branch, NINDS, NIH, Bethesda, MD 20892, USA. ¹³Dartmouth-Hitchcock Medical Center, Department of Neurology, Lebanon NH, USA. 03756. ¹⁴Depts. of Neurology, Physiology & biomedical Engineering, Mayo Clinic, Rochester, MN 55905.
Table S1. Characteristics of the patients included in the study.

| Variable                        | Number, range | %, mean, or SEM |
|--------------------------------|---------------|-----------------|
| **A. Age**                      |               |                 |
| Both                           | [18-64 y]     | mean=37.6       |
| Male                           | 84/166        | 50.6%           |
| Female                         | 80/166        | 48.2%           |
| NA                             | 2/166         | 1.2%            |
| **B. Gender**                   |               |                 |
| Male                           | 84/166        | 50.6%           |
| Female                         | 80/166        | 48.2%           |
| NA                             | 2/166         | 1.2%            |
| **C. Performance**              |               |                 |
| FR1                            | [24.9-25.5]   | 25.17%          |
| CatFR1                         | [31.7-32.6]   | 32.1%           |
| Sessions per subject           | mean=2.41     | SEM=0.13        |
| Trials per subject             | mean=551.2    | SEM=23.8        |
| **D. Handedness**               |               |                 |
| Left                           | 19/166        | 11.4%           |
| Right                          | 135/166       | 81.3%           |
| Ambidextrous                   | 9/166         | 5.42%           |
| NA                             | 3/166         | 1.8%            |
| **E. Dominant hemisphere**      |               |                 |
| Left                           | 108/166       | 65%             |
| Right                          | 9/166         | 5.4%            |
| Bilateral                      | 3/166         | 1.81%           |
| Inconclusive or NA             | 46/166        | 27.7%           |
| **F. Etiologies of epilepsy**   |               |                 |
| Hypoxic-ischemic encephalopathy| 6/166         | 3.6%            |
| Inborn errors of metabolism    | 0/166         | 0%              |
| Intraventricular hemorrhage    | 1/166         | 0.6%            |
| Malformations of cortical development | 16/166 | 9.6% |
| Mesial temporal sclerosis      | 8/166         | 4.82%           |
| NA                             | 2/166         | 1.2%            |
| Neoplasia                      | 4/166         | 2.4%            |
| Neurocutaneous syndrome        | 1/166         | 0.6%            |
| Other etiology                 | 45/166        | 27.1%           |
| Other metabolic or toxic insults | 0/166      | 0%              |
| Stroke                         | 3/166         | 1.8%            |
| Traumatic brain injury         | 48/166        | 28.9%           |
| Unknown cause                  | 70/166        | 42.17%          |
| Viral, bacterial and parasitic infections | 9/166 | 5.42% |
| **G1. MRI impression of specific abnormalities** | | |
| Atrophy or tissue loss         | 15/166        | 9%              |
| Encephalocoele                 | 0/166         | 0%              |
| Encephalomalacia/gliosis       | 27/166        | 16%             |
| Inflammatory/infectious        | 0/166         | 0%              |
| Malformation of cortical development | 14/166 | 8% |
| Mesial temporal sclerosis      | 15/166        | 9%              |
| NA                             | 2/166         | 1%              |
| Neoplasm                       | 3/166         | 2%              |
| Other                          | 32/166        | 19%             |
| Vascular                       | 2/166         | 1%              |
| **G2. MRI Distribution**       |               |                 |
| Left Frontal                   | 20/166        | 12%             |
| Left Temporal Neocortical      | 11/166        | 7%              |
| Left Mesial Temporal           | 25/166        | 15%             |
| Left Parietal                  | 16/166        | 10%             |
| Left Occipital                 | 9/166         | 5%              |
| Right Frontal                  | 19/166        | 11%             |
| Right Temporal Neocortical     | 17/166        | 10%             |
| Right Mesial Temporal          | 23/166        | 14%             |
| Right Parietal                 | 11/166        | 7%              |
| Right Occipital                | 6/166         | 4%              |
| **H. Seizure lateralization**  |               |                 |
| Left                           | 71/166        | 43%             |
| Right                          | 53/166        | 32%             |
| Bilateral                      | 30/166        | 18%             |
| Undetermined                   | 12/166        | 7%              |
**Table S2.** IED detections and rates of epileptiform discharges in specific neuroanatomical regions defined by Brodmann Areas (BA). Number of spikes detected and validated across all subjects, mean rate of epileptiform discharges per minute and standard error of the mean’s rate (SEM, see Methods). For the computation of each statistic we discarded iEEG recordings were the mean rate of epileptiform discharges per minute was equal to 0.

| Brodmann area | N. of subjects | Spikes detected | Mean rate IED/min | SEM |
|---------------|----------------|-----------------|-------------------|-----|
| L-Amygdala    | 23             | 1234            | 5.08              | 0.81|
| L-Brodmann area 13 | 12             | 480             | 5.21              | 1.29|
| L-Brodmann area 19 | 10             | 687             | 7.74              | 1.79|
| L-Brodmann area 20 | 23             | 784             | 5.42              | 0.74|
| L-Brodmann area 21 | 32             | 1693            | 5.30              | 0.69|
| L-Brodmann area 22 | 15             | 548             | 7.89              | 1.50|
| L-Brodmann area 28 | 6              | 989             | 6.98              | 1.81|
| L-Brodmann area 31 | 11             | 233             | 6.59              | 1.35|
| L-Brodmann area 34 | 8              | 246             | 6.04              | 1.90|
| L-Brodmann area 35 | 3              | 191             | 6.54              | 1.90|
| L-Brodmann area 36 | 13             | 277             | 5.92              | 1.43|
| L-Brodmann area 37 | 9              | 719             | 8.45              | 2.09|
| L-Brodmann area 38 | 13             | 618             | 5.21              | 1.01|
| L-Brodmann area 39 | 8              | 583             | 9.38              | 1.71|
| L-Brodmann area 4_6 | 6              | 263             | 4.29              | 0.73|
| L-Brodmann area 40 | 11             | 418             | 6.19              | 1.50|
| L-Brodmann area 7  | 4              | 223             | 7.10              | 2.60|
| L-Caudate Tail  | 10             | 198             | 5.60              | 1.30|
| L-Hippocampus   | 22             | 1092            | 5.01              | 1.04|
| R-Amygdala      | 17             | 567             | 4.39              | 0.81|
| R-Brodmann area 13 | 15             | 240             | 4.72              | 1.00|
| R-Brodmann area 20 | 26             | 1044            | 4.34              | 0.53|
| R-Brodmann area 21 | 20             | 1168            | 4.28              | 0.51|
| R-Brodmann area 22 | 12             | 192             | 4.83              | 1.08|
| R-Brodmann area 24 | 3              | 270             | 2.51              | 1.21|
| R-Brodmann area 31 | 11             | 311             | 3.20              | 0.68|
| R-Brodmann area 36 | 15             | 312             | 3.80              | 0.74|
| R-Brodmann area 38 | 8              | 304             | 6.19              | 1.17|
| R-Brodmann area 40 | 9              | 293             | 5.64              | 1.14|
| R-Brodmann area 47 | 6              | 306             | 5.09              | 1.30|
| R-Caudate Tail  | 6               | 234             | 6.26              | 1.53|
| R-Hippocampus   | 11             | 307             | 3.89              | 0.82|
Table S3. IED detections and rates of epileptiform discharges in specific neuroanatomical regions defined by BA. The areas shown here were discarded from the NBML analysis because the number of spikes detected were not enough to reach the desired statistical significance. Number of spikes detected and validated across all subjects, mean rate of epileptiform discharges per minute and standard error of the mean’s rate (SEM, see Methods). For the computation of each statistic we discarded iEEG recordings were the mean rate of epileptiform discharges per minute was equal to 0.

