INTRODUCTION
There is a clinical need for more accurate and earlier diagnosis of brain disorders using non-invasive methods like electroencephalography (EEG). Unfortunately, we have a very limited understanding about what EEG tells us about the brain, making it difficult to use as a diagnostic tool. Developing a better understanding of the brain activity that underlies EEG is critical to improving accuracy and efficiency of diagnosis.

EEG detects the electrical activity of the brain via voltage measuring electrodes on the scalp’s surface. This activity at the scalp provides us with information about the activation patterns of very large brain regions relative to one another [2]. However, for improved diagnostics, we would need to know how these global changes relate to changes or damage in particular regions of the brain.

Studies that look into the neural activity in specific brain regions often use intracranial electrodes that measure signals directly from the brain. These methods are very precise but also invasive and as a result are almost always performed in animal subjects [1]. In order to bridge the gap between EEG and intracranial recordings, we could record EEG simultaneously with intracranial electrodes in animal models. Such a technique would allow us to translate between internal brain signals observed through more invasive means in animal studies and external brain signals measured in patients through EEG for diagnostic purposes.

In order to do this, we must make sure we can generalize the EEG data from simultaneous EEG and intracranial electrode studies in animals to EEG recorded in humans. One difference that might exist is in order to insert intracranial electrodes into animals, scientists must create a small opening in the skull (craniotomy). The opening in the skull has the potential to change the EEG data recorded. Previous studies have shown that large gaps in the skull can significantly alter the head’s volume conduction properties [2]. However, the craniotomy used for intracranial electrode insertion is very small and since EEG is a voltage relative measure, such a small source of current leakage is likely to have only a small effect that cancels out across the skull [1, 3]. We sought to investigate the craniotomy’s impact on EEG data.

OBJECTIVES
Our main objective was to demonstrate that EEG data recorded before and after a craniotomy have similar underlying components (Pearson’s correlation coefficient $r > 0.5$). Additionally, we wanted to show that we can predict pre-craniotomy EEG with post-craniotomy underlying components to further emphasize the similarity between the pre and post underlying components. A strong prediction would have a higher correlation coefficient with the actual data than the opposite condition’s original data.

HYPOTHESIS/SUCCESS CRITERIA
We hypothesized that the craniotomy would not substantially alter the underlying components of the EEG data and as a result the pre and post-craniotomy underlying components would be highly correlated ($r > 0.5$).

METHOD

Data Collection
Procedures were approved by the Institutional Animal Care and Use Committee of the University of Pittsburgh, and surgeries were performed in aseptic conditions under general anesthesia. We recorded EEG from eight electrodes on the scalp of two rhesus macaque monkeys both before and after a craniotomy procedure that removed a 20 mm diameter piece of skull over visual cortex and replaced it with a protective titanium mesh (we performed the craniotomy in order to implant a multielectrode array for other experiments). The subjects held their gaze fixed on a central point on a computer screen while drift grating stimuli flashed on the right or left side of the screen for 400 ms. 700 trials were performed both pre and post craniotomy for Monkey B and 500 trials were performed in both conditions for Monkey R. Data was collected over a single day before and after the craniotomy.

Independent Components Analysis
To analyze the data, we performed independent components analysis (ICA), which decomposed the data into seven independent component scores and topographies. We analyzed the similarity in magnitude and time course of the component scores via Pearson’s correlation coefficient calculated between the pre and post-craniotomy component score for each component.

Opposite component predictions analysis
To see how well we could predict the EEG of one condition with the components of the other, we used the component score ($C$) matrix from the opposite condition and the topography from the same condition ($X$) to predict the EEG potentials ($V$). For example, to predict the pre-craniotomy EEG ($V_{predict, pre}$), we would multiply the post-craniotomy component scores ($C_{post}$) with the pre-craniotomy topographies ($X_{pre}$): $V_{predict, pre} = C_{post}*X_{pre}$.

We used a bootstrap re-sampling algorithm to select random sub-samples of 250 trials from the data and repeated this 100 times to develop a 95% confidence interval of the original EEG data and the generated predictions.
As a positive control, we tested how well one-half of the data could predict the other half of the same dataset. As a negative control, we randomized the pre and post-craniotomy trials and evaluated how well one-half of the randomized trials could predict the other half.

RESULTS

Figure 1. One of seven ICA components for Monkey B, showing the similarity between the pre and post-craniotomy underlying component scores ($r > 0.8$). Shading represents the 95% CI of the resampling method.

Figure 2. Pre and post-craniotomy EEG predictions graphed with the original data in mV over 400 ms for Monkey B on one of eight electrodes (PO6- back, left of head). Shading represents the 95% CI of the resampling method.

For each of the seven components, the pre-craniotomy scores were highly correlated with the post-craniotomy component scores ($r > 0.8$). Figure 1 shows an example of one of these components.

The pre and post-craniotomy topographies for each component had qualitatively similar mappings, but the post-craniotomy topographies had smaller magnitude topographies.

We performed a qualitative analysis of the opposite component predictions in Figure 2. The pre-craniotomy EEG prediction using the post-craniotomy component scores was more similar in magnitude to the actual pre-craniotomy EEG than the actual post-craniotomy EEG. We observed the same pattern with the post-craniotomy EEG prediction. There was also a small, unexpected time-shift present in the pre-craniotomy EEG prediction for seven of the eight electrodes.

The results of the positive control were predictions that were highly correlated with the other half of the data set ($r > 0.95$) as expected. The results of the negative control were predictions that were approximately random ($r \sim 0.5$) as expected. The quantitative analysis (Pearson’s correlation coefficient and sum-squared error) of the other predictions is currently in progress.

These results were consistent for both Monkey B and Monkey R.

DISCUSSION

The data shows that the component scores of the pre and post-craniotomy EEG are highly correlated, which supports our hypothesis that the craniotomy would not substantially alter the underlying components of the EEG.

When we made predictions with the opposite condition’s component scores, qualitatively, the magnitude of the EEG prediction was similar to the original EEG data as expected from our hypothesis. However, the time courses don’t appear to be correlated as well. Before we can conclude that EEG in a subject with a craniotomy is generalizable to subjects without a craniotomy, we must determine the cause (if any) of these differences.

ICA decomposition would not introduce a time shift because it is a mathematically linear decomposition. This means that a small time shift is present between the pre and post-craniotomy data that is magnified by the prediction analysis. It’s possible that the small time-shift could simply be day to day variability in EEG. We only collected one day of data, so our next step is to collect data in another subject over multiple days before and after the surgery to determine what the day to day variability is.

It is also possible that this shift could signify a change in how signals propagate through the brain as a result of the craniotomy. The skull acts as a filter for EEG signals, so by removing a small piece, it’s possible to change the way the skull filters EEG and as a result causes a small delay for the signals to reach the electrodes.

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REFERENCES