| Brodmann area               | N. of subjects | Spikes detected | Mean rate IED/min | SEM  |
|-----------------------------|---------------|----------------|-------------------|------|
| L-Brodmann area 1_2_3_5     | 4             | 34             | 4.4               | 2.4  |
| L-Brodmann area 10          | 6             | 87             | 6.1               | 2.2  |
| L-Brodmann area 11          | 5             | 92             | 7.3               | 1.9  |
| L-Brodmann area 17          | 1             | 1              | 7.8               | 0.0  |
| L-Brodmann area 18          | 3             | 33             | 7.8               | 2.7  |
| L-Brodmann area 23          | 1             | 13             | 6.5               | 0.0  |
| L-Brodmann area 24          | 2             | 18             | 5.0               | 4.0  |
| L-Brodmann area 25          | 1             | 1              | 3.6               | 0.0  |
| L-Brodmann area 27          | 1             | 39             | 10.1              | 2.2  |
| L-Brodmann area 30          | 5             | 118            | 5.0               | 2.0  |
| L-Brodmann area 32          | 3             | 9              | 6.4               | 1.9  |
| L-Brodmann area 41_42       | 4             | 62             | 7.0               | 2.8  |
| L-Brodmann area 43          | 1             | 116            | 3.5               | 0.0  |
| L-Brodmann area 45          | 1             | 42             | 15.1              | 0.0  |
| L-Brodmann area 46          | 1             | 1              | 0.2               | 0.0  |
| L-Brodmann area 47          | 4             | 169            | 4.7               | 2.6  |
| L-Brodmann area 8           | 1             | 3              | 3.6               | 0.0  |
| L-Brodmann area 9           | 4             | 59             | 4.7               | 1.3  |
| L-Caudate Body              | 1             | 4              | 3.5               | 0.0  |
| L-Corpus Callosum          | 5             | 75             | 7.2               | 2.0  |
| L-Lateral Globus Pallidus   | 1             | 4              | 3.2               | 2.6  |
| L-Optic Tract              | 1             | 98             | 8.4               | 0.0  |
| L-Pulvinar                  | 1             | 42             | 16.8              | 0.0  |
| L-Putamen                   | 5             | 107            | 4.0               | 1.6  |
| R-Brodmann area 1_2_3_5     | 6             | 75             | 3.6               | 1.0  |
| R-Brodmann area 10          | 3             | 91             | 5.5               | 1.2  |
| R-Brodmann area 11          | 3             | 27             | 5.0               | 1.6  |
| R-Brodmann area 19          | 2             | 6              | 7.1               | 4.2  |
| R-Brodmann area 23          | 3             | 76             | 6.5               | 1.4  |
| R-Brodmann area 27          | 1             | 56             | 3.9               | 1.3  |
| R-Brodmann area 28          | 5             | 159            | 2.9               | 0.5  |
| R-Brodmann area 32          | 2             | 33             | 7.3               | 0.2  |
| R-Brodmann area 34          | 1             | 3              | 2.6               | 0.0  |
| R-Brodmann area 35          | 6             | 69             | 2.7               | 0.7  |
| R-Brodmann area 37          | 9             | 104            | 3.7               | 0.6  |
| R-Brodmann area 39          | 3             | 8              | 2.5               | 1.9  |
| R-Brodmann area 4_6         | 8             | 121            | 3.3               | 0.9  |
| R-Brodmann area 44          | 1             | 5              | 3.0               | 0.0  |
| R-Brodmann area 45          | 1             | 10             | 0.3               | 0.0  |
| R-Brodmann area 7           | 3             | 172            | 3.7               | 1.4  |
| R-Brodmann area 8           | 6             | 67             | 3.8               | 1.3  |
| R-Brodmann area 9           | 7             | 115            | 3.6               | 0.9  |
| R-Caudate Body              | 1             | 1              | 8.7               | 0.0  |
| R-Corpus Callosum          | 4             | 167            | 5.1               | 1.6  |
| R-Putamen                   | 3             | 90             | 6.8               | 2.4  |
| R-VPL Nucleus               | 1             | 7              | 0.2               | 0.0  |
Table S4. IED detections and rates of epileptiform discharges in specific neuroanatomical regions defined by mesial temporal regions. Number of spikes detected and validated across all subjects, mean rate of epileptiform discharges per minute and standard error of the mean’s rate (SEM, see Methods). For the computation of each statistic we discarded iEEG recordings were the mean rate of epileptiform discharges per minute was equal to 0.

| Mesial temporal | N. of subjects | Spikes detected | Mean rate IED/min | SEM  |
|----------------|----------------|-----------------|-------------------|------|
| Left Amy       | 16             | 2,754           | 6.68              | 1.17 |
| Left CA1       | 11             | 1,376           | 6.01              | 1.76 |
| Left CA2       | 2              | 342             | 9.61              | 3.81 |
| Left DG        | 3              | 589             | 7.25              | 3.76 |
| Left EC        | 5              | 224             | 3.59              | 0.93 |
| Left PHC       | 2              | 371             | 3.82              | 0.81 |
| Left PRC       | 13             | 1,708           | 4.80              | 0.97 |
| Left Sub       | 3              | 412             | 5.48              | 2.95 |
| Right Amy      | 9              | 836             | 4.52              | 1.33 |
| Right CA1      | 3              | 238             | 3.84              | 2.00 |
| Right DG       | 1              | 10              | 0.63              | 0.00 |
| Right EC       | 4              | 302             | 2.92              | 0.48 |
| Right PHC      | 5              | 697             | 3.92              | 0.85 |
| Right PRC      | 7              | 640             | 3.40              | 0.61 |
| Right Sub      | 1              | 10              | 0.63              | 0.00 |
Table S5. Results of the Naïve Bayesian Machine Learning Model of the effect of epileptiform discharges during encoding in specific Brodmann areas on recall probability. P, probability; p, p value; n, number of IED.

| Brodmann area       | Change in the probability of recall |
|---------------------|-------------------------------------|
| L-Brodmann area 4_6 | P=130.73, p= 0.0400, n=263         |
| L-Brodmann area 13  | P=133.46, p= 0.1160, n=480         |
| L-Brodmann area 19  | P=88.74, p= 0.7920, n=687          |
| L-Brodmann area 20  | P=-33.18, p= 0.3060, n=784         |
| L-Brodmann area 21  | P=-11.61, p= 0.0000, n=1693        |
| L-Brodmann area 28  | P=-15.42, p= 0.6360, n=989         |
| L-Brodmann area 36  | P=-21.41, p= 0.0030, n=277         |
| L-Brodmann area 37  | P=138.48, p= 0.4380, n=719         |
| L-Brodmann area 38  | P=-49.68, p= 0.0130, n=618         |
| L-Brodmann area 39  | P=112.43, p= 0.5640, n=583         |
| L-Hippocampus       | P=150.74, p= 0.7430, n=1092        |
| R-Amygdala          | P=146.94, p= 0.6850, n=567         |
| R-Brodmann area 21  | P=-10.49, p= 0.1330, n=1168        |
| R-Brodmann area 31  | P=-49.20, p= 0.0420, n=311         |
| R-Brodmann area 36  | P=-40.39, p= 0.3730, n=312         |
| R-Brodmann area 38  | P=-32.19, p= 0.0010, n=304         |
| R-Brodmann area 47  | P=-46.48, p= 0.4010, n=306         |
Table S6. Results of the GLMs of the effect of epileptiform discharges during encoding in specific mesial-temporal regions on recall probability. The confidence interval and p values for the Right Dendate Gyrus model was not computed by the SAS algorithm because there was not enough data. ND, not determined.

| Mesial-temporal areas            | OR   | CI 95%    | p corrected |
|----------------------------------|------|-----------|-------------|
| Left Amygdala                    | 0.90 | [0.72-1.12] | 0.351       |
| Left CA1                         | 1.06 | [0.82-1.35] | 0.665       |
| Left CA2                         | 0.76 | [0.59-0.98] | 0.226       |
| Left Entorhinal cortex           | 0.70 | [0.56-0.88] | 0.016       |
| Left Parahippocampal gyrus       | 0.73 | [0.67-0.79] | <0.0001     |
| Left Perirhinal cortex           | 0.80 | [0.73-0.88] | <0.0001     |
| Left Subiculum                   | 0.78 | [0.62-0.97] | 0.025       |
| Right CA1                        | 1.09 | [0.88-1.35] | 0.438       |
| Right Dendate Gyrus              | 0.19 | ND        | ND          |
| Right Entorhinal cortex          | 1.09 | [0.79-1.51] | 0.587       |
| Right Parahippocampal gyrus      | 0.95 | [0.83-1.09] | 0.470       |
| Right Perirhinal cortex          | 1.17 | [0.98-1.39] | 0.078       |
**Table S7.** Characteristics of the patients included in the study. Demographic information, handedness, verbal comprehension index (VCI), dominant hemisphere and method of determination and clinical information. Age at the time of implant. The verbal comprehension assessment was done with the WAIS IV test or WASI II test. Unknown* etiology of epilepsy means that there were not relevant abnormalities on examination, cognition, history or imaging. If the etiology of epilepsy is due to infections, it could be either viral, bacterial or parasitic. The information about the seizure onset localization was retrieved from the clinical notes when this information was available, otherwise, it was retrieved from the electrode notes. Abbreviations: A, ambidextrous; F, female; HIE, Hypoxic Ischemic Encephalopathy; IVH, Intraventricular Hemorrhage; L, left; M, Male; N/A, not available; ND, not determined, R, right; TBI, Traumatic Brain Injury.
| ID | Age | Gender | Handed | VCI | Dominant hemisphere and method | Etiologies of epilepsy | MRI findings and localization | Seizure Onset Zone |
|----|-----|--------|--------|-----|-------------------------------|------------------------|-----------------------------|----------------------|
| 1  | 48  | F      | R      | 81  | Left fMRI Unknown*            | Normal hippocampi      | R temporal lobe             |                      |
| 2  | 49  | F      | R      | 89  | Left fMRI Unknown*            | Multiple prominent parahippocampal cysts | Bilateral neocortical temporal regions |                      |
| 3  | 39  | F      | A      | 107 | Bilateral WADA TBI            | None                   | L anterior temporal lobe     |                      |
| 4  | 52  | F      | R      | 78  | Inconclusive N/A              | Specific etiologies of epilepsy, TBI | L-frontal encephalomalacia/gliosis | L-Frontal Lobe, L-Limbic Lobe, L-Parietal Lobe |
| 5  | 31  | F      | A      | 98  | Left fMRI Specific etiologies of epilepsy | Polymicrogyria along bilateral parieto-occipital sulci | Multifocal |                      |
| 6  | 37  | F      | R      | 100 | Left fMRI Specific etiologies of epilepsy | None                   | R-Occipital Lobe, R-Parietal Lobe |                      |
| 7  | 55  | F      | L      | 89  | Left fMRI Specific etiologies of epilepsy | Post-op changes from previous R ant temporal lobectomy | R-Insula |                      |
| 8  | 49  | F      | L      | 98  | Left fMRI Unknown*            | Abnormal, other        | R-Frontal Lobe, R-Temporal Lobe |                      |
| 9  | 39  | M      | R      | 74  | Left WADA TBI                 | None                   | R-Mesial Temporal, R-Temporal Lobe |                      |
| 10 | 25  | M      | R      | 81  | Left fMRI Unknown*            | L-posterior lateral temporal and L parieto-occipital gliosis | L-cingulate region |                      |
| 11 | 33  | M      | R      | N/A | Left N/A Unknown*             | Post-op changes from previous R ant temporal lobectomy | R Insula, R superior and middle temporal regions |                      |
| 12 | 37  | F      | R      | 100 | Left Specific etiologies of epilepsy | None                   | Multifocal |                      |
| 13 | 25  | F      | R      | 112 | Bilateral WADA TBI            | L-hippocampal tissue loss; L parasagittal inferior occipital lobe encephalomalacia | L anterior temporal lobe, L occipital/suboccipital regions |                      |
| 14 | 48  | M      | R      | 93  | Left fMRI TBI                 | Post-op changes from previous R anterior temporal lobectomy; R frontal/temporal encephalomalacia | R Insular, R temporal, L/R orbitofrontal regions |                      |
| 15 | 27  | F      | R      | N/A | Left Specific etiologies of epilepsy | Abnormal, other        | R-Other, L-Mesial Temporal |                      |
| 16 | 33  | F      | R      | 108 | N/A Left N/A Unknown*         | Abnormal               | Undetermined |                      |
| 17 | 23  | M      | L      | 105 | Left fMRI HIE                 | None                   | L hippocampus, posterior amygdala | Independent L and R temporal onset |
| 18 | 40  | M      | R      | 85  | Left Aphasia TBI, HIE         | Bilateral mesial temporal sclerosis | R Mesial temporal sclerosis | R hippocampus; infrequent L hippocampus |
| 19 | 20  | F      | R      | 85  | Left WADA Mesial temporal sclerosis | R mesial temporal sclerosis | R hippocampus |                      |
| 20 | 32  | F      | R      | 85  | Left WADA Unknown*            | Mild R hippocampal volume loss | R temporal neocortical area |                      |
| 21 | 45  | F      | A      | N/A | Left Unknown*                 | None                   | L hippocampus |                      |
| 22 | 49  | M      | L      | N/A | Bilateral WADA HIE            | L mesial temporal sclerosis, L periventricular leukomalacia | L anterior and mesial temporal regions |                      |
| 23 | 28  | F      | R      | 83  | N/A Malformations of cortical or other brain development | Probably residual malformation of cortical development extending from the R insula to R lateral ventricle | R frontal perisylvian region |                      |
| 24 | 34  | M      | R      | N/A | N/A TBI                       | L and R frontal encephalomalacia/gliosis | R posterior temporal lobe |                      |
| 25 | 28  | F      | L      | N/A | Right fMRI Malformations of cortical or other brain development | Focal cortical dysplasia in R superior temporal gyrus | R posterior temporal lobe |                      |
| 26 | 59  | M      | R      | 105 | N/A Unknown*                  | Post-op changes from previous R anterior temporal lobectomy; old R parieto-occipital infarct | L hippocampus, insula, orbitofrontal regions |                      |
| 27 | 51  | M      | R      | 102 | Left fMRI TBI                 | None                   | L and R anterior hippocampus |                      |
| 28 | 51  | M      | R      | N/A | N/A Unknown*                  | Post-op changes from previous anterior R temporal lobectomy with partial R amygdalohippocampectomy; mild R hippocampal volume loss | Undetermined |                      |
| ID | Age | M/F | Hand | VCI | Dominant hemisphere and method | Etiologies of epilepsy | MRI findings and localization | Seizure Onset Zone |
|----|-----|-----|------|-----|-------------------------------|------------------------|-----------------------------|----------------------|
| 49 | 52  | F   | R    | 95  | Left fMRI                     | Specific etiologies of epilepsy | L temporal lobe lateral to temporal horn signal abnormality, encephalomalacia near previous L partial frontal lobectomy | L anterior to mid hippocampal region |
| 51 | 25  | F   | R    | 108 | Left fMRI                     | Unknown*                     | Focal cortical dysplasia L parasagittal frontal lobe | L SMA |
| 52 | 20  | F   | R    | N/A | N/A                           | Unknown*                     | None                         | R-Limbic Lobe |
| 53 | 39  | F   | R    | N/A | N/A                           | Unknown*                     | None                         | Independent L and R temporal onset |
| 54 | 24  | M   | R    | 95  | fMRI                          | Unknown*                     | Post-op changes from corpus callosotomy; multifocal periventricular nodular gray matter heterotopia including temporal lobes | Multifocal |
| 56 | 34  | M   | A    | N/A | fMRI                          | Unknown*                     | None                         | R hippocampus |
| 57 | 53  | M   | R    | 76  | N/A                           | TBI, Malformations of cortical or other brain development | Post-op changes from R anterior temporal lobectomy; extensive polymicrogyria surrounding R sylvian fissure | L hippocampus |
| 59 | 45  | F   | R    | 70  | fMRI                          | Unknown*                     | None                         | L-Mesial Temporal, L-Temporal Lobe |
| 60 | 37  | F   | R    | N/A | Left WADA                     | Unknown*                     | Post-op changes from previous R anterior temporal lobectomy with adjacent gliosis | Margins of previous R anterior temporal lobectomy |
| 61 | 21  | M   | R    | N/A | Left fMRI                     | Specific etiologies of epilepsy | R parietal resection cavity | R insula |
| 62 | 24  | F   | R    | 93  | Left fMRI                     | Infection                     | Post-op changes related to R anterior temporal lobectomy | R insula and R frontal lobe |
| 63 | 24  | M   | R    | 80  | N/A                           | TBI                          | None                         | L-Mesial Temporal, L-Other |
| 65 | 34  | F   | R    | 116 | N/A                           | Unknown*                     | Punctate acute infarct in R centrum semiovale; developmental venous anomaly in anterior R frontal lobe | Bilateral mesial and noocortical temporal areas |
| 66 | 39  | M   | R    | 107 | Left fMRI                     | TBI, Specific etiologies of epilepsy | R anterior temporal cortical lesions, likely DNET | R-Limbic Lobe, R-Temporal Lobe |
| 67 | 45  | F   | R    | N/A | Left WADA                     | TBI                          | Incidental, other            | L anterior and inferior temporal regions |
| 68 | 39  | F   | A    | N/A | fMRI                          | TBI                          | None                         | R anterior temporal lobe |
| 69 | 27  | M   | R    | N/A | Left fMRI                     | Malformations of cortical or other brain development | Probable focal cortical dysplasia in L motor cortex | L anterior lateral frontal region and L parietal region |
| 70 | 40  | F   | R    | N/A | Left fMRI                     | Infection                     | Prior neurocysticercosis     | L mesial temporo-occipital region |
| 74 | 25  | M   | R    | N/A | N/A                           | Unknown*                     | None                         | Undetermined |
| 77 | 48  | F   | R    | N/A | ND fMRI                       | Specific etiologies of epilepsy | Questionable signal abnormality in R hippocampus | R anterior hippocampus or R temporal pole |
| 80 | 43  | F   | R    | 91  | ND fMRI                       | Infection                     | None                         | L-Occipital L, L-Temporal L, R-Mesial Temporal |
| 81 | 34  | F   | R    | 108 | N/A                           | TBI                          | Slight asymmetry of hippocampal formations | L frontal lobe |
| 83 | 49  | F   | R    | 95  | Left fMRI                     | TBI                          | None                         | L anterior/mesial temporal lobe |
| 84 | 25  | M   | R    | N/A | Left fMRI                     | Unknown*                     | None                         | L medial primary sensory cortex |
| 86 | 21  | M   | R    | N/A | Left WADA                     | Specific etiologies of epilepsy | None                         | R anterior temporal lobe; possible R post. quadrant |
| 89 | 36  | M   | L    | N/A | Left WADA                     | Infection, TBI               | None                         | Bilateral mesial temporal and L-Temporal Lobe |
| 92 | 45  | M   | R    | 95  | Left fMRI                     | Specific etiologies of epilepsy | Slightly larger L amygdala | L and R mesial temporal regions |
| 93 | 25  | M   | R    | 105 | Left fMRI                     | Unknown*                     | None                         | Bilateral mesial temporal and L-Temporal Lobe |
| 94 | 47  | M   | R    | N/A | Left fMRI                     | TBI                          | None                         | L-Mesial Temporal, L-Parietal and L-Temporal Lobe |
| 96 | 34  | F   | R    | 96  | N/A                           | Specific etiologies of epilepsy | Encephalomalacia/gliosis     | L-Mesial Temporal, R-Mesial Temporal |
| ID  | Age | Sex | Handedness | VCI | Dominant hemisphere and method | Etiologies of epilepsy                                                                 | MRI findings and localization                                                                 | Seizure Onset Zone              |
|-----|-----|-----|------------|-----|-------------------------------|--------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------|----------------------------------|
| 100 | 44  | F   | R          | 103 | Left WADA                   | TBI                                                                                  | None                                                                                         | L-Mesial Temporal               |
| 101 | 26  | F   | L          | N/A | Right fMRI                  | Stroke, HIE, Mesial temporal sclerosis                                              | L mesial temporal sclerosis; prior L parietal lobe infarct                                      | L mesial temporal lobe          |
| 102 | 35  | M   | R          | N/A | Left WADA                   | TBI                                                                                  | Atrophy of bilateral hippocampi (likely sequelae of previous head trauma); susceptibility artifacts in bilateral cerebral hemispheres | Bilateral mesial temporal areas |
| 104 | 22  | M   | R          | N/A | Left WADA                   | Unknown*                                                                            | Possible encephalohoele in R anterior paramedian sphenoid sinus                             | R orbitofrontal cortex          |
| 105 | 25  | M   | R          | 89  | Right fMRI                  | TBI                                                                                  | Extensive gliosis/encephalomalacia in R parietal lobe                                        | L-Parietal Lobe                 |
| 106 | 27  | M   | R          | N/A | Right N/A                   | TBI, HIE, Specific etiologies of epilepsy                                            | Ecephalomalacia/gliosis in R cerebral hemisphere from old R MCA stroke                        | Non-diagnostic (intraop. recording only)                                           |
| 107 | 25  | M   | R          | 100 | N/A                          | Specific etiologies of epilepsy                                                     | Other                                                                                         | L-Occipital Lobe                |
| 108 | 24  | F   | R          | 95  | Right WADA IVH              | L posterior temporal and occipital lobes cortical dysplasia                          | L hippocampus                                                                               |                                 |
| 111 | 20  | M   | R          | N/A | Left fMRI                   | TBI                                                                                  | L parieto-occipital encephalomalacia/gliosis; smaller R hippocampus                           | L anterior temporal and parieto-occipital regions                                      |
| 112 | 30  | F   | R          | 74  | Right fMRI                  | TBI, Specific etiologies of epilepsy                                                | Asymmetric prominence of R hippocampal formation                                             | R mesial temporal lobe          |
| 114 | 32  | F   | A          | 110 | Inconclusive fMRI           | TBI, Specific etiologies of epilepsy                                                | Heterotopia in ventricle adjacent to L temporal-occipital lobes                               | L posterior temporal and parietal regions                                        |
| 115 | 47  | M   | L          | N/A | Left fMRI                   | TBI                                                                                  | Other                                                                                         | Bilateral mesial and neocortical temporal areas                                    |
| 118 | 33  | M   | R          | 98  | N/A                          | fMRI                                                                                 | Malformations of cortical or other brain development                                        | L posterior subtemporal region                                           |
| 119 | 27  | F   | L          | 68  | Right WADA Unknown*         | None                                                                                 | L-Temporal Lobe                                                                              |                                 |
| 120 | 34  | F   | L          | 93  | N/A                          | Unknown*                                                                            | None                                                                                         | R mesial temporal lobe            |
| 122 | 48  | F   | R          | 127 | fMRI Unknown*               | L mesial temporal sclerosis                                                          | L mesial temporal lobe                                                                       |                                 |
| 123 | 29  | F   | R          | 95  | Left WADA                   | Mesial temporal sclerosis                                                            | L posterior temporal and occipital lobes cortical dysplasia                                   |                                 |
| 124 | 41  | F   | R          | 114 | Left fMRI Unknown*          | L mesial temporal sclerosis                                                          | Bilateral mesial and neocortical temporal areas                                               |                                 |
| 125 | 45  | F   | R          | N/A | Left WADA Unknown*          | None                                                                                 | Undetermined                                                                                |                                 |
| 127 | 40  | F   | R          | N/A | Left fMRI Unknown*          | None                                                                                 | R parietal lobe                                                                              |                                 |
| 128 | 26  | M   | R          | 122 | N/A                          | Specific etiologies of epilepsy                                                     | Previous R temporal lobe resection                                                           | L-Mesial Temporal               |
| 130 | 57  | M   | R          | N/A | Left fMRI                   | TBI                                                                                  | Anterior and inferior R frontal and temporal lobe encephalomalacia; small lesions in inferior L frontal and temporal lobes | L mesial and lateral superior frontal regions                                      |
| 131 | 24  | M   | R          | N/A | Left fMRI                   | Specific etiologies of epilepsy                                                     | Multiple patchy linear lesions in R frontal white matter                                     | R frontotemporal regions         |
| 134 | 65  | M   | R          | N/A | Left WADA                   | Specific etiologies of epilepsy                                                     | L lateral occipital cortical/subcortical encephalomalacia                                      | L-Mesial Temporal, L-Occipital Lobe, R-Limbic Lobe                                |
| 135 | 48  | M   | R          | 78  | Left fMRI Unknown*          | Bilateral perisylvian polymicrogyria (R>L)                                           | L inferior parieto-occipital region                                                          |                                 |
| 136 | 57  | F   | R          | 100 | N/A                          | fMRI Unknown*                                                                        |closure lenticulostriate system                                                               |                                 |
| 138 | 42  | M   | R          | N/A | Left N/A                    | Specific etiologies of epilepsy                                                     | L mesial temporal sclerosis                                                                 | L-Mesial Temporal, R-Parietal Lobe                                             |
| 141 | 45  | F   | R          | 93  | Left fMRI Unknown*          | L mesial temporal sclerosis                                                          | Bilateral mesial and neocortical temporal areas                                               |                                 |

11
| ID  | Age | M/F | Handedness | VCI  | Dominant hemisphere and method | Etiologies of epilepsy                                | MRI findings and localization                        | Seizure Onset Zone                        |
|-----|-----|-----|------------|------|-------------------------------|-------------------------------------------------|-----------------------------------------------------|------------------------------------------|
| 142 | 44  | F   | L          | N/A  | N/A fMRI                       | Unknown*                                        | R frontal lobe hamartoma vs low grade glioma        | R-Frontal Lobe, R-Limbic Lobe, R-Other   |
| 144 | 53  | M   | R          | 125  | Right fMRI                     | TBI                                             | R mesial temporal encephalomalacia/gliosis          | R-Mesial Temporal                        |
| 145 | 46  | M   | R          | 95   | N/A N/A                        | Stroke                                          | R anterior temporal region, anterior insula         |                                         |
| 146 | 45  | F   | R          | 81   | Left fMRI                      | Malformations of cortical or other brain development | Posterior R frontal lobe cortical dysplasia | R-Frontal Lobe                           |
| 147 | 48  | M   | R          | 96   | Left WADA                      | TBI, Specific etiologies of epilepsy            | L middle and inferior temporal gyri cortical dysplasia | L mesial temporal lobe                   |
| 148 | 60  | F   | R          | 98   | Left fMRI                      | Specific etiologies of epilepsy                 | Mild R hippocampal volume loss; old infarct in aneurysm with post-surgical changes | R mesial temporal lobe                   |
| 150 | 50  | F   | R          | 81   | Left fMRI                      | Specific etiologies of epilepsy                 | L fronto-insular cavernous malformation            | L-Other                                 |
| 151 | 36  | M   | R          | 103  | Left WADA                      | Infection                                       | Normal; Bilateral frontal DBS stimulator leads     | L-Mesial Temporal, L-Other, L-Temporal Lobe |
| 153 | 38  | M   | L          | N/A  | Left N/A                       | Mesial temporal sclerosis                       | R mesial temporal sclerosis                        | L anterior hippocampus; R mesial temporal lobe |
| 154 | 36  | F   | R          | 98   | Left WADA                      | Unknown*                                        | None                                                 | Undetermined                             |
| 157 | 22  | M   | R          | 98   | Left N/A                       | Unknown*                                        | None                                                 | R-Mesial Temporal, R-Parietal Lobe, R-Temporal Lobe |
| 158 | 45  | F   | R          | 89   | Left fMRI                      | Unknown*                                        | None                                                 | L-Mesial Temporal, L-Temporal Lobe        |
| 159 | 43  | F   | R          | 96   | Left WADA                      | Specific etiologies of epilepsy                 | None                                                 | R-Mesial Temporal                        |
| 161 | 54  | F   | R          | N/A  | N/A N/A                        | Malformations of cortical or other brain development | Post-surgical changes in L parietal and occipital lobes | L-Parietal Lobe, L-Temporal Lobe          |
| 163 | 46  | M   | R          | N/A  | Left fMRI                      | Specific etiologies of epilepsy                 | L parietal cystic encephalomalacia                  | L-Mesial Temporal, L-Parietal Lobe, L-Temporal Lobe |
| 164 | 38  | M   | R          | 80   | Left fMRI                      | TBI                                             | None                                                 | L-Mesial Temporal                        |
| 166 | 39  | M   | L          | N/A  | Left WADA                      | Specific etiologies of epilepsy                 | L frontal post-op changes, encephalomalacia        | L-Frontal Lobe, L-Parietal Lobe          |
| 167 | 33  | M   | R          | N/A  | Left fMRI                      | Specific etiologies of epilepsy                 | Prominent sulcation in L anterior temporal lobe    | L-Frontal Lobe, L-Temporal Lobe          |
| 168 | 24  | M   | R          | 93   | Left fMRI                      | Unknown*                                        | None                                                 | R-Mesial Temporal, R-Temporal Lobe        |
| 169 | 43  | F   | R          | 85   | Left WADA                      | TBI, Specific etiologies of epilepsy            | R frontal craniotomy and anterior R frontal lobe resection | R posterior frontal area                |
| 170 | 21  | M   | R          | 78   | N/A N/A                        | HIE                                             | Post-op changes of previous L temporal lobectomy  | Margin surrounding previous partial L temporal lobectomy |
| 171 | 36  | M   | R          | N/A  | N/A N/A                        | TBI, Neoplasia, Specific etiologies of epilepsy | Lesion in R hippocampus and amygdala              | R-Mesial Temporal                        |
| 172 | 22  | F   | R          | 93   | Left WADA                      | Unknown*                                        | None                                                 | L-Mesial Temporal, R-Temporal Lobe        |
| 173 | 19  | F   | R          | 102  | N/A N/A                        | Unknown*                                        | Small lesion in R medial temporal lobe             | R SMA                                   |
| 174 | 29  | M   | R          | N/A  | Left WADA                      | Unknown*                                        | None                                                 | L-Temporal Lobe                         |
| 176 | 41  | F   | R          | N/A  | Left WADA                      | Unknown*                                        | Post-op changes of R anterior temporal lobectomy with partial amygdałohippocampsectomy | R insula                               |
| 177 | 23  | F   | R          | N/A  | Left WADA                      | Aphasia                                         | L posterior tempo-parietal tuber                    | L posterior temporal region               |
| 178 | 40  | M   | R          | N/A  | Left WADA                      | TBI, Specific etiologies of epilepsy            | Previous L temporal resection with L limbic atrophy; L frontal lobe encephalomalacia | L hippocampus                           |
| 180 | 21  | F   | R          | N/A  | Left WADA                      | Specific etiologies of epilepsy                 | None                                                 | L-Other, L-Temporal Lobe                |
| 181 | 22  | M   | R          | 95   | Left fMRI                      | Unknown*                                        | None                                                 | R-Frontal Lobe, R-Parietal Lobe          |
| ID  | Age | M/F | Hand ed | VCI | Dominant hemisphere and method | Etiologies of epilepsy                                                                 | MRI findings and localization                                                                 | Seizure Onset Zone                              |
|-----|-----|-----|---------|-----|-------------------------------|----------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------|-----------------------------------------------|
| 184 | 42  | M   | R       | N/A | N/A                           | Malformations of cortical or other brain development, Specific etiologies of epilepsy | R posterior perisylvian region cortical dysplasia; post-op changes from previous R anterior temporal lobectomy and hippocampectomy | R-Frontal Lobe, R-Parietal Lobe, R-Temporal Lobe |
| 186 | 28  | M   | N/A     | 68  | N/A                           | TBI, Specific etiologies of epilepsy                                                   | Lesion in R posterior temporal lobe, likely capillary telangiectasia                          | L mesial and neocortical temporal lobe, R anterior neocortical temporal lobe                  |
| 187 | 52  | F   | R       | 98  | Left WADA                     | TBI, Malformations of cortical or other brain development                               | L frontal malformation of cortical development                                              | L-Frontal Lobe, L-Parietal Lobe               |
| 188 | 26  | F   | R       | 108 | N/A                           | Unknown*                                                                                 | None                                                                                         | R-Other, R-Temporal Lobe                     |
| 189 | 23  | M   | R       | N/A | N/A                           | TBI, Specific etiologies of epilepsy                                                   | Incidental, other                                                                            | R-Frontal Lobe, R-Limbic Lobe                |
| 190 | 58  | F   | R       | 96  | Left WADA                     | Unknown*                                                                                 | None                                                                                         | L-Frontal Lobe, L-Frontal Lobe, R-Temporal Lobe |
| 191 | 19  | M   | R       | 68  | N/A                           | Specific etiologies of epilepsy                                                        | None                                                                                         | R-Parietal Lobe, R-Temporal Lobe             |
| 192 | 28  | M   | R       | 81  | Left fMRI                     | Specific etiologies of epilepsy                                                        | None                                                                                         | Undetermined                                  |
| 193 | 37  | M   | R       | 83  | N/A                           | Unknown*                                                                                 | None                                                                                         | R-Other                                      |
| 195 | 44  | M   | R       | 108 | Left fMRI                     | TBI                                                                                      | None                                                                                         | Undetermined                                  |
| 196 | 19  | M   | L       | 122 | N/A                           | Specific etiologies of epilepsy                                                        | Abnormal                                                                                     | L-Parietal Lobe                               |
| 198 | 60  | F   | A       | N/A | Left fMRI                     | Neoplasia                                                                                 | L frontal, mesial and parietal neoplasm                                                     | L-Frontal Lobe, L-Parietal Lobe               |
| 200 | 26  | M   | R       | N/A | Left fMRI                     | Unknown*                                                                                 | L frontal encephalomalacia/girosis                                                          | L-Frontal Lobe, L-Other, L-Temporal Lobe     |
| 201 | 37  | M   | R       | 93  | Left fMRI                     | TBI, Specific etiologies of epilepsy                                                   | R parietal neoplasm                                                                          | R-Mesial Temporal                             |
| 203 | 36  | F   | R       | 87  | Left fMRI                     | Unknown*                                                                                 | Incidental, other                                                                            | L-Limbic Lobe, R-Limbic Lobe, R-Other         |
| 204 | 26  | F   | R       | 74  | Left fMRI                     | Unknown*                                                                                 | None                                                                                         | L-Mesial Temporal                             |
| 207 | 40  | F   | L       | 76  | Right fMRI                    | Infection                                                                                 | Mesial temporal sclerosis, encephalomalacia/girosis                                          | Multifocal                                    |
| 212 | 47  | M   | R       | N/A | N/A                           | TBI, Neoplasia, Specific etiologies of epilepsy                                         | None                                                                                         | R-Frontal Lobe, R-Limbic Lobe, R-Temporal Lobe |
| 214 | 31  | M   | R       | N/A | N/A                           | Malformations of cortical or other brain development                                   | L malformation of cortical development, Other                                               | L-Frontal Lobe, L-Limbic Lobe, L-Parietal Lobe |
| 215 | 51  | F   | R       | 83  | Left fMRI                     | Unknown*                                                                                 | None                                                                                         | L-Limbic Lobe                                 |
| 216 | 23  | F   | R       | 85  | Left fMRI                     | Unknown*                                                                                 | None                                                                                         | L-Mesial Temporal, L-Temporal Lobe           |
| 217 | 37  | M   | R       | N/A | Left WADA                     | TBI                                                                                      | Incidental, other                                                                            | L-Limbic Lobe                                 |
| 221 | 57  | M   | R       | 87  | Left fMRI                     | TBI, Specific etiologies of epilepsy                                                   | Abnormal                                                                                     | L-Mesial Temporal, L-Other, L-Temporal Lobe  |
| 223 | 43  | F   | R       | 78  | Left fMRI                     | Mesial temporal sclerosis, Specific etiologies of epilepsy                              | Mesial temporal sclerosis, Other                                                             | L-Limbic Lobe, L-Temporal Lobe               |
| 226 | 42  | F   | R       | 93  | Left fMRI                     | TBI, Mesial temporal sclerosis                                                          | Mesial temporal sclerosis                                                                     | L-Frontal Lobe, L-Mesial Temporal            |
| 227 | 33  | M   | R       | N/A | Left fMRI                     | TBI                                                                                        | R occipital encephalomalacia/girosis                                                         | R-Parietal Lobe                               |
| 228 | 59  | F   | R       | N/A | N/A                           | Malformations of cortical or other brain development                                   | L frontal malformation of cortical development                                              | R-Parietal Lobe, L-Other, R-Limbic Lobe      |
| 229 | 29  | M   | R       | N/A | Left fMRI                     | Unknown*                                                                                 | None                                                                                         | L-Parietal Lobe, L-Other                     |
| 230 | 56  | F   | R       | N/A | N/A                           | Stroke                                                                                     | None                                                                                         | R-Frontal Lobe, R-Limbic Lobe, R-Other        |
| 231 | 21  | M   | R       | N/A | N/A                           | Unknown*                                                                                 | None                                                                                         | L-Frontal Lobe, L-Parietal Lobe              |
| 232 | 28  | M   | A       | 143 | Left fMRI                     | TBI                                                                                        | None                                                                                         | L-Frontal Lobe, L-Parietal Lobe              |
| 236 | 52  | F   | R       | 95  | Left fMRI                     | TBI                                                                                        | None                                                                                         | L-Mesial Temporal                             |
| ID  | Age | M/F | Handenedness | VCI | Dominant hemisphere and method | Etiologies of epilepsy                                                                 | MRI findings and localization                                      | Seizure Onset Zone                  |
|-----|-----|-----|-------------|-----|-------------------------------|--------------------------------------------------------------------------------------|---------------------------------------------------------------------|-----------------------------------|
| 239 | 27  | M   | R           | 93  | Left WADA                     | Malformations of cortical or other brain development                                  | Bilateral encephalomalacia/gliosis, Other                            | R-Frontal Lobe, R-Limbic Lobe, R-Temporal Lobe                      |
| 240 | 38  | F   | R           | N/A | Left fMRI                     | Infection                                                                            | Bilateral atrophy or tissue loss                                    | Bilateral mesial and neocortical temporal areas                    |
| 241 | 52  | M   | R           | N/A | Left N/A                      | TBI                                                                                  | None                                                                 | Bilateral mesial and neocortical temporal areas                    |
| 243 | 64  | M   | A           | 87  | Left N/A                      | Unknown*                                                                             | None                                                                 | Undetermined                                                       |
| 247 | 62  | F   | R           | 100 | N/A N/A                       | Unknown*                                                                             | None                                                                 | L-Other, L-Temporal Lobe                                            |
| 251 | 32  | M   | L           | N/A | Left fMRI                     | Malformations of cortical or other brain development                                  | R atrophy or tissue loss, Encephalomalacia/gliosis                  | Multifocal                                                         |
| 260 | 57  | F   | R           | 95  | Left N/A                      | Infection                                                                            | None                                                                 | L-Frontal Lobe, L-Limbic Lobe, L-Temporal Lobe                    |
| 264 | 53  | F   | R           | 83  | Left WADA                     | Specific etiologies of epilepsy                                                    | Other                                                                | L-Mesial Temporal, L-Temporal Lobe                                |
| 269 | 42  | F   | R           | 110 | N/A N/A                       | Malformations of cortical or other brain development, Neoplasia, Mesial temporal sclerosis | R mesial temporal sclerosis, R encephalomalacia/gliosis                | R-Limbic Lobe                                                      |
| 273 | 48  | F   | L           | 90  | Left N/A                      | TBI                                                                                  | Abnormal                                                            | L-Frontal Lobe, L-Parietal Lobe, L-Other                           |
| 274 | 44  | F   | R           | N/A | Left fMRI                     | Unknown*                                                                             | None                                                                 | L-Parietal Lobe, L-Other, L-Temporal Lobe                          |
| 277 | 34  | M   | R           | 116 | N/A fMRI                      | TBI                                                                                  | None                                                                 | R-Frontal Lobe, R-Limbic Lobe, R-Parietal Lobe                     |
| 278 | 22  | F   | R           | 72  | Left fMRI                     | Specific etiologies of epilepsy                                                    | None                                                                | R-Parietal Lobe, R-Temporal Lobe                                  |
| 291 | 35  | M   | R           | N/A | Left fMRI                     | Infection, Specific etiologies of epilepsy                                          | Incidental, other                                                   | Multifocal                                                         |
| 422 | 57  | F   | R           | N/A | Left fMRI                     | Unknown*                                                                             | None                                                                 | L-Limbic Lobe, L-Temporal Lobe                                    |
| 423 | 49  | M   | L           | N/A | Right fMRI                    | Unknown*                                                                             | None                                                                 | R-Mesial Temporal, R-Temporal Lobe                                |
| 424 | -   | M   | L           | N/A | N/A fMRI                      | Unknown*                                                                             | N/A                                                                 | Undetermined                                                       |
| 425 | -   | -   | N/A         | N/A | -                            | N/A                                                                                  | N/A                                                                 | Undetermined                                                       |
